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THE DISEASES OF INFANTS AND CHILDREN

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SECOND EDITION, RESET
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VOLUME I

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JAMES TYSON, M.D., LL.D.

Physician, Clinician, Gentleman.

My One-time Teacher and

Always My Trusted Adviser and Friend

PREFACE TO THE SECOND EDITION

THE exhausting of the first edition and the passing of time, with the appearance of very many additions to our knowledge of pediatrics, have made the preparation of a new edition of this text-book necessary. In this work I have had the able coöperation of my friend and former assistant and associate Dr. A. Graeme Mitchell, now of the University of Cincinnati. Together and independently we have reviewed much of the very large pediatric literature which has accumulated since the printing of the first edition, and have tried to select and refer to that which seemed to us likely to be the most useful to physicians interested in any particular subject in the domain of children's diseases. Some of the older illustrations have been replaced by more serviceable ones and a number of new ones added. There are in addition several new tables, which it is hoped may be of service to the reader. Every part of the book has been subjected to a careful review, certain of the subjects have been completely rewritten, and a number of entirely new articles have been added. The numerous insertions and changes have necessitated a complete resetting.

J. P. CROZER GRIFFITH.

1810 SPRUCE STREET,
PHILADELPHIA, PA.,
February, 1927.

PREFACE

It has been the effort of the author in the following pages to present a review of the subject of medical pediatrics, as complete as seemed desirable without attempting to make it encyclopedic. Inclusion is made of such subjects in surgery and the special branches with the recognition of which physicians treating the diseases of children should be more or less familiar. While endeavoring to embody the results of his own experience through many years of contact with disease in children, he has also made free use of the numèrous excellent text-books on the subject, including the valuable contributions by American authors, and of home and foreign pediatric journal-literature. To all these he would here acknowledge his indebtedness.

In the course of his own reading he has found quotations from medical authorities of much impaired service unless accompanied by reference to the places of publication, thus rendering possible the consulting of the originals. With the feeling that others may share this sentiment, he has in footnote form given the references to literature whenever such quotations are used, believing that in this way the value of the book to many readers would be increased, while the footnote method interferes in no way with its usefulness to those others who are not interested in this line of research.

Temperature-charts and photographic and other illustrations have been reproduced freely, generally accompanied by brief synopses of the histories of the cases, without which their value would be much lessened. These are original or unpublished except in the instances where none such were obtainable, or where superior ones were found in the publications of other writers. Acknowledgment has naturally been made in every case.

Throughout the text-book the metric and the English systems of measurements have been used together, in nearly all instances putting in parentheses in the terms of one the equivalents in the other. The statistics quoted from any author have been given in the system employed by him, and the corresponding figures in the other then placed in parentheses. The equivalent values are largely those found in the tables of the United States Pharmacopœia. Ounces are respectively avoirdupois or liquid measure, except in designating the doses of solid medicaments when Troy ounces are used. Fractional amounts in grains, drams, cubic millimeters, cubic centimeters, and grams are omitted unless the quantities are small. Grams are assumed to be the equivalent of cubic centimeters, ignoring the specific gravity of many liquids, where the figures as given would not be absolutely correct. In the parentheses the abbreviations designating grams and cubic centimeters are omitted, the sense of the text making them unnecessary. An exception to the employment of both systems of measurement will be found in discussing the preparation of food in the artificial feeding of infants. Here for the most part only English measures are given, since the preparation must be made in conjunction with the graduated nursing-bottles and the liquid measures in common household use.

Numerous cross-references will be found throughout the work, thus calling attention to discussions of the subjects on other pages, which would otherwise be overlooked unless the index were consulted. Although every effort has been made to avoid inaccuracy of statements, and particularly of references, the author must expect to share the experience of others, that these will creep in to some extent.

The author has waived his own preferences in the matter, yielding to the desire of the publishers for uniformity in the system of spelling and of punctuation adopted throughout the numerous works upon medical subjects published by them.

1810 Spruce Street.
Philadelphia, Pa.

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THE DISEASES OF CHILDREN

DIVISION I

GENERAL SUBJECTS

CHAPTER I

ANATOMY AND PHYSIOLOGY

THE DIVISIONS OF LIFE

For the purpose of study human life is usually sub-divided into several stages. These are, of course, somewhat arbitrary divisions, and vary considerably with different writers. As convenient an arrangement as any is that which separates life into *Intra-uterine*, or fetal life; *Infancy*, including the *New-Born* as a sub-division; *Childhood*, early and later; *Youth* or adolescence, with *Puberty* as the initial period of this stage; *Adult* life, and its sub-division, *Old Age*.

Intra=uterine Life.—Although apparently closely resembling a new-born child, the fetus is in reality so different that it is not capable of independent existence. Many of its organs, though perfect, are not yet active. Circulation is not in the complete state which it will later assume. Respiration and digestion are entirely in abeyance, oxygen and nutriment being obtained through the maternal blood.

Being so closely dependent upon the mother for its life, it follows that the condition of her health and her methods of living cannot fail to exert a great influence upon the health of the fetus, and, indeed, upon the whole later existence of the child.

The fetus may also directly inherit a feeble constitution from a delicate father or mother, irrespective of the production of debility by the preventable causes mentioned. Actual disease may be transmitted from mother to fetus. This is occasionally true of such infectious disorders as measles, whooping-cough, typhoid fever, malaria, tuberculosis and the like, and very frequently of syphilis. The tendency to rheumatism or to gout is directly transmitted, as are certain nervous maladies, although the latter are oftener the result of other nervous conditions in the parents, or of imperfect hygiene of the nervous system of the mother while pregnant.

There are, besides, a variety of accidents which may happen to the fetus *in utero*, such as fractures, dislocations, amputations and the like. Various diseases may occur, or anomalies of development arise, producing monsters or lesser degrees of malformations, the reason for the occurrence of which is little understood so far as any preventive measures are concerned. Other characteristics and disorders of this period are fully treated of in works upon obstetrics and diseases of the embryo and fetus.

The condition of development seen in infants the subject of *premature birth* will be considered when discussing that topic (p. 284).

The New-Born.—The first period of infancy is that in which the child is designated as *New-Born.* This term is applied not only to the time immediately after birth, but to a slightly longer period, regarding the exact duration of which there is some variance of opinion. Bouchut[1] and Rilliet and Barthez,[2] for instance, limited it to the first few days of life; while Henoch[3] extended it to the age of 4 to 6 weeks, and Parrot[4] to 3 months. It seems best to apply the title to the first 2 or, at longest, 3 weeks, since in this period no very distinct alterations show themselves during the process of development. The change from intra-uterine to extra-uterine life is so great that the new-born are very subject to disturbances of health, especially to those accidents and disorders caused by birth or appearing shortly after it. There are also a number of pathological conditions seen at birth which developed during fetal life (p. 283).

Infancy.—This taken as a whole is a division of life to which various arbitrary and rather confusing limitations have been set. A classification common especially among French and German writers makes infancy (première enfance; Säuglingsalter) close with the age of 7 or 8 months, the beginning of dentition, or with the age of 1 year. Childhood (seconde enfance; Kindersalter) now begins—the first portion of it up to the age of about 36 months being called the *period of the first dentition*—and terminates with the age of 6 years. Youth (Knabensalter) then commences with 6 years, and forms the period of the second dentition, lasting until puberty. Popularly, however, in English-speaking countries infancy is generally considered as lasting until the age of 2 years and this seems the most convenient and best definition, since during this period the important epoch of the first dentition is nearly closed, and features are exhibited, especially in the earlier part of it, which differ in many respects from those seen in the later years of child-life. Children in the first year may be called *nurslings.*

Development, both of the body and of the mind, during infancy is very rapid, except that for the first few months there is little discoverable gain in intellectual power. Infants, especially those of an early age, are extremely susceptible to external depressing influences, such as cold and fatigue, often easily affected by drugs, and rapidly exhausted by disease. On the other hand they exhibit remarkable recuperative power when recovery begins.

Childhood.—Following the classification adopted here the term childhood is best used to cover the period from the close of infancy to the development of puberty. It may conveniently be divided into *Early Childhood* from the age of 2 to that of 6 years, the latter marking the beginning of the second dentition, and *Later Childhood* (Knabensalter) from the age of 6 years to puberty.

During childhood growth continues with a rapidity which, though great, is less than in infancy. The mental and physical differences between the sexes become constantly more apparent. The nervous system is still much in evidence although to a less extent than in infancy. The incidence of diseases at different periods of infancy and childhood will be discussed later (p. 235).

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[1] Maladies des nouveau-nés, 1885, 1.
[2] Sanné, Maladies des enfants, 1884, I, 5.
[3] Vorlesungen über Kinderkrankheiten, 1895, 21.
[4] Clinique des nouveau-nés, 1877, 4.

Youth.—According to the generally accepted definition the term *Youth* or *Adolescence* is applied to the period beginning with puberty and terminating with the commencement of adult life; *i.e.*, 21 to 25 years. The exact time for the development of puberty varies not only with individuals but with race and climate. In general for temperate climates it may be placed at from 14 to 16 years for boys and from 12 to 15 years for girls. Common law places it at 14 years for males and 12 years for females. With the occurrence of puberty the sexual functions are established in both sexes, the genital organs increase in size, and the growth of hair begins upon the pubis and later in the axillæ. In the female the breasts enlarge and become rounded by a deposit of fat. In the male the voice changes and hair begins to grow upon the face. The psychic characteristics belonging to each sex now become accentuated. The attributes and diseases most peculiar to youth do not, of course, come under special consideration in the study of pediatrics.

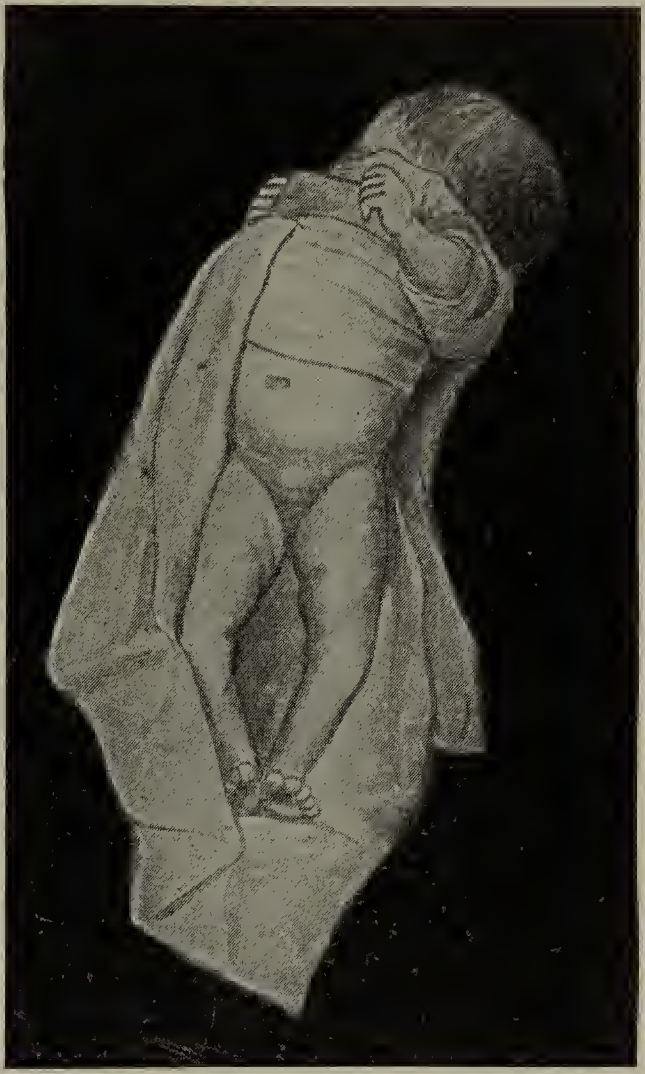

Fig. 1.—Baby a Few Weeks Old, Showing the Natural Curve of the Legs with the Bending in of the Soles. *(From a photograph.)*

THE NEW-BORN

The infant at birth is more or less covered by a white, waxy substance, the *vernix caseosa*, which protected it during fetal life. This is especially abundant on the flexor surfaces, the back and in the folds of the body, although some children have almost none of it upon them. It is composed of the thickened secretions of the sebaceous glands and of scales of epidermis. When it has been removed by washing the skin is found to be thin, smooth, delicate and of a deep-reddish color. The lanugo characteristic of fetal life has generally largely disappeared in fully developed new-born children. Many infants, however, still show a fine soft down.

The child's flesh should be plump and firm from a good development of subcutaneous fat. The head is proportionately very large and often rather thickly covered with long hair. The eyes are an indeterminate blue, and are usually kept shut or but half open. The face is expressionless. The chest is small and narrow and the abdomen large and prominent. The arms are short and are held most of the time flexed and pressed against the body in the position maintained in the uterus. The hands are generally closed, but will grasp firmly any object placed in them. The nails are well-developed, and project beyond the tips of the fingers and toes. The legs are comparatively small and short, and curved with an outward bow (Fig. 1). This curvature, however, is only apparent, as is shown by the dissecting away of the muscles (Zschocke).[1] They are much of the time held flexed at the knees and drawn up to the abdomen as in the fetal position. Intellectual activity is practically absent and the power of the special senses largely in abeyance.

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[1] Jahrb. f. Kinderheilk, 1921, XCVI, 32.

DEVELOPMENT

A more detailed study is necessary of the characteristics of the new-born child and of its development as growth advances. These subjects will be considered in course.

Increase in Weight.—The weight of the normal healthy child at birth may be taken as from 7 to 7½ lbs. (3175 to 3402). (See Vol. II, pp. 858; 862 for abnormally great birth-weights.) The children of primiparæ are slightly lighter than those of multiparæ, the difference averaging about 5 oz. (142). Adair and Stewart[1] found the average birth-weight in 149 first-born infants 3310 grams (7.29 lbs.) and that of an equal number of later born infants 3502 grams (7.72 lbs.). Investigations by Dr. J. C. Gittings and myself upon 226 new-born infants gave an average initial birth-weight of 3455.79 grams (7.62 lb.). There is, however, a great range in the weight of the new-born even within physiological limits. This is shown by the variation in the statistics, as seen in the following table:

Table 1.—Average Weight at Birth

Kézmársky[2]	73 cases	Average 3330 grams (7.34 lbs.)
Ingerslev[3]	3450 cases	Average 3334 grams (7.35 lbs.)
Holt[4]	1158 cases	Average 3330 grams (7.34 lbs.)
Schäffer[5]	94 cases	Average 3151 grams (6.95 lbs.)
Camerer[6]	119 cases	Average 3433 grams (7.57 lbs.)
Peterson[7]	1675 cases	Average 3527 grams (7.78 lbs.)
Griffith and Gittings.[8]	226 cases	Average 3456 grams (7.62 lbs.)
Fuhrmann[9]	1000 cases	Average 3337 grams (7.36 lbs.)
Benestad[10]	1979 cases	Average 3466 grams (7.64 lbs.)
Bachman[11]	511 cases	Average 3190 grams (7.03 lbs.)

Male children are somewhat heavier than female, the difference being about 100 to 200 grams (3.5 to 7 oz.). In the observations of Gittings and myself 111 males averaged 3494.06 grams (7.70 lb.), and 115 females 3418.08 grams (7.54 lb.). Some of the statistics illustrating the differences between the sexes are seen in the following table:

[1] Jour. Amer. Med. Assoc., 1922, LXXVIII, 1865.
[2] Arch. f. Gynäk., 1873, V, 547.
[3] Nord. Med. Ark., 1875, VII, No. 7, 8.
[4] Dis. of Inf. and Childh., 1911, 16.
[5] Arch. f. Gynäk., 1896, LII, 282.
[6] Jahrb. f. Kinderheilk, 1901, LIII, 413.
[7] Upsala läkaref. förhandl., 1882, XVIII, 15.
[8] Arch. of Pediat., 1907, XXIV, 321.
[9] Med. Klinik, 1907, III, 510.
[10] Arch. f. Gynäk., 1913–14, CI, 292.
[11] Amer. Jour. Dis. Child., 1923, XXVI, 349.

TABLE 2.—BIRTH-WEIGHT OF MALE AND OF FEMALE INFANTS RESPECTIVELY

	Males	Females
Ingerslev[1]	3381 grams (7.45 lbs.)	3280 grams (7.23 lbs.)
Gregory[2]	3386 grams (7.46 lbs.)	3331 grams (7.34 lbs.)
Quetelet[3]	3200 grams (7.05 lbs.)	2910 grams (6.41 lbs.)
Altherr[4]	3214 grams (7.08 lbs.)	3077 grams (6.78 lbs.)
Kézmársky[5]	3383 grams (7.46 lbs.)	3284 grams (7.24 lbs.)
Holt[6]	3400 grams (7.49 lbs.)	3260 grams (7.19 lbs.)
Peterson[7]	3595 grams (7.92 lbs.)	3455 grams (7.62 lbs.)
Griffith and Gittings[8]	3494 grams (7.70 lbs.)	3418 grams (7.54 lbs.)
Ramsey and Alley[9]	3391 grams (7.48 lbs.)	3276 grams (7.22 lbs.)
Taylor[10]	3484 grams (7.53 lbs.)	3377 grams (7.44 lbs.)

Immediately after birth the loss of weight begins in nearly all cases. This depends upon the excretion of meconium and urine, the removal of the vernix caseosa, the excretions of the skin and lungs, the vomiting of allantoic fluid, and the metabolic changes progressing in the tissues. The loss of weight due to metabolic changes may be spoken of as physiological, and that from other causes classed as mechanical. The chief loss of a metabolic nature is water from the skin and tissues (Birk and Edelstein;[11] Rott;[12] Benestad;[13] Bailey and Murlin;[14] Bakwin;[15] Coda[16]). In addition there is a loss of body-tissue due to the oxidation of fat and of a small amount of protein and carbohydrate (Benedict and Talbot;[17] Bakwin[18]). The weight of meconium, urine and vernix ranges from 3 to 5 oz. (85 to 142) according to the observations of Townsend.[19] A review made by Fleischmann[20] of the results obtained by a number of observers showed the total loss to average 222 grams (7.83 oz.); *i.e.*, about 1/15 (6.66 per cent.) of the whole weight if we assume this as 3300 grams (7.28 lb.). Certain other observations, however, give a loss somewhat greater than this as normal. In an examination of 226 infants made by Gittings and myself[21] the total average loss equalled about 11 oz. (312), the relative loss being approximately 1/11 (9 per cent.) of the initial weight. It is usually accepted that the heavier the full-term baby, the greater will be the absolute loss of weight and the slower the regain. It would appear from the studies of Longridge[22] that the relative loss is also greater. Thus in 400 infants examined by him, the 7-lb. children lost $\frac{1}{14}$ of their body-weight, and the 5-lb. children only $\frac{1}{16}$. However, Pies'[1] studies and those of Bergmann[2] do not show any relatively greater loss in the heavier infants, and Kirstein[3] in a study of 1768 new-born infants observed an average loss of 7.8 per cent. of the initial weight, whether this were light or heavy. Adair and Stewart[4] found that first-born infants lost a larger percentage of their total body-weight (8 per cent.) than later infants (6.4 per cent.). This was due, in part at least, to the fact that the infants of multiparæ received larger feedings than those of primiparæ. The loss of weight is, as stated, chiefly physiological, and therefore cannot usually be entirely prevented. Observations carried out by Gittings and myself[5] on 61 infants confirmed the results of Cramer[6] and others, that by wet-nursing from the beginning until the mother's secretion is established, the initial loss of weight could be reduced very considerably, yet that this offered no advantage. Studies by Schick[7] gave similar results, except that it was found that forced feeding was often necessary, merely putting the infant to the breast not being sufficient. It has also been recommended by a number of writers, as by Adair and Stewart,[8] Benedict and Talbot,[9] Bailey and Murlin,[10] Williamson[11] and Bachmann[12] that artificial feeding be commenced in the first days of life, using solutions of lactose or glucose or milk-mixtures, in the effort to prevent the loss of weight; but, as with early wet-nursing, this does not seem to be required in healthy infants. Keilmann[13] found that when artificial feeding was commenced immediately after birth, the infants, although losing somewhat less, regained the initial weight more slowly than did those who had been nourished solely at the mother's breast. It should be said, however, that Bachmann's[14] observations in 511 infants do not confirm this view, those who were fed artificially in the first few days of life regaining the original weight more promptly than did the others. Faber[15] finds artificial feeding unnecessary, because the colostrum on the 2nd and 3rd days will supply the caloric needs

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[1] *Loc. cit.*

[2] Arch. f. Gynäk., 1871, II, 52.

[3] Sur l'homme et le dévelopment, etc., 1836, II, 49. Ref. Fleischmann, Wien. Klinik, 1877, June and July.

[4] Ueber regelmässige Wägung der Neugeborenen, 1874.

[5] *Loc. cit.*

[6] *Loc. cit.*

[7] *Loc. cit.*

[8] *Loc. cit.*

[9] Amer. Jour. Dis. Child., 1918, XV, 408.

[10] Amer. Jour. Dis. Child., 1919, XVII, 353.

[11] Monatsschr. f. Kinderheilk., 1910–1911, IX, 505.

[12] Zeitschr. f. Kinderheilk., 1910–1911, I, 43.

[13] Jahrb. f. Kinderheilk., 1914, LXXX, 21.

[14] Amer. Jour. Obst., 1915, LXXI, 526.

[15] Amer. Jour. Dis. Child., 1922, XXIV, 497.

[16] La Pediat., 1923, XXXI, 709.

[17] Carnegie Institute of Washington, 1915, Pub. No. 233, 10; 11.

[18] *Loc. cit.*

[19] Bost. Med. and Surg. Jour., 1887, CXVI, 157.

[20] *Loc. cit.*

[21] *Loc. cit.*

[22] Brit. Jour. Child. Dis., 1905, II, 403.

The diminution of weight continues until the 3d or 4th day and sometimes even longer, but cannot in the latter event be called physiological. The greatest loss takes place on the 1st day, from 3.5 to 4 per cent., and the next upon the 2d day, 2 to 2.6 per cent. of the birth-weight. In the cases of Adair and Stewart,[16] however, the greatest loss was on the 2nd day, being 44 per cent. of the total loss. The 3d day shows usually but little loss or the beginning of gain. The original weight is not regained before the 8th or 9th day, and often not until the 14th day. In a study upon 600 infants by Schulz[17], 228, or 48 per cent., had regained their original weight in 10 days, illness of a number of the children preventing an earlier average time of regain. On the other hand, in 611 cases recorded by Bergmann[1] only 21.17 per cent. regained their birth-weight by the end of the 2nd week, and in the 300 cases of Ramsey and Alley[2] only 25 per cent. by the 10th day.

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[1] Monatsschr. f. Kinderheilk., Orig., 1910, IX, 514.
[2] Zeitschr. f. Kinderheilk., Orig., 1916, XIV, 149.
[3] Zeitschr. f. Geburtsh. u. Gynäkol., 1918, LXXX, 448.
[4] Jour. Amer. Med. Assoc., 1922, LXXVIII, 1865.
[5] *Loc. cit.*
[6] Münch. med. Wochenschr., 1900, XLVII, 1585.
[7] Zeitschr. f. Kinderheilk., Orig., 1915, XIII, 257.
[8] *Loc. cit.*
[9] *Loc. cit.*
[10] *Loc. cit.*
[11] Arch. of Pediat., 1923, XL, 253.
[12] Amer. Jour. Dis. Child., 1923, XXVI, 349.
[13] Jahrb. f. Kinderheilk., 1896, XLI, 312.
[14] *Loc. cit.*
[15] Amer. Jour. Dis. Child., 1922, XXIV, 56.
[16] *Loc. cit.*
[17] Inaug. Dissert. Greifswald, 1903. Ref. Arch. f. Kinderheilk., 1904, XXXIX, 207.

Published statistics regarding the influence of sex are contradictory, some showing a greater loss in males, others in females. In our own[3] studies upon 105 infants, the average loss in boys was somewhat greater and the regain of the initial weight slower in being accomplished. Townsend[4] noticed that children of primiparæ lose about 1½ oz. (42) more than those of multiparæ.

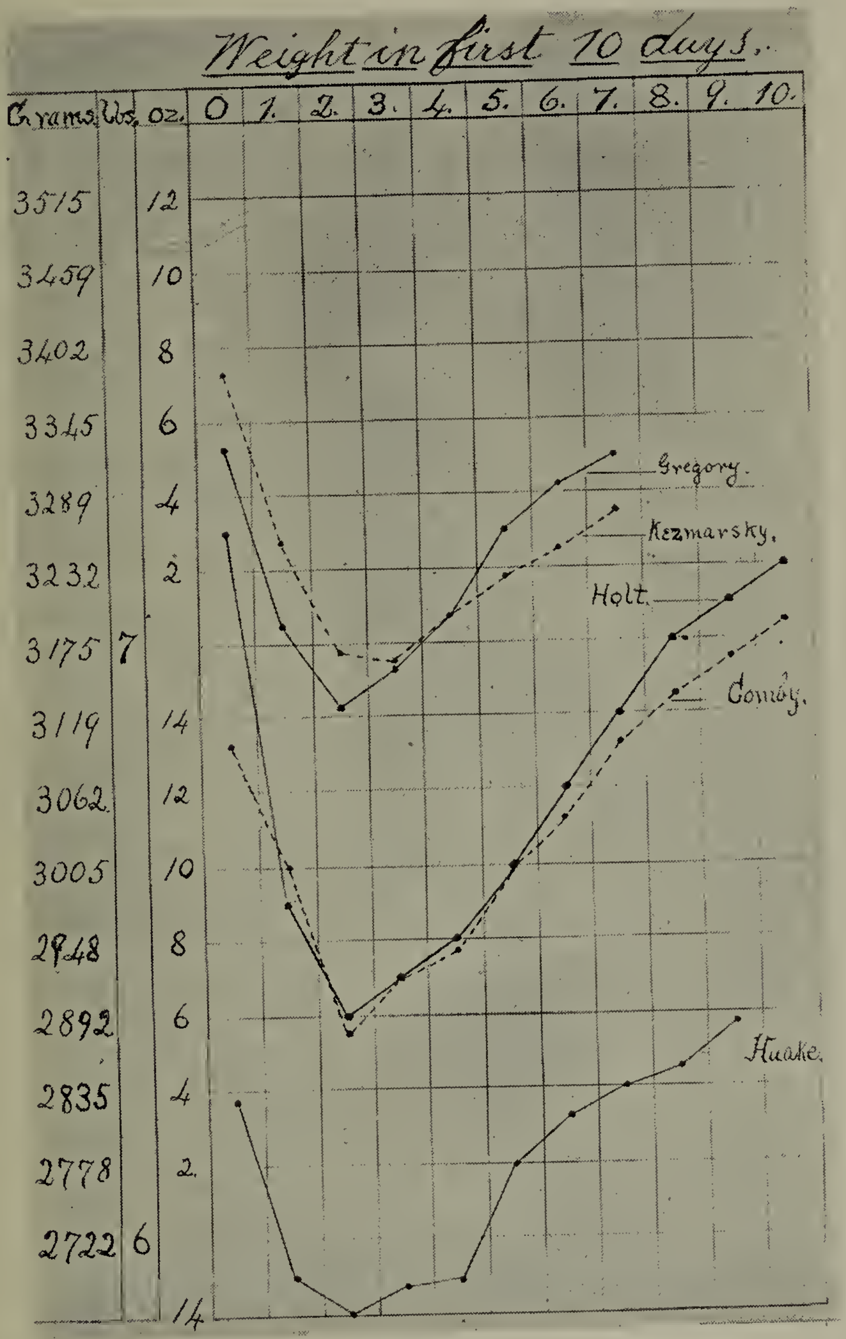

Fig. 2.—Graphic Curves Showing Gain and Loss of Weight in the First 10 Days of Life According to Different Observers.

(*Griffith, New York Med. Jour.*, 1899, *March* 4.)

The accompanying chart[5] (Fig. 2.) shows the loss and regain of weight in the first 10 days according to the observations of a number of investigators.

[1] *Loc. cit.*

[2] *Loc. cit.*

[3] Griffith and Gittings, *loc. cit.*, 336.

[4] *Loc. cit.*

[5] New York Med. Jour., 1899, March 4.

After gain in weight begins increase is rapid. Various estimations have been made of its rate, some of the more important of which are shown in graphic form in the illustration (Fig. 3).

Quetelet[1] assumed that growth continues at the same rate throughout the 1st year; hence his line in the chart is a *straight* one. Later investigations show that the rate of growth progressively decreases, this factor making the graphic plotting a *curved* line.

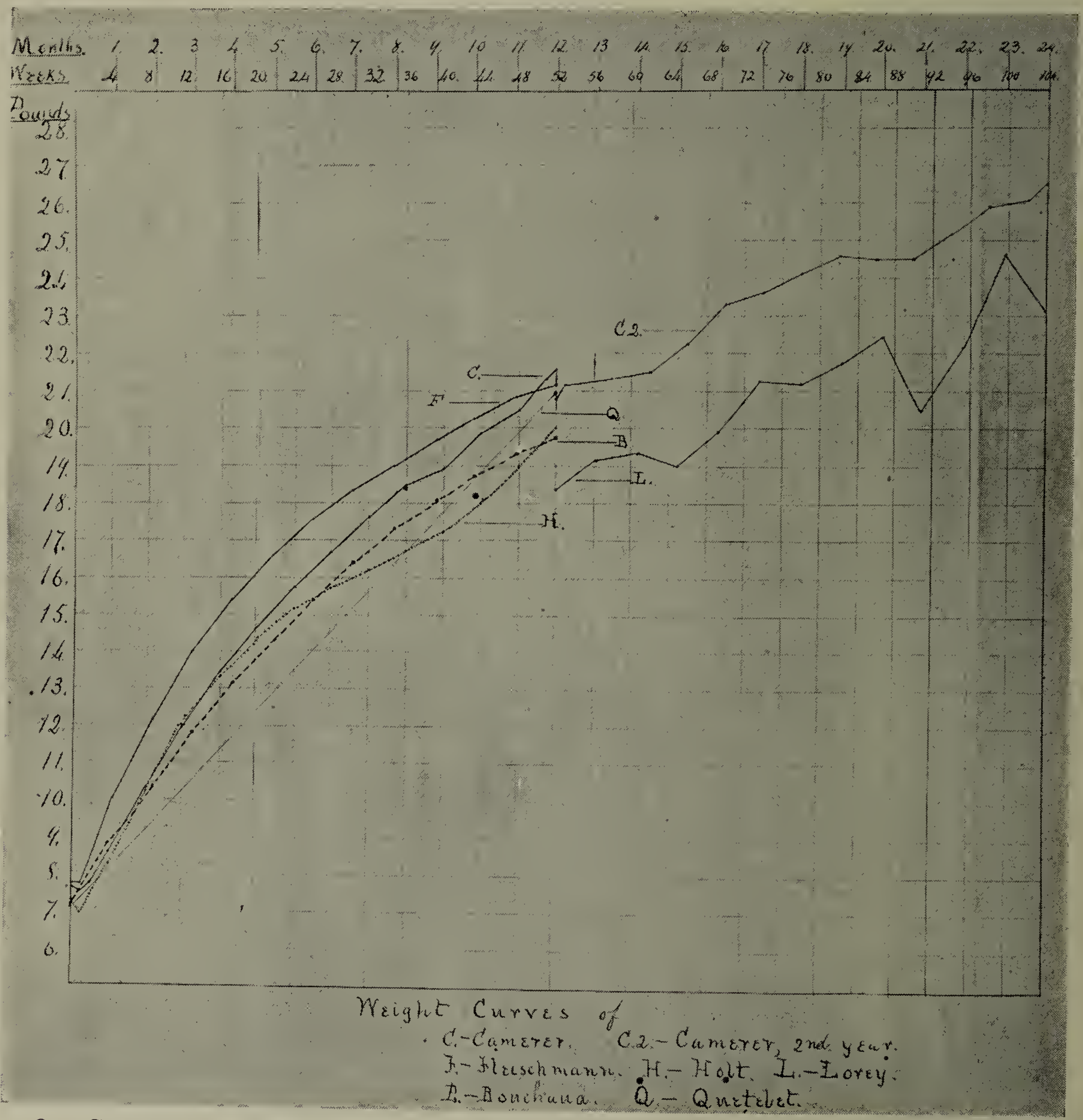

FIG. 3.—GRAPHIC CURVES SHOWING THE NORMAL GAIN IN WEIGHT DURING THE 1ST AND 2D YEARS ACCORDING TO SEVERAL OBSERVERS.
(*Griffith, New York Med. Jour.*, 1899, *March* 4.)

It must be remembered that all tables of weight represent only the *average* rate of growth. A considerable variation in the individual child within physiological limits is possible.

The following figures give approximate weights of well-developed breast-fed children during the 1st year of life. They follow to a considerable extent the investigations of Camerer[2] and start with a birth-weight somewhat higher, viz., 3400 grams (7½ lbs.), than that given by many investigators.

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[1] Ref. Fleischmann, *loc. cit.*
[2] Jahrb. f. Kinderheilk., 1893, XXXVI, 249.

Table 3.—Showing Increase in Weight During the First Year

Age	Weight	
Birth	7 lbs. 8 oz.	(3402)
1 week	7 lbs. 7½ oz.	(3388)
2 weeks	7 lbs. 10½ oz.	(3473)
3 weeks	8 lbs. 2 oz.	(3685)
1 month	8¾ lbs.	(3969)
2 months	10¾ lbs.	(4876)
3 months	12¼ lbs.	(5557)
4 months	13¾ lbs.	(6237)
5 months	15 lbs.	(6804)
6 months	16¼ lbs.	(7371)
7 months	17¼ lbs.	(7824)
8 months	18¼ lbs.	(8278)
9 months	18¾ lbs.	(8505)
10 months	19¾ lbs.	(8958)
11 months	20½ lbs.	(9299)
1 year	21½ lbs.	(9752)

Analyzing these figures it will be noticed that in the last 3 weeks of the 1st month and during the 2d month the gain in weight is about 1 oz. (28) a day, 7 oz. (198) a week; in the 3d and 4th months slightly over ¾ of an oz. (21) a day, 5½ oz. (156) a week; in the 5th and 6th months ⅔ of an oz. (19) a day, 4⅔ oz. (132) a week, and in the remainder of the 1st year about ½ oz. (14) a day, 3¾ oz. (106) a week, or 1 lb. (454) a month; except that somewhere about the 9th month there is liable to be a temporary diminution in the rapidity of increase.

The initial average weight is doubled at the age of 5 months and nearly trebled at the age of 1 year. Raudnitz[1] has formulated a certain law according to which the increase in weight shall bear a certain definite mathematical relation to the age. The application of the rule is, however, not very practical.

The weight of males during the 1st year is on the average somewhat greater than that of females, amounting at the age of a year to a difference of ½ up to even 1¼ lb. (227 to 567). Children fed artificially generally gain weight less rapidly than those at the breast, but this is by no means an invariable rule. This difference in weight depending on the food may persist during the first 3 or 4 years, but very often a healthy infant artificially fed and thriving on its diet, even although temporarily behind, will equal the breast-fed child in weight by the end of the 1st year. Allen and Bagg[2] made an analysis of 500 cases showing that infants who at birth are over the average weight continue to gain better than do others. The babies of multiparæ gain better than do those of primiparæ.

There are fewer extended observations made upon the weight of children from the 1st to the 6th year. Those of Camerer[3] appear reliable. The weights obtained by him are as follows:

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[1] Prag. med. Wochenschr., 1892, Nos. 7 and 8.
[2] Bost. Med. and Surg. Jour., 1923, CLXXXIX, 349.
[3] Jahrb. f. Kinderheilk., 1901, LIII, 418.

Table 4.—Increase in Weight from the Second to the Fifth Year of Life Inclusive

(Camerer)

12 months	10,030 grams (22.11 lbs.)
13 months	10,220 grams (22.53 lbs.)
14 months	10,600 grams (23.36 lbs.)
15 months	10,870 grams (23.96 lbs.)
16 months	10,900 grams (24.03 lbs.)
17 months	11,450 grams (25.24 lbs.)
18 months	11,480 grams (25.30 lbs.)
19 months	11,850 grams (26.12 lbs.)
20 months	12,050 grams (26.56 lbs.)
21 months	11,950 grams (26.34 lbs.)
22 months	12,220 grams (26.80 lbs.)
23 months	12,480 grams (27.51 lbs.)
2 years	12,740 grams (28.08 lbs.)
3 years	14,930 grams (32.91 lbs.)
4 years	16,410 grams (36.16 lbs.)
5 years	18,710 grams (41.25 lbs.)

The studies of Perret and Planchon[1] give figures about 100 grams (3.53 oz.) less than these. Those of Freeman[2] upon 278 well cared for children in private practice gave results decidedly higher, as is to be expected. In general it may be assumed that the average healthy infant gains in its 2d year from 5 to 6 lbs. (2268 to 2722); *i.e.*, ½ lb. (227) a month or ¼ oz. (7) a day, the increase in the 1st half of the year being greater than in the 2d half. (See chart, p. 14.) In the 3d and 4th years the child gains about 5 lbs. (2268) and in the 5th year about 4 lbs. (1814). In tabular arrangement the figures read as follows:

Table 5.—Gain in Weight from One to Five Years

End of 1st year weighs 21½	lbs. (9,752)	
End of 2d year weighs 27	lbs. (12,247)	Gained 5½ lbs. (2495)
End of 3d year weighs 32	lbs. (14,515)	Gained 5 lbs. (2268)
End of 4th year weighs 37	lbs. (16,783)	Gained 5 lbs. (2268)
End of 5th year weighs 41	lbs. (18,597)	Gained 4 lbs. (1814)

Girls continue to be from 1 to 1½ lbs. (454 to 680) lighter than boys to the end of this period.

The following table shows the mean rate of increase for the two sexes according to the observations of Camerer:[3]

Table 6.—Gain in Weight of Boys and Girls Respectively

(Camerer)

	Boys	Girls
1st year	10,310 grams (22.73 lbs.)	9,460 grams (20.86 lbs.)
2d year	13,210 grams (29.12 lbs.)	12,010 grams (26.48 lbs.)
3d year	15,460 grams (34.08 lbs.)	13,970 grams (30.78 lbs.)
4th year	16,810 grams (37.06 lbs.)	15,720 grams (34.66 lbs.)
5th year	19,300 grams (42.59 lbs.)	17,540 grams (38.67 lbs.)

A large number of measurements made upon 167,024 white children without clothing, from early infancy to the age of 6 years, and in different parts of the United States, were collected and analyzed by the Children's

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[1] L'Obstetrique, 1904, IX, 193.
[2] Amer. Jour. Dis. Child., 1914, VIII, 321
[3] *Loc. cit.*

Bureau,[1] only those children being selected who were free from disease and apparently as nearly normal as possible. A condensation of the results taken from the published table reads as follows:

Table 7.—Weight and Height from One to Six Years

Age	Boys				Girls			
	Height		Weight		Height		Weight	
	In.	Cm.	Lbs.	Gm.	In.	Cm.	Lbs.	Gm.
12 mos. (under 13 mos.)....	29½	74.93	21⅜	9695.53	28⅞	73.34	20	9071.84
24 mos. (under 25 mos.)....	33⅝	85.40	26⅝	12076.89	33⅛	84.14	25⅛	11396.49
36 mos. (under 37 mos.)....	36⅝	93.02	30¾	13947.95	36¼	92.07	29½	13380.96
48 mos. (under 49 mos.)....	39¼	99.69	34½	15648.92	38⅞	98.74	33⅛	15025.23
60 mos. (under 61 mos.)....	41⅝	105.72	38⅛	17293.19	41⅜	105.09	36⅞	16726.20
71 mos. (under 72 mos.)....	43⅞	111.44	41⅝	18880.77	43⅝	110.80	40½	18370.48

Estimating roughly from different sources the combined average weight of the sexes we find children gaining about 4 lbs. (1814) in the 6th and 7th years, 4½ lbs. (2041) in the 8th, 5 lbs. (2268) in the 9th, 5½ to 6 lbs. (2495 to 2722) in the 10th and 11th years, 7 to 8 lbs. (3175 to 3629) in the 12th year, and then from 9 to 10 lbs. (4082 to 4536) per year to the age of 16 years. In tabular form the approximate weight for the different years according to this rate of increase reads as follows:

Table 8.—Weight from Six to Sixteen Years

6 years....................................	45 lbs. (20,412)
7 years....................................	49 lbs. (22,226)
8 years....................................	53½ lbs. (24,267)
9 years....................................	58½ lbs. (26,535)
10 years....................................	64 lbs. (29,030)
11 years....................................	70 lbs. (31,751)
12 years....................................	78 lbs. (35,380)
13 years....................................	87 lbs. (39,463)
14 years....................................	96 lbs. (43,549)
15 years....................................	106 lbs. (48,080)
16 years....................................	116 lbs. (52,617)

It will be seen that the child nearly doubles its normal weight at 1 year by the age of 5 years, and trebles it by 10 years.

The difference, however, which the sexes show in the rate of increase in weight is so decided in later childhood and in youth, that a separate consideration is necessary. The rate of growth continues about the same in each sex until about the age of 8 or 9 years, at which time the increase in girls is somewhat retarded, and boys advance decidedly beyond them. By 11 years, however, girls begin to gain in weight rapidly, and by 12 years decidedly surpass boys. This continues until the age of 15 or 16 years when boys again take and maintain the lead. The relative weights and rates of increase in boys and girls respectively during this period, according to the statistics of Bowditch,[2] Porter[3] and Peckham,[4] can be learned from the accompanying table. The figures of Bowditch are based upon 24,595 observations upon public school-children of Boston; those of Porter upon 30.817 children of St. Louis, and those of Peckham upon 5403 children of Milwaukee. It should be stated, however, that there appears to be a variation in different countries for the age at which boys finally surpass girls in weight, European statistics differing somewhat from American.

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[1] U. S. Dept. of Labor. Community Child-Welfare, 1921, Series No. 2, Bureau Pub. No. 84.

[2] 8th Ann. Rep. Mass. State Board of Health, 1877, 275.

[3] Transac. Acad. Science, St. Louis, VI, No. 12, 312.

[4] 6th Ann. Rep. State Board of Health, Wisconsin, 1881, p. 28.

TABLE 9.—WEIGHT OF BOYS AND GIRLS FROM FIVE TO EIGHTEEN YEARS

Years	Bowditch				Porter				Peckham			
	Boys		Girls		Boys		Girls		Boys		Girls	
	Pounds	Kilograms	Pounds	Kilograms	Pounds	Kilograms	Pounds	Kilograms	Pounds	Kilograms	Pounds	Kilograms
5	41.09	18.64	39.66	17.99	.....	.....	.....	.....	41.09	18.64	40.03	18.16
6	45.17	20.49	43.28	19.63	43.54	19.75	41.74	18.93	44.81	20.33	43.12	19.56
7	49.07	22.26	47.46	21.10	47.77	21.67	45.90	20.82	49.10	22.27	46.97	21.21
8	53.92	24.46	52.04	23.44	52.42	23.78	50.44	22.88	53.81	24.41	50.87	23.07
9	59.23	26.87	57.07	25.91	57.45	26.06	55.29	25.08	59.46	26.97	56.44	25.60
10	65.30	29.62	62.35	28.29	62.43	28.32	60.60	27.49	65.35	29.64	62.45	28.33
11	70.18	31.84	68.84	31.23	68.34	31.00	66.47	30.15	70.92	32.17	68.84	31.22
12	76.92	34.89	78.31	35.53	73.73	33.51	74.21	33.66	76.08	34.51	77.82	35.30
13	84.84	38.49	88.65	40.21	80.71	36.61	84.85	38.49	84.89	38.50	87.96	39.90
14	94.91	42.95	98.43	44.65	89.15	40.44	93.23	42.29	95.76	43.43	97.64	44.29
15	107.10	48.59	106.08	48.12	101.90	46.22	102.93	46.69	109.05	49.46	105.87	48.02
16	121.01	54.90	112.03	50.81	113.76	51.60	110.78	50.25	122.06	55.36	110.58	50.16
17	127.49	57.84	115.53	52.41	122.73	55.67	115.98	52.61	130.35	59.12	113.32	51.40
18	132.55	60.13	115.16	52.24	......	.....	......	.....	137.76	62.49	112.48	51.02

The figures given (Table 9) are those for children of 5 years and over including clothing. Those for younger periods (p. 10) are without clothing. The average weight of the clothes, as estimated by Schmidt-Monnard[1] is 6 per cent. to 7 per cent. of the weight of the body at the age of from 3 to 6 years, and 7 per cent. to 8 per cent. from 6 to 14 years. According to a series of personal estimations made on over 200 children, the average weight of the clothes in the 1st year is approximately 1¾ lbs. (794); from 1 to 4 years, 2 lbs. (907); and from 4 to 6 years, 2¼ to 2½ lbs. (102 to 113). The ratio of the weight of the clothing to the gross weight of the normal child dressed is approximately 16 per cent. in the first 3 months, but after this during the first 2 years approximately 8 per cent. to 10 per cent.; and from 2 to 5 years 6 per cent. The weight of the clothing in the first 2 years, according to the studies of Sauer[3] is decidedly less than my own figures. Bowditch[4] estimates the weight of the clothes at 5 years for boys as 2.8 lbs. (1270), equalling 6. 45 per cent. of the total gross weight for boys, and for girls as 2.48 lbs. (1288) or 6.79 per cent. of the total weight for that sex; at 8 years 4 lbs. (1814) or 7.23 per cent. for boys and 3.5 lbs. (1587) or 6.54 per cent. for girls; at 11 years 6.7 lbs. (3039) or 9.88 per cent. for boys, and 4.9 lbs. (2223) or 6.88 per cent. for girls; at 14 years 8.1 lbs. (3674) or 8.15 per cent. for boys and 7.5 lbs. (3402) or 6.76 per cent. for girls.

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[1] Jahrb. f. Kinderheilk., 1901, LIII, 53.

[2] Griffith, New York Med. Jour., 1917, CVI, 823.

[3] Amer. Jour. Dis. Child., 1919, XVIII, 20.

[4] *Loc. cit.*, 306.

The influence of any *illness* upon the increase of weight is very decided, especially in infancy. Growth ceases, and weight may even be rapidly lost, depending upon the nature of the disease. There appears to be a positive influence of *season* upon the growth of children. The investigations of Malling-Hansen[1] upon children of from 9 to 17 years showed the most rapid increase occurring from August to December. Very similar conditions were observed by Holt[2] and by Porter.[3] Schmidt-Monnard[4] confirms this for even younger children from the age of 2½ years and even in the 2d year, and Bleyer[5] found it true even of the 1st year of life.

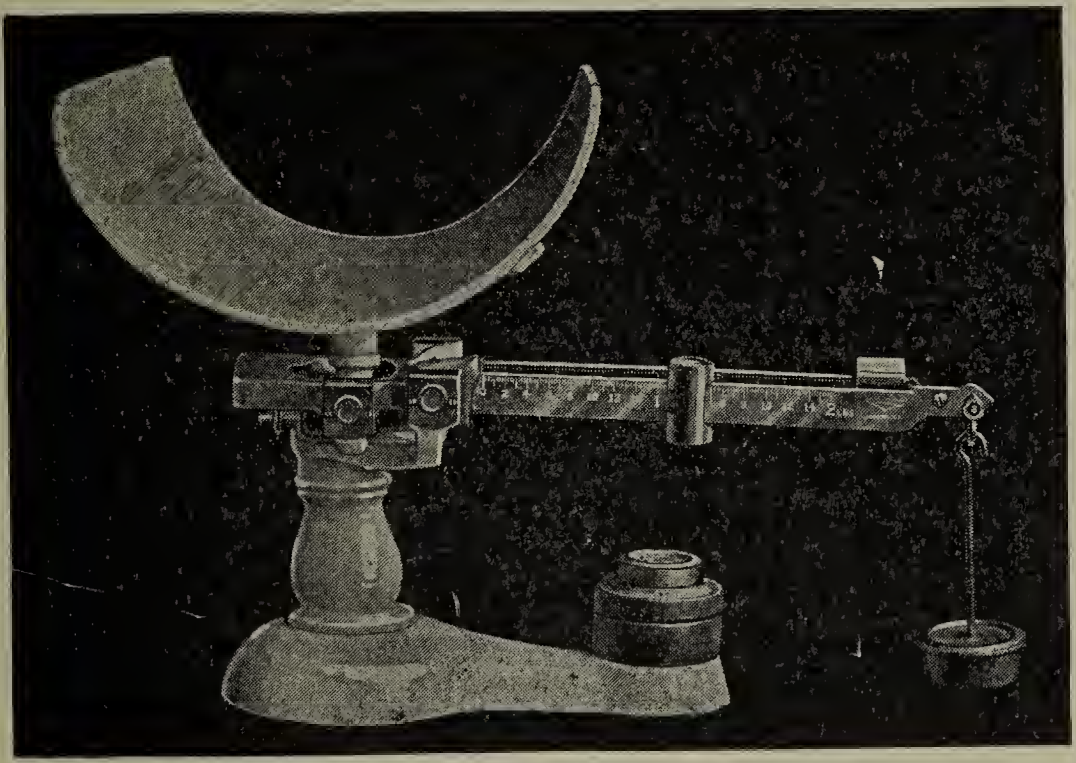

Fig. 4.—The "Mead" Baby Scales.

Indeed the influence of the season may extend so far that, as claimed by Stepanoff,[6] children of the school-age who were born in summer are larger than those born in winter. Malling-Hansen also called attention to the daily fluctuations, with an increase in weight and a decrease in height during the day and a reversal of these during the night. Coerper[7] classifies children according to their build. He quotes Sigaud who groups children into the digestive, the respiratory, and the muscular types, degrees of nutrition being compared only in each group. In addition to these factors nationality, climate, environment, social status, exercise, and, of course, food, affect growth (Putnam).[8] Hence it is that in the United States the problem of constructing a reliable standard weight-curve is greatly complicated (Robertson).[9]

[1] Perioden in Gewicht, etc., Copenhagen, 1886. Ref. Vierordt's Daten u. Tabellen, 1906, 25.

[2] Amer. Jour. Dis. Child., 1918, XVI, 359.

[3] Amer. Jour. Physiol., 1920, LII, 121.

[4] *Loc. cit.*

[5] Arch. of Pediat., 1917, XXXIV, 366.

[6] Thèse de Lausanne. Ref. Monatsschr. f. Kinderheilk., 1903, II, 242.

[7] Zeitschr. f. Kinderheilk., 1922, XXXIII, 144.

[8] Arch. of Pediat., 1922, XXXIX, 71.

[9] Abt's Pediatrics, 1923, I, 487.

The importance of weighing children regularly during the first 2 years of life, and especially during the 1st year, cannot be over-estimated, since cessation of gain is often the first sign of illness or of a lack of sufficient nourishment. The child should be weighed at least weekly, divested of clothing, or dressed and the weight of the clothes subsequently deducted. Scales should be used which indicate ounces. Standing spring scales

Designed by J. P. Crozer Griffith, M. D.,
Professor of Pediatrics in the University of Pennsylvania.

INFANTS WEIGHT-CHART.

Name.......................... Date of Birth..................

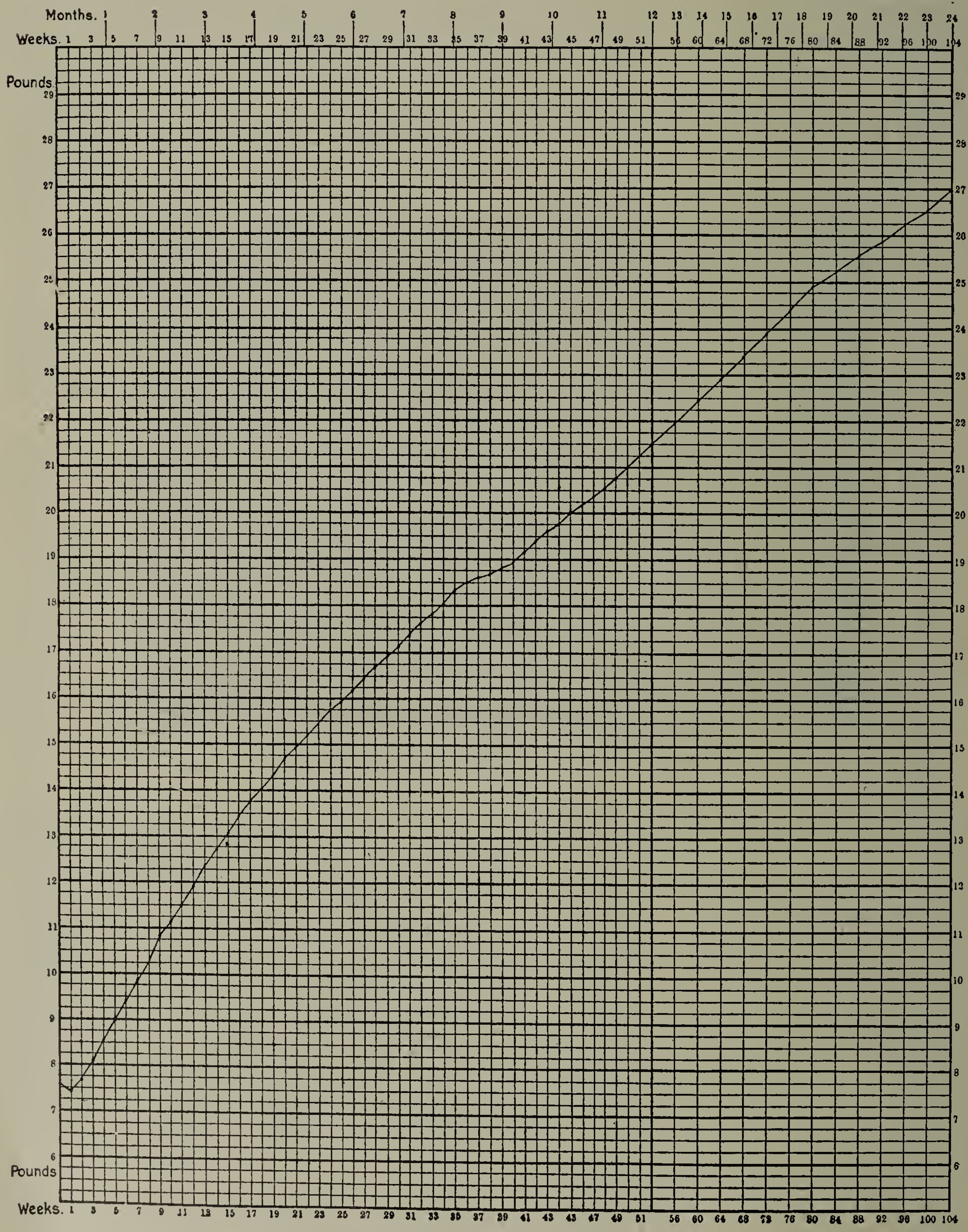

Fig. 5.—Author's Weight-Chart for the First 2 Years of Life. Reduced size. (*New York Med. Jour.*, 1899, *March* 4.)

with a scoop or basket attached are very convenient, but some form of balance scale is more accurate (Fig. 4). For recording the results a *weight-chart* is a great convenience. The illustration (Fig. 5) is a reduced reproduction of one which I have employed for a number of years.[1]

Infant's Weight-Chart

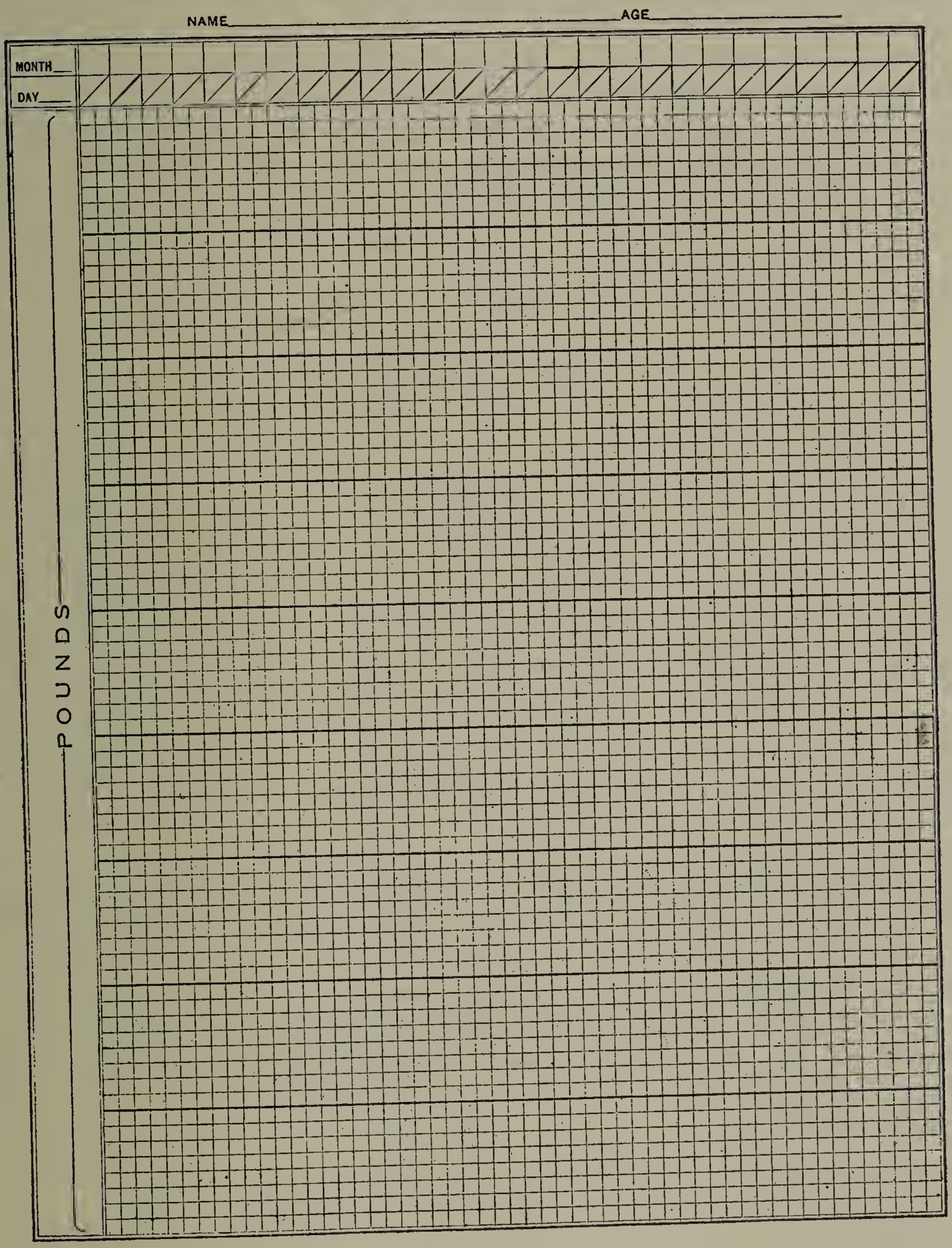

Fig. 6.—Daily Weight-Chart.

To be used for daily weighings at any period of infancy. As employed in the Children's Hospital and in the Children's Medical Ward of the University Hospital, Philadelphia. Reduced size.

The curve already plotted upon it represents the normal average gain in weight for a healthy breast-fed infant. To economize space the portion for the 2d year is narrowed one-half, which necessarily distorts the line. A very convenient form for recording daily weighings is that employed at The Children's Hospital, Philadelphia, of which a reduced reproduction is shown (Fig. 6).

[1] W. B. Saunders Co. For sale singly by E. Pennock, 3609 Woodland Ave., Phila.

Length.—Estimations upon growth in length in the 1st year have not been nearly so numerous as those upon weight, and the results of different observations show decided variations, since accurate determination requires much care. The average length at birth may be assumed as 49.5 to 50 cm. (19.5 to 20 inches), with males about 0.5 to 1 cm. (0.2 to 0.4 inch) longer than females. Gain in length goes on rapidly but with decreasing speed. Thus in the first 3 months, according to Camerer,[1] the gain is 9 cm. (3.5 inches), in the next 3 months 8 cm. (3.2 inches), and in the third and fourth 3-month periods 3 to 5 cm. (1.2 to 2 inches), making a total gain of 24 cm. (9.4 inches) for the year. These figures are somewhat in excess of certain other statistics. Heubner[2] gives the following table for average figures taken from different investigators):

TABLE 10.—GAIN IN LENGTH IN BOYS AND GIRLS

(Heubner)

	Boys	Girls
Birth	51.0 cm. (20.1 in.)	49.0 cm. (19.3 in.)
1 month	52.5 cm. (20.7 in.)	51.3 cm. (20.2 in.)
2 months	55.3 cm. (21.8 in.)	54.8 cm. (21.6 in.)
3 months	57.8 cm. (22.7 in.)	56.7 cm. (22.3 in.)
4 months	60.1 cm. (23.7 in.)	58.7 cm. (23.1 in.)
5 months	61.3 cm. (24.1 in.)	60.2 cm. (23.7 in.)
6 months	62.6 cm. (24.6 in.)	61.5 cm. (24.2 in.)
7 months	64.6 cm. (25.4 in.)	63.2 cm. (24.9 in.)
8 months	65.6 cm. (25.8 in.)	64.3 cm. (25.3 in.)
9 months	67.8 cm. (26.7 in.)	65.4 cm. (25.8 in.)
10 months	67.0 cm. (26.4 in.)	67.2 cm. (26.5 in.)
11 months	69.0 cm. (27.2 in.)	68.1 cm. (26.8 in.)
12 months	70.3 cm. (27.7 in.)	69.2 cm. (27.2 in.)

As the table shows, the length of male children continues slightly in excess of that of females, the differences at the end of the year equalling about 1 cm. (0.4 inch).

During the periods covered by the ages from 1 to 5 years the gain in the 2d year of life is about 10 cm. (3.9 inches); in the 3d, 9 cm. (3.5 inches) and in the 4th and 5th years 7 cm. (2.8 inches). The following figures show this in tabular form, assuming the length at 1 year to be 28 inches (71 cm.):

TABLE 11.—GAIN IN LENGTH IN THE FIRST FIVE YEARS

1 year	28.0 in. (71 cm.)
2 years	32.0 in. (81 cm.)
3 years	35.5 in. (90 cm.)
4 years	38.25 in. (97 cm.)
5 years	41.0 in. (104 cm.)

The studies of Freeman[3] upon 278 well-cared-for children in private practice showed a length for the first 5 years decidedly above these figures. This depends doubtless on the patients being of a better social class. During this period the difference in length between the sexes continues, but does not increase. (See also Table 7, p. 11.)

[1] Jahrb. f. Kinderheilk., 1901, LIII, 425.

[2] Lehrb. d. Kinderheilk., 1911, I, 8.

[3] Amer. Jour. Dis. Child., 1914, VIII, 321.

From the age of 5 years onward the combined rate of increase in the two sexes is about 2 inches (5 cm.) a year until the age of 11 years, after which girls slightly exceed boys in height until the age of 15 years, when boys again take the lead. Increase in length is very slight in girls after they reach 16 years. The normal birth-length is doubled between the ages of 4 and 5 years and trebled at about 13 or 14 years.

The following table shows the gain in height in boys and girls respectively, according to the extensive observations of Bowditch,[1] Porter,[2] Peckham[3] and Variot and Chaumet.[4] Very similar statistics in both height and weight are given by Tuxford and Glegg.[5]

Growth in length exhibits certain definite seasonal relationships, analogous to, though differing from, those affecting that in weight (p. 13). The greatest increase in length, according to the studies of Schmidt-Monnard[6] in children from 2 to 13 years, takes place in July and August, and the least from September to February. Holt[7] found the greatest growth in boys of from 6 to 18 years of age take place from May to November. Increase in length is less influenced by illness than that in weight, although rachitic children are often unduly short.

As with weight, the season of the year at which birth takes place would appear to influence the later growth in length, the longest school children among those studied by Stepanoff[8] being those born in the summer.

TABLE 12.—LENGTH IN BOYS AND GIRLS FROM SIX TO EIGHTEEN YEARS OF AGE

Years	Bowditch				Porter				Peckham				Variot and Chaumet			
	Boys		Girls		Boys		Girls		Boys		Girls		Boys		Girls	
	Inches	Cm.	Inches	Cm.	Inches	Cm.	Inches	Cm.	Inches	Cm.	Inches	Cm.	Inches	Cm.	Inches	Cm.
5	41.57	105.6	41.29	104.9	.....	.....	.....	.....	42.28	107.39	41.72	105.97	40.68	103.3	40.11	101.9
6	43.75	111.1	43.35	110.1	42.92	108.94	42.42	107.67	44.08	111.96	43.78	111.20	43.26	109.9	42.87	108.9
7	45.74	116.2	45.52	115.6	44.93	114.03	44.50	112.95	46.09	117.07	45.93	116.66	45.04	114.4	44.81	113.8
8	47.76	121.3	47.58	120.9	46.94	119.13	46.63	118.36	48.05	122.05	47.59	120.88	47.13	119.7	47.05	119.5
9	49.69	126.2	49.37	125.4	48.99	124.35	48.73	123.67	50.00	127.00	49.81	126.52	49.21	125.0	49.09	124.7
10	51.68	131.3	51.34	130.4	50.77	128.87	50.60	128.43	51.85	131.70	51.89	131.80	51.37	130.3	50.98	129.5
11	53.33	135.4	53.42	135.7	52.73	133.84	52.48	133.19	53.76	136.55	53.80	136.65	52.60	133.6	52.91	134.4
12	55.11	140.0	55.88	141.9	54.45	138.21	54.81	139.11	54.98	139.65	56.47	143.43	54.17	137.6	55.71	141.5
13	57.21	145.3	58.16	147.7	56.31	142.91	57.73	146.53	57.47	135.97	58.68	149.05	57.13	145.1	58.51	148.6
14	59.88	152.1	59.04	152.3	58.54	148.58	59.43	150.84	59.89	152.12	60.50	153.67	60.56	153.8	60.19	152.9
15	62.30	158.2	61.10	155.2	61.03	154.90	61.09	155.04	62.34	158.34	61.59	156.44	62.84	159.6	60.71	154.2
16	65.00	165.1	61.59	156.4	63.15	160.27	62.06	157.52	65.07	165.28	62.16	157.89				
17	66.16	168.0	61.92	157.2	65.06	165.13	62.78	159.33	66.60	169.16	62.91	159.79				
18	66.66	169.3	61.95	157.3	66.46	170.41	62.81	159.42	67.44	171.30	62.53	158.83				

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[1] *Loc. cit.*

[2] *Loc. cit.*

[3] *Loc. cit.*

[4] Bul. soc. de péd. de Paris, 1906, Feb., 53.

[5] Brit. Med. Jour., 1911, I, 1423.

[6] Jahrb. f. Kinderheilk., 1895, XL, 84.

[7] Amer. Jour. Dis. Child., 1918, XVI, 359.

[8] *Loc. cit.*

Relation of Weight to Length.—In recent years much attention has been given by different investigators to the normal relationship of weight and height, in the effort to determine what should be considered abnormal, especially in the former. For the period up to the age of 6 years the following valuable table has been published by the Children's Bureau,[1] based upon the measurement of 167024 white children without clothes. This shows the normal relationship of height to weight at these ages.

Table 13.—Normal Relations of Height to Weight.

Height		Weight			
Inches	Cm.	Boys		Girls	
		Lbs.	Gm.	Lbs.	Gm.
20	50.80	8¼	3742.13	8⅛	3685.43
21	53.34	9½	4309.12	9¼	4195.63
22	55.88	10½	4762.72	10½	4762.72
23	58.42	12	5443.10	11⅞	5386.40
24	60.96	13½	6123.49	13⅜	6066.79
25	63.50	15	6803.88	14⅞	6747.18
26	66.04	16⅝	7540.97	16⅜	7427.57
27	68.58	18	8164.66	17⅝	7994.56
28	71.02	19⅜	8788.34	19	8618.25
29	73.66	20⅝	9355.33	20¼	9185.24
30	76.20	22	9979.02	21¼	9638.83
31	78.74	23¼	10546.01	22⅝	10262.52
32	81.28	24½	11113.00	23¾	10772.81
33	83.82	25¾	11679.99	25	11339.80
34	86.36	27⅛	12303.68	26⅜	11963.49
35	88.90	28⅜	12870.67	27¾	12587.18
36	91.44	29¾	13494.36	29⅛	13210.87
37	93.98	31⅛	14118.05	30⅜	13777.86
38	96.52	32½	14741.74	31¾	14401.55
39	99.06	33⅞	15365.43	33	14968.54
40	101.60	35¼	15989.12	34½	15648.92
41	104.14	36⅞	16726.20	36	16329.31
42	106.68	38⅜	17406.59	37½	17009.70
43	109.22	40⅛	18200.38	39¼	17803.49
44	111.76	41¾	18937.47	41	18597.27
45	114.30	43½	19731.25	42¾	19391.05
46	116.84	45⅜	20581.74	45	20411.64
47	119.38	47¼	21432.22	46⅞	21262.12

For periods after early childhood, one of the most extensive collections of statistics is that made by Wood,[2] based upon 250,000 observations of each sex, collected from many different sources. In the table published by him, the age is taken as the nearest birthday, the weight and length are with the clothing, but without shoes and with the coats of the boys removed. For measurements made in this way Wood[3] considers that 4 pounds (1814.36) should be allowed for the weight of the clothes of boys and 3 pounds (1360.77) for those of girls. If shoes are worn, 1 inch (2.54 cm.) should be deducted to obtain the correct height according to the table.

[1] U. S. Dept. of Labor, Community Child-Welfare, 1921, Series No. 2, Bureau Publ. No. 84.

[2] Child Health Organization, 1920.

[3] Quoted by Gray and Jacomb, Amer. Jour. Dis. Child., 1921, XXII, 265.

The standard or normal weight is found where the horizontal column opposite the height crosses the vertical column under the age. Thus the standard weight for a boy of 57 inches and 13 years would be 84 lbs. The *average* weight will fall nearly in a line drawn obliquely in the table for boys from 36 to 176 lbs., and that for girls from 35 to 148 lbs.

The tables show that any figure in the vertical column for a certain age may be considered within the normal range for that age, the figure to be selected as the standard depending upon the height as read in the column on the extreme left. Height, although subject to variation is so to a much less degree than is weight, and can conveniently be assumed as the standard with which weight may be compared. It is not so much the actual weight which is of importance, as the relationship of this to the normal weight for the height. Emerson and Manny[1] conclude from a study of available data that a deficiency of over 7 per

TABLE 14.—HEIGHT AND WEIGHT TABLE FOR BOYS
(Wood)

Height Inches	5 Yrs.	6 Yrs.	7 Yrs.	8 Yrs.	9 Yrs.	10 Yrs.	11 Yrs.	12 Yrs.	13 Yrs.	14 Yrs.	15 Yrs.	16 Yrs.	17 Yrs.	18 Yrs.
39	35	36	37											
40	37	38	39											
41	39	40	41											
42	41	42	43	44										
43	43	44	45	46										
44	45	46	46	47										
45	47	47	48	48	49									
46	48	49	50	50	51									
47	....	51	52	52	53	54								
48	....	53	54	55	55	56	57							
49	....	55	56	57	58	58	59							
50	....	....	58	59	60	60	61	62						
51	....	....	60	61	62	63	64	65						
52	....	....	62	63	64	65	67	68						
53	....	....	....	66	67	68	69	70	71					
54	....	....	....	69	70	71	72	73	74					
55	....	....	....	....	73	74	75	76	77	78				
56	....	....	....	....	77	78	79	80	81	82				
57	....	....	....	....	....	81	82	83	84	85	86			
58	....	....	....	....	....	84	85	86	87	88	90	91		
59	....	....	....	....	....	87	88	89	90	92	94	96	97	
60	....	....	....	....	....	91	92	93	94	97	99	101	102	
61	....	....	....	....	....	....	95	97	99	102	104	106	108	110
62	....	....	....	....	....	....	100	102	104	106	109	111	113	116
63	....	....	....	....	....	....	105	107	109	111	114	115	117	119
64	....	....	....	....	....	....	....	113	115	117	118	119	120	122
65	....	....	....	....	....	....	....	....	120	122	123	124	125	126
66	....	....	....	....	....	....	....	....	125	126	127	128	129	130
67	....	....	....	....	....	....	....	....	130	131	132	133	134	135
68	....	....	....	....	....	....	....	....	134	135	136	137	138	139
69	....	....	....	....	....	....	....	....	138	139	140	141	142	143
70	....	....	....	....	....	....	....	....	....	142	144	145	146	147
71	....	....	....	....	....	....	....	....	....	147	149	150	151	152
72	....	....	....	....	....	....	....	....	....	152	154	155	156	157
73	....	....	....	....	....	....	....	....	....	157	159	160	161	162
74	....	....	....	....	....	....	....	....	....	162	164	165	166	167
75	....	....	....	....	....	....	....	....	....	....	169	170	171	172
76	....	....	....	....	....	....	....	....	....	....	174	175	176	177

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[1] Arch. of Pediat., 1920, XXXVII, 468.

cent. underweight for the height indicates malnutrition, whereas 20 per cent. overweight for the height is to be considered obesity. Of course a child who is much below the average both in weight and height is abnormal, although the ratio of weight to height may be normal in his case.

Even here a word of caution is to be uttered. There is so much variation in both weight and height, depending, among other factors, upon the race and upon the social position, that too much importance must not be attributed to the relationship, and especially to that between either of these and the age. Much has been written, too, upon the interrelationships of stem-length, sitting height, vital capacity, weight, and the like, and various formulæ and tables have been constructed to express these. (See Gray and Jacomb[1] and Gray[2]). It does not appear that for practical purposes these relationships are as yet superior to the commonly accepted one of height and weight.

Table 15.—Height and Weight Table for Girls
(Wood)

Height Inches	5 Yrs.	6 Yrs.	7 Yrs.	8 Yrs.	9 Yrs.	10 Yrs.	11 Yrs.	12 Yrs.	13 Yrs.	14 Yrs.	15 Yrs.	16 Yrs.	17 Yrs.	18 Yrs.
39	34	35	36											
40	36	37	38											
41	38	39	40											
42	40	41	42	43										
43	42	42	43	44										
44	44	45	45	46										
45	46	47	47	48	49									
46	48	48	49	50	51									
47	....	49	50	51	52	53								
48	....	51	52	53	54	55	56							
49	....	53	54	55	56	57	58							
50	....	....	56	57	58	59	60	61						
51	....	....	59	60	61	62	63	64						
52	....	....	62	63	64	65	66	67						
53	....	....	....	66	67	68	68	69	70					
54	....	....	....	68	69	70	71	72	73					
55	....	....	....	....	72	73	74	75	76	77				
56	....	....	....	....	76	77	78	79	80	81				
57	....	....	....	....	....	81	82	83	84	85	86			
58	....	....	....	....	....	85	86	87	88	89	90	91		
59	....	....	....	....	....	89	90	91	93	94	95	96	98	
60	....	....	....	....	....	....	94	95	97	99	100	102	104	10
61	....	....	....	....	....	....	99	101	102	104	106	108	109	116
62	....	....	....	....	....	....	104	106	107	109	111	113	114	111
63	....	....	....	....	....	....	109	111	112	113	115	117	118	115
64	....	....	....	....	....	....	....	115	117	118	119	120	121	129
65	....	....	....	....	....	....	....	117	119	120	122	123	124	122
66	....	....	....	....	....	....	....	119	121	122	124	126	127	125
67	....	....	....	....	....	....	....	....	124	126	127	128	129	138
68	....	....	....	....	....	....	....	....	126	128	130	132	133	130
69	....	....	....	....	....	....	....	....	129	131	133	135	136	134
70	....	....	....	....	....	....	....	....	....	134	136	138	139	147
71	....	....	....	....	....	....	....	....	....	138	140	142	143	140
72	....	....	....	....	....	....	....	....	....	....	145	147	148	144
														9

Relation in Length of the Head, Trunk and Extremities.—In the new-born the lower portion of the body, measured from the level of the iliac crest, is, according to the figures of Zeising,[1] about equal to the upper portion in length, while in adults it measures 62 to 63 per cent. of the total. The measurements of Taylor[2] on 250 new-born infants gave very similar results, 214 having the mid-point of the body at or above the navel. It is this which makes the infant's legs appear so short. Later the lower portion grows more rapidly, but it is not until near puberty that its rate of growth becomes markedly greater than that of the upper. Grover[3] who measured 500 children, found that the length of the leg at birth was 43 per cent. of the total length and at 7 years 54 per cent. The head in the new-born, measured from the vertex to the larynx, is very long, about 25 per cent. of the total body-length, against about 11 per cent. in the adult (Hoffmann).[4] The proportionate length of the upper extremities does not alter materially with increase in years. The comparison of the length of the different parts of the body is well shown in the accompanying diagram (Fig. 7). The influence of sex is somewhat apparent. According to the studies of Peckham[5] the length of the trunk of the girl is less than that of the boy until about the age of 9 years, after which, until the age of 15, it is greater. On the other hand while the legs of the girl at 9 years are longer than those of the boy, the boy much surpasses her later in length of leg. In fact, girls increase very little in length of leg after the age of 14 or 15 years, while boys grow both in legs and trunk.

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[1] *Loc. cit.*

[2] Amer. Jour. Dis. Child., 1922, XXIII, 406.

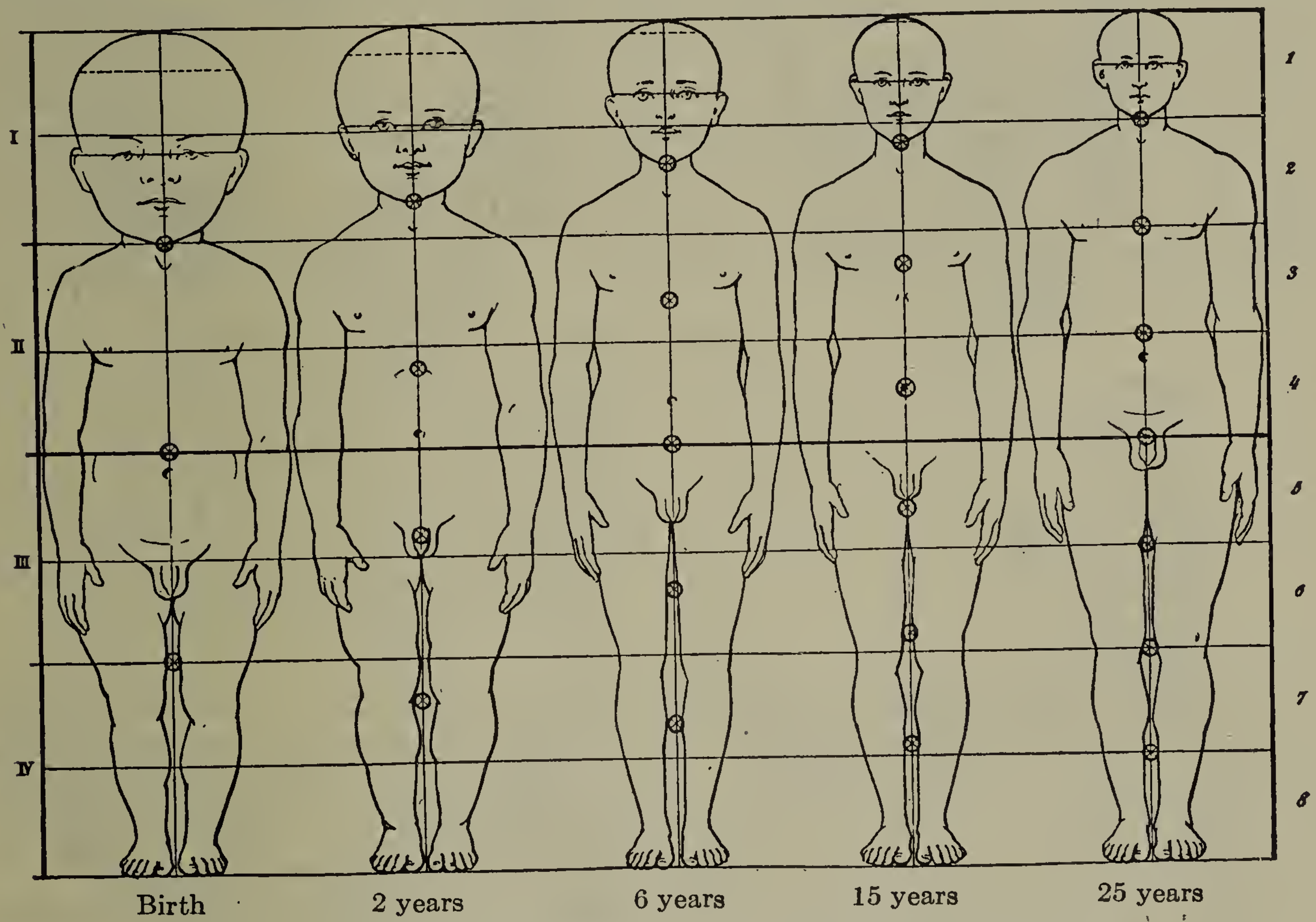

FIG. 7.—GROWTH-PROPORTIONS AT DIFFERENT LIFE-PERIODS.

Showing the length of different portions of the body as compared with the head at different periods of life. At birth the body is 4 heads high, while in the adult it is 8 heads high. (*After Stratz, Der Körper des Kindes*, 1904, 64, *Fig.* 42.)

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[1] Nova Acta Acad. Caes. Leop. Carol. natur. curios., 1858, XXVI, 2, 783. Ref. Vierordt, Daten u. Tabellen, 1906, 30, 31.

[2] Amer. Jour. Dis. Child., 1919, XVII, 353.

[3] Arch. of Pediat., 1915, XXXII, 473.

[4] Ref. Vierordt, Daten u. Tabellen, 1906, 15.

[5] 6th Ann. Rep. Wisconsin State Board of Health, 1881, 60.

General Surface.—By the age of 2 weeks the dark-red color of the skin of the body has changed to the rose-pink characteristic of infancy. Generally by the end of the 1st week or earlier any lanugo remaining begins to fall, and this is accompanied by a more or less extensive shedding of the epidermis, sometimes in small scales, sometimes in large flakes. This shedding lasts from 2 to 3 weeks. The sudoriparous glands are not very active in the first 4 weeks of life in healthy children. Very soon, however, they secrete well and the insensible and even sensible perspiration is considerable, although free perspiration normally does not occur for several months.

Head. Size and Shape.—The *circumference* of the head at birth averages about 13 to 13¾ inches (33 to 35 cm.). The measurement in new-born girls is from 0.2 to 0.4 inch (0.5 to 1 cm.) less than in boys. (Taylor[1] 125 male infants 34.7 cm. (13.5 in.); 125 female infants 34.1 cm. (13.3 in.). Growth is at first rapid, but is very slow after the age of 5 years. Published statistics vary considerably, but a comparison of a number of investigations gives the following average figures of the circumference at birth and of the increase:

TABLE 16.—CIRCUMFERENCE OF THE HEAD

Birth	33 to 35 cm.	(13.0 to 13.8 in.)
6 months	42 to 45 cm.	(16.5 to 17.7 in.)
1 year	45 to 46 cm.	(17.7 to 18.1 in.)
2 years	47 to 48 cm.	(18.5 to 18.9 in.)
3 years	48.5 to 50 cm.	(19.1 to 19.7 in.)
4 years	50 to 52 cm.	(19.7 to 20.5 in.)
5 years	52 to 53 cm.	(20.5 to 20.9 in.)
Adult	53 to 55 cm.	(20.9 to 21.7 in.)

It will be noticed that the growth in circumference is about 10 cm. (3.9 inches) during the 1st year, which is approximately one-half of the increase of the child in length. In the 2d year the increase is about 2 cm. (0.8 inch). Girls gradually fall behind to the amount of 1½ to 3 cm. (0.6 to 1.2 inches) in the circumference of the head.

The *shape* of the head differs decidedly in infants from that in adult life (Fig. 8). The facial portion is small as compared with the cranial; the ratio of the respective dimensions in the new-born being 1:8 and in the adult 1:2 or 2½ (Froriep).[2] Its rate of growth is, however, more rapid (Weissenberg).[3] The caput succedaneum, *i.e.*, the swelling of the scalp resulting from pressure during labor, generally disappears by the 10th day or earlier (see p. 306.)

Owing to the yielding nature of the bones of the skull and the absence of union of the sutures the shape may be altered by long-continued pressure as by that during labor, or by the child lying too much upon the back or side, thus flattening the occiput or one of the parietal regions. This distortion is usually only of temporary duration.

Fontanelles.—The posterior fontanelle can be felt open until the age of 6 or 8 weeks. The anterior, of rhomboidal shape, increases in size according to the views of most observers until the 9th month, owing to the fact that the brain grows faster than the bones covering it. By the 12th month it should be decidedly smaller. According to Kassowitz,[4] however, it should grow steadily smaller from the time of birth, and any increase in its size must be attributed to rickets. The fontanelle should be closed by the age of 17 or 18 months. Even in healthy children it sometimes closes earlier or remains open longer than the average time allows, but closure should be accomplished by 2 years at the latest. It is slightly larger in boys, and in the children of primiparæ (Fehling).[1] There are great variations in the size of the anterior fontanelle at birth, and in the statements made regarding this. A diameter ranging from 2 to 2½ cm. (0.8 to 1 inch) between the parallel sides perhaps expresses the average. Elsässer[2] gives the following figures for the changes which take place with time:

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[1] *Loc. cit.*

[2] Die Characteristik des Kopfes nach dem Entwickelungsgesetze desselben. Berl.. 1845. Ref. Henke, Gerhardt's Handb. d. Kinderkr., 1877, I, 250.

[3] Jahrb. f. Kinderheilk., 1908, LXVIII 316.

[4] Verhandl. d. deutsch. Gesellsch. f. Kinderheilk., Strassburg, 1885.

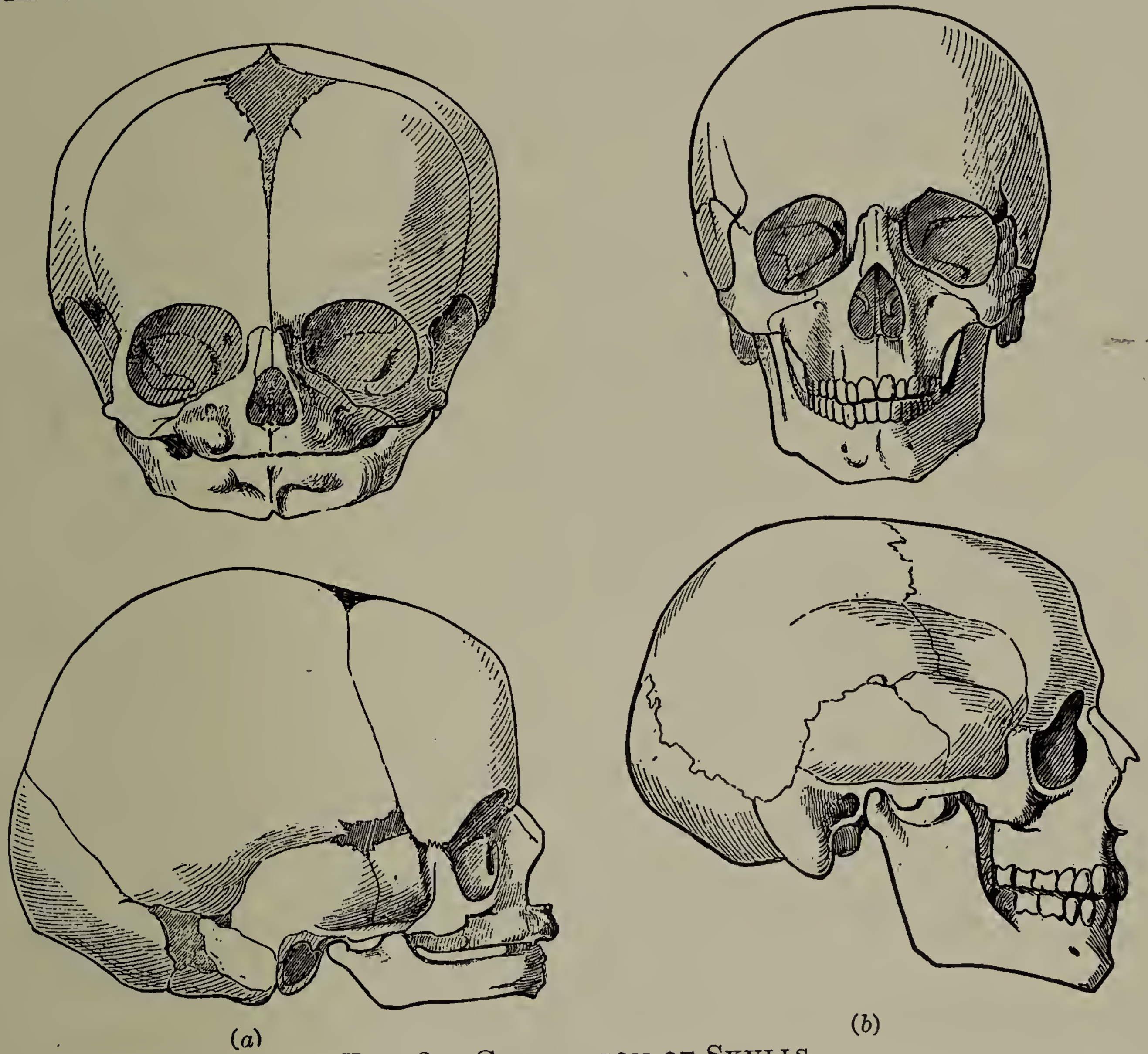

Fig. 8.—Comparison of Skulls.

Of the infant (*a*) and of the adult (*b*). (*After Henke, Gerhardt's Handb. d. Kinderkr.,* 1877, *I*, 248; 249.)

Table 17.—Size of Anterior Fontanelle (Elsässer)[3]

1 to 3 months	2.51 cm. (1.0 in.)
4 to 6 months	3.12 cm. (1.2 in.)
7 to 9 months	3.63 cm. (1.4 in.)
10 to 12 months	3.11 cm. (1.2 in.)
13 to 15 months	2.03 cm. (0.8 in.)

Scalp.—Although the scalp at birth frequently shows only short, sparse hair it is often covered with a thick, rather dark, comparatively long growth, measuring from 1½ to 2 inches (3.8 to 5.1 cm.). This begins to fall at the end of the 1st week and generally leaves the head almost bald, although some stays on much longer than this, and many infants retain a heavy growth of hair for months. The new hair comes in only slowly, and is of firmer texture and generally lighter color. Unless precautions are taken a collection of oily scales very rapidly accumulates on the scalp.

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[1] Arch. f. Gynäk., 1875, VII, 507.

[2] Der weiche Hinderkopf, 1843, 10.

[3] I have used the metric equivalents for Elsässer's statistics as given by Vierordt (Daten und Tabellen, 1906, 104). The original is in "the old Parisian lines" (p. 9 of his treatise).

Eyes.—The eyes at birth and in the first weeks of life are kept shut or half open and are largely devoid of expression. Their color is a somewhat indefinite gray-blue, which only later changes into the permanent hue of the iris, varying in different subjects. By the end of the first 6 months Schindler[1] found 70 per cent. of normal infants blue-eyed, and even by the age of 1 year the pigmentation of the iris in those to be dark-eyed may not be complete. The eyes of atrophic infants become darker much earlier. The movements of the eyes are largely incoördinated. Tears are usually not shed until the age of 3 or 4 months.

Ears.—The meatus is not fully developed at birth, the portion finally osseous not becoming so until the 4th year. The direction of the meatus is inward and downward and the tympanic membrane is horizontal or inclined slightly upward. The tympanic cavity contains no air at birth but only swollen mucous membrane and mucus. The **nose** of the new-born is relatively small and, as a whole, situated higher in the face than in adult life. The maxillary antrum and ethmoidal cells are present at birth; the other sinuses develop later (Coffin).[2] The fat in the **cheeks** is comparatively well-developed, and forms in each what is called the "sucking-cushion." By a curious provision of Nature the fat in the sucking cushion contains less volatile fatty acids than other body-fat, and for this reason is the last to disappear in conditions of emaciation (Lehndorff).[3] The **jaws** are small and in a rudimentary condition at birth. The angle of the ramus and body of the lower jaw is much more obtuse than later in life. During infancy the jaws grow considerably, but especially in later childhood they enlarge in order to permit the development of the permanent molar teeth.

Spine.—The spinal column of the new-born is of such flexibility that the existence of any of the natural curves of later life is doubtful. The sacrococcygeal curve is present, it is true, but the remainder of the spine either forms one long continuation of this or is straight. Symington[4] has pointed out that the *neck*, as compared with the rest of the spinal column, is in reality relatively longer than in adult life, about equalling the lumbar portion. Its apparent shortness is due to the large amount of fat covering it, and to the high position of the sternum. The curve in the neck, with the convexity forward, does not appear until the child begins to hold its head erect, and never becomes fixed. Still later, when the child learns to stand and walk, a similar curve forms in the lumbar region and one with the concavity forward in the dorsal region. The lumbar spine grows faster than the other portions until a little after puberty.

Thorax.—The chest in infancy is small as compared with the abdomen and with the thoracic development of later life. The combination of small chest and large abdomen, together with the high position of the narrow and insignificant shoulder-girdle, gives the trunk a peculiar barrel-shaped appearance. The *nipples* are small and are situated in the 4th interspace or over the 4th rib as in adult life.

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[1] Zeitschr. f. Kinderheilk., Orig., 1919, XIX, 153.
[2] Amer. Jour. Med. Sci., 1905, CXXIX, 302.
[3] Jahrb. f. Kinderheilk., 1907, LXVI, 286.
[4] Anatomy of the Child, 1887.

The *ribs* are more horizontally placed than in adult life and the false ribs particularly project upward to a greater extent. The *diaphragm* extends somewhat higher, and the *sternum* is relatively smaller than in adult life. The upper border of the manubrium of the sternum stands higher than later while the lower projects more sharply forward.

Whereas in adults the transverse diameter of the thorax is to the anteroposterior as 2 : 1 (Fetzer),[1] or 3 : 1 (Symington),[2] in the new-born the diameters according to Eckerlein[3] are nearly equal, the transverse being to the antero-posterior as 3 : 2. As a result the horizontal section of the thorax in early infancy appears nearly circular, while that of the adult is elliptical. The transverse diameter grows more rapidly than the other, and the adult shape of the chest is present to a large degree by the beginning of childhood.

There is considerable diversity in the estimates of different investigators regarding the circumference of the chest at birth taken at the height of the nipples, and the rate of growth in the first 5 years of life. This depends upon the great variation to which this growth is susceptible, rendering any statistics only average ones. Approximate figures, the average obtained from the statistics of a number of authors, read as follows:

Table 18.—Growth of Chest in the First Five Years

Birth	32 to 33 cm. (12.6 to 13.0 in.)
6 months	41 to 42 cm. (16.1 to 16.5 in.)
1 year	44 to 46 cm. (17.3 to 18.1 in.)
2 years	45 to 48 cm. (17.7 to 18.9 in.)
3 years	50 to 51 cm. (19.7 to 20.1 in.)
4 years	52 to 53 cm. (20.5 to 20.9 in.)
5 years	54 to 56 cm. (21.3 to 22.0 in.)

In female children the circumference is from 0.5 to 1.5 cm. (0.2 to 0.6 inch) less than in male.

The difference in the rate of growth of the thorax in each sex after the age of 5 years up to that of 18 years is illustrated by the observations of Porter[4] in over 34,000 measurements of school-children in St. Louis, Mo., and is shown in the following table:

Table 19.—Growth of Chest in Boys and Girls

Years	Chest	
	Boys	Girls
6	59.05 cm. (23.2 in.)	58.34 cm. (23.0 in.)
7	60.62 cm. (23.9 in.)	59.47 cm. (23.4 in.)
8	62.18 cm. (24.5 in.)	60.81 cm. (23.9 in.)
9	63.90 cm (25.2 in.)	62.51 cm. (24.5 in.)
10	65.59 cm. (25.8 in.)	63.02 cm. (24.8 in.)
11	67.24 cm. (26.5 in.)	65.85 cm. (25.9 in.)
12	68.76 cm. (27.1 in.)	68.34 cm. (26.9 in.)
13	70.61 cm. (27.8 in.)	71.29 cm. (28.1 in.)
14	73.27 cm. (28.9 in.)	74.13 cm. (29.2 in.)
15	76.56 cm. (30.1 in.)	76.78 cm. (30.6 in.)
16	79.22 cm. (31.2 in.)	78.85 cm. (31.0 in.)
17	81.39 cm. (32.0 in.)	80.39 cm. (31.7 in.)
18	84.52 cm. (33.3 in.)	80.45 cm. (31.7 in.)

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[1] Ueber den Einfluss d. Militärdienstes auf d. Körperentwickelung, 1879, 198; Ref. Vierordt's Daten u. Tabellen, 1906, 98.

[2] *Loc. cit.*

[3] Zeitschr. f. Geburtsh. u. Gyn., 1890, XIX, 120.

[4] Transac. Acad. Science St. Louis, VI, No. 12, 354.

According to the observations of Monti[1] the average increase in circumference in the 1st year is 12 cm. (4.7 inches), in the 2d year 3 cm. (1.2 inches), and in the remaining years up to 12, 1.25 cm. (0.5 inch). There then occurs a sudden increase in the rate of growth, averaging 4 cm. (1.57 inches) a year.

The comparison of the rate of growth in the circumference of the chest and of the head respectively is of great practical utility. The combination of the two tables already presented (pp. 22 and 25) gives the following results:

Table 20.—Comparison of Circumferences of the Head and Chest

	Head	Chest
Birth	33–35 cm. (13.0 to 13.8 in.)	32–33 cm. (12.6 to 13.0 in.)
6 months	42–45 cm. (16.5 to 17.7 in.)	41–42 cm. (16.1 to 16.5 in.)
1 year	45–46 cm. (17.7 to 18.1 in.)	44–46 cm. (17.3 to 18.1 in.)
2 years	47–48 cm. (18.5 to 18.9 in.)	45–48 cm. (17.7 to 18.9 in.)
3 years	48.5–50 cm. (19.1 to 19.7 in.)	50–51 cm. (19.7 to 20.1 in.)
4 years	50–52 cm. (19.7 to 20.5 in.)	52–53 cm. (20.5 to 20.9 in.)
5 years	52–53 cm. (20.5 to 20.8 in.)	54–56 cm. (21.3 to 22.0 in.)

The studies of Grover[2] on 500 children of different ages are largely confirmatory of the table as given, except that a decided relationship of both circumferences to the weight was noted; that of the head being greater up to a body-weight of 21 pounds (9525.43), while after 25 pounds (11339.80) that of the chest exceeded.

This relationship is, however, open to considerable variation. The chest often grows quite rapidly, its circumference equalling that of the head by the age of 1 year. Still Frøbelius[3] has found from a large number of measurements, that the circumference of the chest at birth should never be over 2 to 2.5 cm. (0.8 to 1 inch) less than that of the head, and that in proportion as these figures are exceeded the mortality is much increased. It is certainly a sign of feeble development when the circumference of the chest has not exceeded that of the head by the age of 3 years. There is no fixed relationship of the circumference of the chest and the length, but the increase seems rather to keep pace with that of the weight (Schlesinger).[4]

Abdomen.—The abdomen appears relatively large and prominent in infancy, depending in part upon the large size of the liver, in part on the great amount of subcutaneous fat present, and in part on the small size of the pelvis and of the chest. Its circumference measures about the same as that of the chest up to 2 years after which period it is decidedly less. The stump of the *umbilical cord* remaining after birth gradually shrivels, exhibiting a small red area around it at its junction with the body. About the 4th to the 6th day the stump falls, leaving an ulcer which heals rapidly, closing after 5 or 6 days. The umbilicus occupies nearly the central part of the body during the first 2 years. In adult life its distance above the soles is ⅗ of the total length of the body.

[1] Kinderheilk., in Einzeldarst., 1899, I, 565.
[2] Arch. of Pediat., 1915, XXXII, 473.
[3] St. Petersb. med. Zeitschr., 1873–74, IV, 363.
[4] Zeitschr. f. Kinderheilk., Orig., 1917, XVI, 265.

Pelvis.—The pelvis in the infant is very small and more obliquely situated than later. According to Liharzik,[1] the width of the body at the hips at all ages equals that of the shoulders in males, but the hips are always wider than the shoulders in females.

Limbs.—The bowing of the short *legs* present at birth (p. 3) persists until in the course of the 2d year. The tendency to hold the arms and legs in the fetal position with the feet dorsally flexed is exhibited more or less during the early months. Although infants are seemingly flat-footed, in reality the feet are shaped much as in adult life, the shortness and thickness seen especially in later infancy depending in reality upon the large amount of fat in the subcutaneous tissue. Taylor[2] found in 250 new-born infants that the span from finger-tip to finger-tip with the arms fully spread was 51 cm. (20 in.).

DIGESTIVE APPARATUS

Mouth.—The mouth in early infancy is comparatively dry, with the tongue also drier than later and generally with a whitish coating. The jaws at birth will not meet. They are covered with gums of a pale-red color, and exhibit rather hard narrow opposing ridges. No signs of the presence of teeth are visible.

Dentition.—The 20 *temporary teeth*, also called *deciduous*, *first*, or *milk* teeth, that appear later are at birth enclosed in dental follicles, and in alveoli in the jaw which are already osseous in nature. The crowns of the incisor teeth are even then entirely calcified, as are those of the other teeth to a considerable extent. The teeth and tooth-sacs are covered only by mucous membrane and sub-mucous connective tissue, and the alveoli are broad and allow of free growth. As calcification and elongation of the roots take place, the crowns are gradually forced onward in the direction of least resistance. Under this constant pressure outward the gums covering them atrophy, flatten and grow paler, and the teeth finally push through. Probably the only resistance offered has come from the sub-mucous tissue. Complete calcification of the fangs is not present when the teeth appear, and does not occur until the child is several years of age.

The germs of the *permanent* teeth, or *second* teeth, except the second and third molars, are already present in the gums at birth, resting against the posterior walls of the dental sacs of the temporary teeth. The crowns of the anterior molars are calcified. In the other teeth of this set the calcification begins at different periods between the 1st and the 8th year. Like the first set they increase in length, their eruption depending principally upon the calcification of the roots. Probably from insufficient supply of blood, occasioned by pressure of the advancing permanent teeth, the roots of the temporary set finally undergo absorption and the teeth drop out in much the same order as they came in.

Eruption of the Temporary Teeth (Fig. 9).—Many different statements have been made by authors regarding the time of the eruption of the 20 temporary teeth. The following table expresses a very generally accepted view. It will be noticed that the teeth erupt in distinct groups, with a period between each group:

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[1] Das Gesetz des Wachsthums, etc., 1862; Ref. Vierordt's Datenu. Tabellen, 1906, 30.

[2] Amer. Jour. Dis. Child., 1919, XVII, 353.

Table 21.—Eruption of the Temporary Teeth

First Group	2 lower central incisors	7 months
Pause	3 to 8 weeks.	Total 2.
Second Group	4 upper incisors	8 to 10 months
Pause	1 to 3 months.	Total 6.
Third Group	4 anterior molars and 2 lower lateral incisors	12 to 15 months
Pause	2 to 3 months.	Total 12.
Fourth Group	4 canines	18 to 24 months
Pause	2 to 4 months.	Total 16.
Fifth Group	4 posterior molars	20 to 30 months
		Total 20.

The upper and lower canines are popularly called the "eye-teeth" and the "stomach-teeth" respectively. In the third group the lower lateral incisors appear, according to many observers, before the molars, but there is no absolute rule for this. In fact the whole table represents only the order of eruption very frequently seen, and it is certain that there may be an extremely wide variation within physiological limits. The age of 7 months may be considered the average time of appearance of the first tooth, yet any time not exceeding the 1st year may be called normal. From 6 to 8 months is a very common range, and eruption at 3 or 4 months is not at all uncommon.

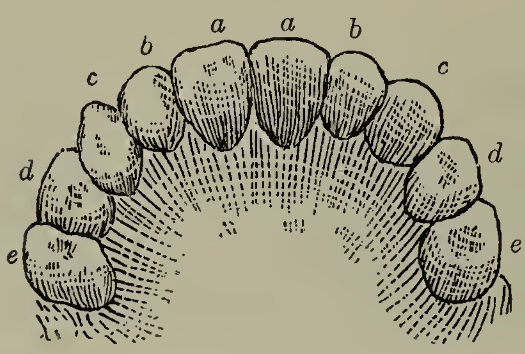

Fig. 9.—Diagram Showing the Temporary Teeth.

a, central incisors; *b*, lateral incisors; *c*, canines; *d*, anterior molars; *e*, posterior molars.

In perfectly normal states the eruption of the teeth is unattended by any symptoms whatever. The pathological conditions which are believed to develop will be considered in the section upon Disorders of Dentition (Vol II, p. 4).

Eruption of the Permanent Teeth (Fig. 10).—The permanent teeth, or teeth of the second dentition, are 32 in number. The earliest to be cut are the first molars, which come in just posterior to the temporary second molars. They appear at about the age of 6 years and are consequently often called the "6-year-old molars." The other teeth erupt in much the same order as those of the temporary set. Although the order and dates of appearing are subject to considerable variation, the following table is a fair expression of them:

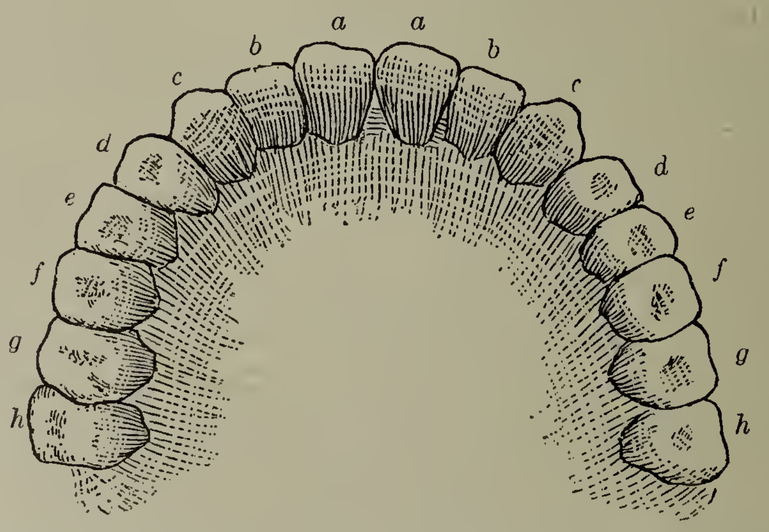

Fig. 10.—Diagram Showing the Permanent Teeth.

a, central incisors; *b*, lateral incisors; *c*, canines; *d*, first bicuspids; *e*, second bicuspids *f*, first molars; *g*, second molars; *h*, third molars.

Table 22.—Eruption of the Permanent Teeth

First molars	6 years
Central incisors	7 years
Lateral incisors	8 years
First bicuspids	9 years
Second bicuspids	10 years
Canines	11 to 13 years
Second molars	12 to 15 years
Third molars (Wisdom teeth)	17 to 25 years

The teeth of the lower jaw usually erupt somewhat before the corresponding ones of the upper jaw, the interval being often as much as several months. It will be observed that the permanent molars do not replace any of the temporary teeth, but constitute 12 new teeth additional to the original 20. The bicuspids replace the temporary molars and the canines and incisors the corresponding teeth of the first set. The bicuspids are smaller than the temporary molars, and the permanent incisors larger than those which preceded them. At about the age of 6 years the jaws contain all the teeth of both sets, visible or concealed, except the third permanent molars (Fig. 11).

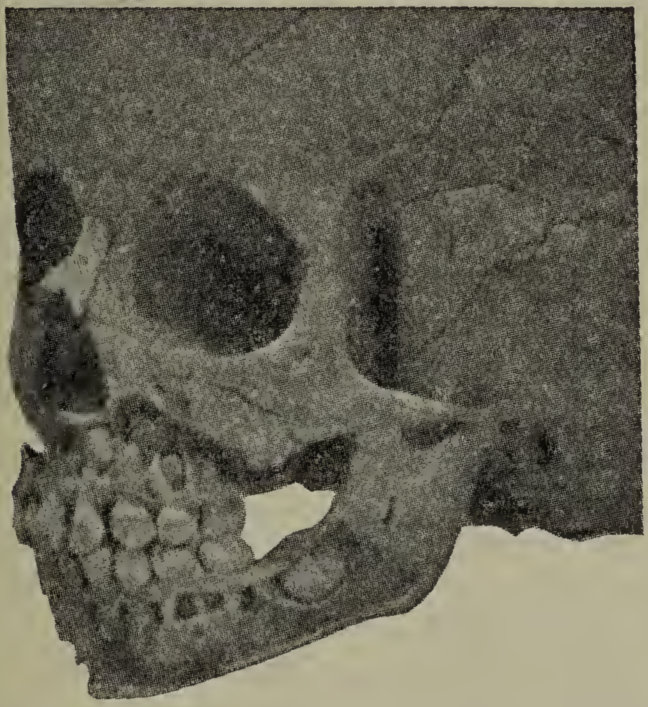

Fig. 11.—Jaws of Child About 7 Years Old, Showing Temporary and Permanent Sets of Teeth, Except the Wisdom Teeth.

(*McClellan, in Keating's Cyclopedia of the Diseases of Children*, 1889, *I*, 18, *Fig.* 12).

Salivary Glands.—The comparatively small amount of saliva secreted by the new-born accounts for the dryness of the mouth and the coating of the tongue referred to. By the 2d month increase in the secretion begins and by the age of 3 or 4 months this is still further augmented and the child begins to "dribble." This increase takes place, as a rule, before the eruption of teeth, and appears to be in no way connected with it. Although according to the investigations of Korowin[1] and of Zweifel[2] ptyalin is present in the salivary glands at birth, and even as early as 1½ months before term (Nicory),[3] any diastatic activity is inconsiderable until the increased secretion of saliva begins. Even then, since much of the fluid runs out of the mouth, its influence upon digestion is probably not great. As the 1st year advances the amount of saliva grows greater and by about the age of 1 year its diastatic power is about as great as in adult life (Finizio).[1] The reaction remains fairly constantly neutral or alkaline. Urea is excreted in the saliva in about the same concentration as it is present in the blood (Hench and Aldrich).[2] It is increased in the saliva when there is urea-retention in the blood, and the determination of the content of it in the former may be used as an index of nonprotein nitrogen-retention, such as may occur in nephritis. The average normal amount in children is about 3.5 mg. per 100 c.c. (.016 grains per fl. oz.) (Calvin and Isaacs).[3]

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[1] Centralbl. f. d. med. Wissensch., 1873, XI, 261.

[2] Untersuch. ü. d. Verdaungsapparat der. Neugeboren., 1874.

[3] Biochem. Jour., 1922, XVI, 387.

Esophagus.—The total length of the esophagus in the new-born, according to the statistics collected by Vierordt,[4] is about 10 cm. (3.9 inches) or, including the total distance from the teeth to the cardiac orifice, 17 cm. (6.7 inches). This latter distance at the age of 3 years is 30 cm. (11.8 inches) and in adult life 40 cm. (15.8 inches). The pseudovalvular opening at the gastric end of the esophagus is stated by Gubaroff[5] to be imperfectly developed in infancy. This accounts in part for the greater ease with which vomiting occurs at this time of life.

Stomach.—The capacity of the stomach varies greatly in different infants within normal limits, as is shown by the results obtained by different observers. This is illustrated in the following table from which, however, an idea of the approximate size may be obtained:

Table 23.—Capacity of the Stomach at Different Periods*

	Fleischmann[6]		Holt[7]		Frolowsky[8]		Pfaundler[9]		Beneke[10]	
	C.c.	Fl.oz.	C.c	Fl.oz.	C.c.	Fl.oz.	C.c.	Fl.oz.	C.c.	Fl.oz.
Birth	...	...	36	1.2	...	...	...	...	35–43	1.2–1.5
1 week	46	1.6	...	....	50	1.7				
2 weeks	72	2.4	44	1.5	70	2.4	...	...	153–160	5.2–5.4
1 month	80	2.7	59	2.0	112	3.8	90	3.0		
2 months	140	4.7	100	3.37	158	5.3	100	3.4		
3 months	...	...	133	4.50	167	5.6	110	3.7		
4 months	...	...	148	5.00	178	6.4	125	4.2		
6 months	...	...	176	5.75	...	...	160	5.4		
9 months	...	...	...	....	253	8.6	225	7.6		
1 year	...	...	263	8.9	...	...	290	9.8		
2 years	...	...	...	....	...	...	...	...	740	25

Scammon and Doyle[11] in a careful clinical study on 323 infants found the physiological gastric capacity on the 1st day of life equalling 7 grams (.24 fl. oz.), with a rapid increase to 81 grams (2.75 fl. oz.) by the 10th day. The maximum capacity was distinctly greater than these figures. Roughly we may estimate the capacity of the stomach at birth at 1¼ fl. oz. (37), at 1 month as 2½ fl. oz. (74), at 6 months as 5 fl. oz. (148), and at 1 year as 9¾ fl. oz. (288). It depends, however, on the size of the child as much as or more than on the age alone. This must always be taken into consideration in determining the amount of food required by an infant of a certain age. On the other hand, it is also true that an infant not infrequently can digest, and requires, a larger amount of food than its age or size would indicate. This may well be because some of the liquid taken passes into the intestine before the meal is completed. Basing the dietetic requirements upon the estimated size of the stomach is consequently a procedure to be depended upon only with limitations. The capacity in artificially fed infants is somewhat greater than in those fed at the breast. The orifice of the pylorus measures 2 cm. (0.78 inch) in circumference in the new-born, according to Pfaundler.[1] The position of the stomach early in infancy had been generally considered to be nearly vertical or slightly oblique, and the form cylindrical; but the more recent investigations are not entirely in accord with this. Flesch and Péteri[2] found it nearly horizontal in the 1st year of life, but becoming more vertical after this period. Smith[3] believes it to be not vertically situated, but rather obliquely, and Pisek and LeWald[4] showed that the organ changes its size and shape from time to time, depending upon the amount of food contained in it. It is oftener horizontally situated rather than vertically, and the pylorus generally occupies a comparatively high and anterior position. The amount of gastric secretion is large in infancy. Pepsin, rennin and hydrochloric acid are present at all periods, although the amount of rennin and of acid occurring in the new-born is proportionately much less than in adult life (Leo).[5] Griswold and Shohl[6] on the other hand, found a higher acidity in the newborn, and Hess[7] states that hydrochloric acid is present even in the first few hours of life in infants who have never been fed. However this may be, the amount rapidly increases as the infant grows older. According to Langendorf[8] pepsin is found in the fetus even as early as the 4th month of intra-uterine life. It is possible that rennin is not a substance distinct from pepsin. A fat-splitting ferment is also present, as demonstrated by Sedgwick[9] and others; and Ibrahim and Kopeć[10] found it in the 6-months' fetus.

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* The original tables vary slightly from this, the data being for the number of weeks rather than of months. As the differences, however, are inconsiderable, I have purposely distorted the figures a little to apply to the age in months.

[1] Rev. d'hyg. et de méd. inf., 1909, VIII, 224.

[2] Jour. Amer. Med. Assoc., 1922, LXXIX, 1409.

[3] Amer. Jour. Dis. Child., 1925, XXIX, 70.

[4] Daten u. Tabellen, 1906, 112.

[5] Arch. f. Anat. u. Entwickelungsgeschichte, 1886, 395.

[6] Klinik d. Pädiatrik, 1875, I, 17.

[7] Arch. of Pediat., 1890, VII, 963.

[8] St. Petersb. Dissertation, 1876. Ref. Vierordt, Daten u. Tabellen, 1906, 115.

[9] Wien. klin. Wochenschr., 1897, X, 964.

[10] Deutsch. med. Wochenschr., 1880, VI, 448.

[11] Amer. Jour. Dis. Child., 1920, XX, 516.

Liver.—The liver at birth is hyperemic and large and its edge can be distinctly felt below the costal margin. To a great extent it covers the anterior and outer surface of the stomach. It continues to extend below the costal margin until about the age of 5 years, and, according to Zamkin[11] may reach as much as 3.5 cm. (1.36 in.) below in healthy children up to the age of 12 years. The area of hepatic dullness is also greater in early life, the upper margin of this reaching the 4th rib. According to the statistics collected by Vierordt[12] its absolute weight averages

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[1] Ueber Magencapacität, etc., Bibliotheca medica, 1898, 35.
[2] Zeitschr. f. Kinderheilk., Orig., 1911, II, 263.
[3] Arch. of Pediat., 1914, XXXI, 784.
[4] Amer. Jour. Dis. Child., 1913, VI, 232.
[5] Berl. klin. Wochenschr., 1888, XXV, 981.
[6] Amer. Jour. Dis. Child., 1925, XXX, 541.
[7] Amer. Jour. Dis. Child., 1913, VI, 264.
[8] Arch. f. Physiolog., 1879, 95.
[9] Arch. of Pediat., 1906, XXIII, 414.
[10] Zeitschr. f. Biol., 1910, LIII, 201.
[11] Arch. of Pediat., 1926, XLIII, 169.
[12] Arch. f. Anat. u. Entwickelungsgeschichte, 1890, Suppl. Band.

141.7 grams (5 oz.) in the male and 164 grams (5.8 oz.) in the female; *i.e.*, 4.5 per cent. and 5.5 per cent. of the body-weight, while in the adult it is but from 2.8 per cent. to 3 per cent. The weight increases little or none during the first 6 months, and, in fact, diminishes slightly in the first weeks. Kowalski's[1] figures, however, do not corroborate this failure to increase in weight in the early months. Bile is secreted even before birth. The investigations of Jacubowitsch[2] indicate that the bile of the infant differs from that of the adult especially in the larger proportion of water and the smaller amount of biliary acids and of bile-salts present, and the consequent lesser power of digesting the fats.

Pancreas.—The pancreas at birth weighs 3.5 grams (0.12 oz.) (Vierordt),[3] bearing about the same relation to the body-weight as in adult life. Its secretion has the power of digesting fat in early infancy, but not to the degree possessed later. The diastatic power (amylopsin) was claimed by Korowin[4] to be absent during the first 3 weeks, feeble after this, and not to attain its full power until about the end of the 1st year; but Hess[5] by the use of the duodenal tube found it present in the new-born, although not in large amount, thus confirming the observation of Ibrahim,[6] who obtained it in a 6-months' fetus. It would appear to increase more rapidly in power than formerly supposed. The peptonizing function (trypsin) is active in the 1st month (Zweifel).[7] Langendorf[8] showed its presence in the fetus of 5 months.

Intestine.—The total length of the small intestine in the new-born would appear from Vierordt's[9] statistics to vary considerably, ranging from 2 to 3.5 metres (6.4 to 11.5 feet). The large intestine according to the same authority measures from 0.42 to 0.48 metres (1.38 to 1.57 feet). The relative length of the entire bowel as compared with that of the body is greater in children than in adults. The small intestine grows rapidly. Beneke[10] found it at birth 5¾ times the body-length, at 2 years about 6½ times, at 7 years 5 times, and in adult life but about 4½ times. The large intestine was about equal in length to that of the body, both in the new-born and in the adult. Robbin[11] observed that the relative length of the large intestine equaled from 80 to 130 per cent. of the body-length in 91.3 per cent. of 185 infants from birth to 3 years of age, and that of the small intestine equaled from 500 to 900 per cent. of the body-length in 79.9 per cent. of the cases. The sigmoid flexure is especially long in infants, being at birth, as stated by Treves,[12] almost equal to ½ the total length of the large intestine. It often forms a huge loop running up to the lower border of the liver. It is perhaps partly due to this great length that constipation occurs so frequently in infancy. During the first 4 months the sigmoid diminishes greatly in length, while the rest of the colon proportionately increases, the total length of the large intestine thus remaining unchanged. The vermiform appendix reaches its full length early in life. It is particularly prone to kinking in infancy and childhood, and is abundantly supplied with adenoid tissue, both factors tending to the production of appendicitis. According to the conclusions of Gundobin[1] Peyer's patches are well developed, but less numerous in the new-born as compared with adults; the number of solitary follicles greater; Lieberkühn's follicles and the villi more abundant, and Brunner's glands numerous but not completely developed. The muscular strength of the intestine is comparatively feeble. Amylolytic ferments are present in the small intestine from birth.

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[1] Dissert. St. Petersb., 1900, 21. Ref. Morse and Talbot, Diseases of Nutrition and Infant Feeding, 1920, 16.

[2] Jahrb. f. Kinderheilk., 1886, XXIV, 377.

[3] Daten u. Tabellen, 1906, 44.

[4] Centralb. f. d. med. Wissensch., 1873, 261.

[5] Amer. Jour. Dis. Child., 1912, IV, 205.

[6] Verhandl. d. Gesellsch. f. Kinderheilk., 1908, XXV, 31.

[7] Untersuch. ü. d. Verdaungsapparat. d. Neugeboren., 1874.

[8] Arch. f. Physiol., 1879, 95.

[9] Daten u. Tabellen, 1906, 117.

[10] Deutsch. med. Wochenschr., 1880, VI, 433.

[11] Amer. Jour. Dis. Child., 1920, XIX, 370.

[12] Hunterian Lectures, 1885, 10.

DIGESTION IN INFANCY

(See also Absorption and Metabolism, p. 40)

The act of sucking has a pump-like quality. The base of the nipple is seized firmly by the infant's lips; the tongue is pressed against the nipple, making a longitudinal gutter in which this may lie, and along which the milk is conducted backward; the soft palate and base of the tongue approach each other and shut off the posterior opening of the oral cavity from the nose; the cheeks sink in slightly, while the lower jaw is depressed, thus producing a partial vacuum, by means of which the milk is drawn from the breast or bottle. As noted by Kasahara,[2] this type of suction is quite different from that seen in older children, where the action is by inspiration. Auerbach[3] found that this suction by inspiration did not begin until a child was 2 or 3 years of age. The sucking cushions already referred to (see Cheeks, p. 24) are supposed to prevent a too great collapse of the cheeks. To nurse satisfactorily the infant must be able to breathe easily through the nose, respiration through it not being interfered with by sucking, although it ceases momentarily during the act of swallowing.

The saliva plays but small part in digestion in early infancy (see Salivary Glands, p. 29). Later its diastatic action is of service in the case of infants receiving starch. Yet this action is, however, not completed in the mouth, and, after the food is swallowed, should continue only so long as the gastric contents are alkaline or but faintly acid, which is but for a short time. Jackson[4] had children with total atresia of the oesophagus spit saliva into a funnel connected with a gastrostomy tube, and noted that the nutrition, previously poor, immediately improved. He believes that saliva plays a much more important part in the nutritive processes of children than is generally realized.

The stomach is more of a simple receptacle in infancy than is the case later in life. Some of the milk entering the stomach passes immediately through the pylorus (Hess).[5] That remaining is promptly coagulated by the action of the rennin or by the hydrochloric acid present. Tobler[6] found that the coagulation commenced in 2 or 3 minutes after the milk was ingested. Human milk forms small, loose curds, while cow's milk produces a much firmer, larger mass. This may be due to the greater percentage of casein present in cow's milk. The acidity of the gastric contents appears very soon after the ingestion of food, and steadily increases. In infants the normal range of the pH of the gastric contents with the milk test-meal at the end of an hour is pH 3.2–pH 5, with 76 per cent. of the readings between pH 3.9 and pH 4.6, and the acidity increases with the length of time after ingestion (Babbott, Johnston, Haskins and Shohl).[1] In new-born infants the acidity is higher, averaging pH.2.5–pH. 2.6. (Griswold and Shohl)[2] Van Puteren[3] found an acid reaction after 10 minutes, and Leo[4] after 15 minutes. Yet as the hydrochloric acid as fast as secreted unites with the elements of the milk, it is only toward the end of gastric digestion that free hydrochloric acid can be obtained in small amount, and generally not at all. Jacobsen[5] states that free acid was not found in over 50 per cent. of the cases examined by him. The degree of acidity in infants is decidedly less than in adult life according to most observers. Muhl,[6] for example, found that in normal children between 1 and 3 years of age the quantity of hydrochloric acid was markedly less than in adults, and in children between 3 and 13 years it was still slightly so. The work of McClendon,[7] however, does not support this view. Bauer and Deutsch[8] believed lactic acid to be the predominant one after the ingestion of food during the 1st half-year; and Heiman[9] found it in half of the breast-fed infants examined. Other investigators, as Sçdziuk,[10] state that lactic acid is seldom found in the stomach of the healthy breast-fed infant. Butyric acid develops in the stomach only in pathological conditions when the amount of hydrochloric acid is diminished.

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[1] Jahrb. f. Kinderheilk., 1892, XXXIII, 439.
[2] Amer. Jour. Dis. Child., 1916, XII, 73.
[3] Du Bois-Raymond's Arch. f. Physiol., 1888, 59.
[4] Arch. of Pediat., 1923, XL, 324.
[5] Amer. Jour. Dis. Child., 1914, VII, 428.
[6] Verhandl. der Gesellsch. f. Kinderheilk., 1906, XXIII, 144.

After the milk has been coagulated its solution by the pepsin begins, the hydrochloric acid now aiding in accomplishing this. Yet much the greater portion of the coagulated milk passes out of the stomach of the infant but little affected in other respects by gastric digestion. There is no action exerted by the gastric secretion upon the carbohydrate, although the fat is acted upon to some extent by the lipase of the stomach.

The length of time during which the ingested food remains in the infant's stomach probably varies with the infant. Leo[11] found that a considerable portion of the milk had left the stomach within ½ hour, the liquid portion containing the sugar and salts and the uncoagulated milk passing out first, and that in the 1st week of life the organ would sometimes be empty within 1 hour, and in the 1st month generally within 1½ hours. This is confirmed by Clarke.[12] Later a somewhat longer time is required, but the average healthy breast-fed infant retains but little of the food in the stomach longer than at most 2 or 2½ hours. This has been practically confirmed radiologically by Leven and Barret,[13] Tobler and Bogen,[14] Flesch and Péteri,[15] Ladd,[16] Pisek and LeWald[17] and others. The onset of hunger-contractions was shown by Ginsburg, Tumpowsky and Carlson[1] to begin in 2 to 3 hours after the ingestion of food. It has been claimed, according to the experiments of Cannon[2] on animals, that the cardiac orifice closes automatically with a certain degree of normal acidity of the gastric contents. The pyloric orifice, on the other hand, as shown by Cowie and Lyon[3] for infants, opens when a certain degree of acidity is attained. According to Babbott, Johnston, Haskins and Shohl[4] the greater the gastric acidity in terms of hydrogen ion concentration, the more rapid is the emptying of the stomach. Investigations by McClure, Reynolds and Schwartz[5] would indicate that these various statements are incorrect, their results showing that the pylorus opens with each antral peristaltic wave independently of the degree of acidity; and Theile[6] even found that alkalinization of the gastric contents shortened the time of their remaining in the stomach. A lack of influence of alkalies in delaying the emptying of the stomach was observed also by Lockwood and Chamberlin.[7] The nature of the food taken influences the time it remains in the stomach. Human milk passes from it more rapidly than cow's milk. The greater the amount of casein, the longer the continuance of the food in the stomach. This fact, previously accepted, was demonstrated radiologically by Ladd,[8] and more recently confirmed in other ways by Bessau, Rosenbaum and Leichtentritt.[9] Delay also depends to a large extent upon the amount of fat present (Tobler).[10] DeBuys and Henriques[11] demonstrated that the body-posture has much to do with the time for the emptying of the stomach, the rapidity being greatest in the right lateral position. The stomach is of little service in the absorption of food, but a small amount of the sugar and digested protein being taken up here; the fats, water and salts practically not at all.

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[1] Amer. Jour. Dis. Child., 1923, XXVI, 475.

[2] Amer. Jour. Dis. Child., 1925, XXX, 541.

[3] Ueber die Verdauung der Säugekinder in den ersten zwei Lebensmonaten. Arbeiten der Gesellsch. der Kinderärzte in St. Petersburg, 1889. Ref. Bauer and Deutsch, Jahrb. f. Kinderheilk., 1898, XLVIII, 27; 68.

[4] Berl. klin. Wochenschr., 1888, XXV, 981.

[5] Ugeskrift f. Laeger, 1915, LXXXI, 863.

[6] Acta Pædiat., 1925, IV, 356.

[7] Jour. Amer. Med. Assoc., 1915, LXV, 12.

[8] Jahrb. f. Kinderheilk., 1898, XLVIII, 22.

[9] Arch. of Pediat., 1910, XXVII, 570.

[10] Przegl. Pedyat., 1913, V, 14. Ref. Arch. f. Kinderheilk., 1914, LXIII, 271.

[11] *Loc. cit.*

[12] Arch. of Pediat., 1911, XXVIII, 648.

[13] Presse médicale, 1906, XIV, 503.

[14] Monatsschr. f. Kinderheilk., 1908, VII, 12.

[15] Zeitschr. f. Kinderheilk., Orig., 1911, II, 263.

[16] Amer. Jour. Dis. Child., 1913, V, 345.

[17] Amer. Jour. Dis. Child., 1913, VI. 232.

In the intestine the trypsin of the pancreatic juice accomplishes the peptonizing of the milk not already digested in the stomach; this being probably, as stated, much the larger part. If the contents of the stomach are expelled from it in a hyperacid state no peptonizing by the trypsin takes place, since this ferment requires the presence of an alkaline reaction, and is, in fact, destroyed by acidity of the gastric secretion. Inasmuch as the saliva is in such small amount in early infancy and the diastatic ferment of the pancreas is not secreted to any considerable extent in the 1st month, the power of digesting any starchy food at this period of life would be thought to be very limited. It must be stated, however, that investigations by Heubner,[12] Carstens,[13] Shaw[14] and others apparently prove that even infants of 2 months possess a positive power to digest starch; and clinical experience, as in the feeding of cases of pyloric stenosis, indicates a power of digestion at a decidedly earlier age. The bile-salts in infancy in combination with the steapsin of the pancreas split

[1] Jour. Amer. Med. Assoc., 1915, LXIV, 1822.
[2] Amer. Jour. Physiol., 1908, XXIII, 105.
[3] Amer. Jour. Dis. Child., 1911, II, 252.
[4] Amer. Jour. Dis. Child., 1923, XXVI, 475.
[5] Arch. Int. Med., 1920, XXVI, 410.
[6] Zeitschr. f. Kinderheilk., Orig., 1917, XV, 318.
[7] Arch. Int. Med., 1923, XXXII, 71.
[8] Arch. of Pediat., 1913, XXX, 740.
[9] Jahrb. f. Kinderheilk., 1921, XCV, 123.
[10] Ergebnisse der inn. Med. u. Kinderheilk., 1908, I, 514.
[11] Amer. Jour. Dis. Child., 1918, XV, 190.
[12] Berlin. klin. Wochenschr., 1895, XXXII, 201.
[13] Berlin. klin. Wochenschr., 1895, XXXII, 1100.
[14] Albany Med. Annals, 1904, XXV, 148.

the fat and permit of saponification and the forming of an emulsion. The secretion of the small intestine converts the various carbohydrates into the monosaccharides,—galactose, levulose and dextrose—and the protein-derivatives into amino-acids, in which form they are absorbed.

The peristalsis of infancy is less active than in later childhood or adult life, and the combination of this condition with the great relative length of the intestine probably gives the infant an especial ability to digest and absorb the large amount of milk taken. Independently of this, the infant possesses a very active power of absorption. This takes place principally from the small intestine, whence all the elements of the food enter the system, the fat passing in practically only from this region. The large intestine absorbs all, but to a very limited degree. The time of passage through the entire gastro-intestinal tract varies, according to Kahn,[1] from 4 to 20 hours, with an average of 15 hours. The results of Hymanson's[2] experiments on healthy nurslings in the early months of life gave very similar results. Lesné, Binet and Paulin[3] by adding small amounts of carmine to the infant's feedings, found the average time of passage at from 1 to 2 years 10 hours, the duration being less in younger subjects and in the breast-fed.

Bacteria of the Gastro-intestinal Tract. **Mouth.**—A considerable number of species of bacteria appear in the mouth even very soon after birth, and with the beginning of the taking of food increases decidedly. With the appearance of the teeth there is a further increase in the number of varieties. Among those oftenest found by Nobécourt and de Vicariis[4] are the bacillus lactis aërogenes, micrococcus candidans, bacillus coli, micrococcus pyogenes aureus and albus, and the streptococcus pyogenes and salivaris.

Stomach.—The number of bacteria found normally in the stomach is limited, the hydrochloric acid having destroyed many of those swallowed with the food. Yet there are many contained within the masses of coagulated milk, which the acid cannot affect (Tobler).[5]

Intestine.—The healthy normal infant exhibits soon after birth a considerable number of species of bacteria in this region. The subject was originally investigated especially by Escherich[6] and later by Tissier,[7] Moro[8] and others. After breast-feeding commences the number of germs increases greatly, although the varieties are not numerous. In general the most prominent bacteria in the intestinal tract in breast-fed infants are the bacillus bifidus (Tissier), the bacillus acidophilus (Moro), and to a less degree the bacillus coli and the bacillus lactis aërogenes. There are also sometimes present the bacillus perfringens, bacillus butyricus, micrococcus ovalis, streptococci, and certain others in smaller numbers. The upper part of the small intestine exhibits very few germs of any sort. The bacillus lactis aërogenes and the colon bacillus in small numbers are those chiefly found. The former is more abundant here than in any other portion of the intestinal canal. The lower part of the ileum and the colon contain an abundant growth of bacteria, the bacillus bifidus being the predominating germ. There are also present the bacillus acidophilus, the colon bacillus, the micrococcus ovalis, and a diminished number of the bacillus lactis aërogenes. The colon and rectum have a smaller number of living bacteria than is found in the cecum.

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[1] Zeitschr. f. Kinderheilk., 1921, XXIX, 321.

[2] Amer. Jour. Dis. Child., 1916, XI, 112.

[3] Arch. de méd. des enf., 1920, XXIII, 449.

[4] Arch. gén. de méd., 1905, CXCVI, 3201.

[5] Ergebnisse d. inn. Med. u. Kinderheilk., 1908, I, 495.

[6] Darmbakterien des Säuglings, 1886.

[7] Comptes rend. soc. de biol., 1899, VI, 943. XIII Internat. Med. Cong., 1900 Méd. de l'enf, 208.

[8] Jahrb. f. Kinderheilk., 1905, LXI, 687; 870.

In artificially fed infants the variety of germs is greatly increased. The colon bacillus and intestinal cocci are the predominating microörganisms, but the others mentioned are also present in large numbers. The colon bacillus and the bacillus lactis aërogenes are more numerous than in breast-fed children, and the bacillus aërogenes capsulatus is regularly present (Knox and Ford).[1] Bahrdt and Beifeld[2] emphasize the fact that in the breast-fed infants the germs are especially those producing fermentation; while in the bottle-fed the process is largely decomposition. The bacilli of the breast-fed infants tend to be Gram-positive, and those of the artficially fed infants Gram-negative. Very similar results were obtained by Bosworth and his colleagues.[3] Kendall[4] maintains that the character of the food is largely instrumental in determining the nature of the germs which will be found in the intestinal canal, the relatively high percentage of carbohydrate in human milk and of protein in cow's milk being the deciding elements. Bass[5] found that with the ingestion of large amounts of lactose a rapid increase in the numbers of bacillus acidophilus took place. The studies of Sisson[6] and of Ford, Blackfan and Batchelor[7] are rather at variance with this. The latter observers found that the germs present in the intestine were very much those which were ingested with the food.

Under pathological conditions there may be an enormous increase in the number of the normal bacteria, and various others are present, not native to the intestine; while at the same time germs which are normally present and harmless may assume a special virulence and be productive of diseased conditions. Among those found are the bacillus proteus vulgaris, bacillus enteritidis, streptococcus enteritidis, bacillus pyocyaneus, and forms of the dysentery-bacillus.

Gases of the Digestive Tract.—The gas in the stomach is in part swallowed by the infant while nursing, and in part enters from the intestine. But little is produced by fermentation of the food, and a small quantity appears to be secreted by the gastric mucous membrane. In healthy children it consists of the elements of the atmospheric air only.

The gas in the intestine depends principally on the decomposition of the milk-sugar and consists of CO_2 and H (Escherich).[8] There are no foul-smelling gases in the intestine of milk-fed infants who are in a healthy condition.

Feces.—The first passages of the infant consist of the meconium. This is a tarry, dark, greenish-brown, almost odorless and faintly acid substance. It is sometimes passed before or during birth, and 3 to 5 times during the first 2 or 3 days of life. It contains cells from the intestine and the skin, minute hairs, fatty granules and globules, cellular detritus, and intestinal mucus, together with biliary acids, coloring matter, cholesterin-crystals and other substances derived from the bile.

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[1] Johns Hopk. Hosp. Bull., 1915, XXVI, 27.
[2] Jahrb. f. Kinderheilk., 1910, LXXII, Ergänzungsh., 71.
[3] Amer. Jour. Dis. Child., 1922, XXIII, 323.
[4] Wisconsin Med. Jour., 1913, XII, 1.
[5] Southern Med. Jour., 1923, XVI, 1.
[6] Amer. Jour. Dis. Child., 1917, XIII, 117.
[7] Amer. Jour. Dis. Child., 1917, XIV, 354.
[8] Darmbakterien des Säuglings, 1886, 160.

The source of some of the elements is the amniotic liquid which the fetus has swallowed from time to time. Vierordt[1] estimates the total amount of meconium passed as equalling 60 to 90 grams (2.12 to 3.17 oz.). Should the secretion of milk be delayed, the meconium is replaced after 2 or 3 days by stools consisting of brownish or greenish mucus. On the 3d or 4th day, or sometimes earlier or later, the ordinary milk-feces of the infant appear. These are golden-yellow or canary-yellow in color, of salve-like consistence or of that of thin mush, and faintly acid in odor and reaction. When cow's milk is the food employed the stools are alkaline (Biedert),[2] neutral or faintly acid, of a paler yellow color, have a more unpleasant odor due to the decomposition of the protein, and are often more consistent. Thoroughly digested breast-milk stools appear almost entirely homogenous (Fig. 12). The reaction is, however, according to Schlossmann[3] not necessarily an indication of the health of the infant, nor is it dependent upon the actual amount of the different ingredients in the food as much as upon the relationship between them. A ratio of 3 to 1 between the fat and the protein produces an acid stool, while one of 1 to 1 makes the stool alkaline. Consequently an alkaline reaction of the stools of the bottle-fed infant is a natural result of a relatively high percentage of protein as compared with that of the fat. The acid reaction is commonly attributed to the lactic acid and the fatty acids present. Bosworth and his coworkers[4] claimed that the acid reaction of the stool in healthy breast-fed infants was dependent upon the presence chiefly of formic and acetic acids. When the stool of healthy bottle-fed infants was acid in reaction it was due to acetic acid. Tisdall and Brown[5] have observed that the pH of the stools of normal new-born infants varies from 4.7 to 5.1. An acidity as great as 4.6 was encountered only in severe diarrheal conditions in bottle-fed babies. Infants fed on protein milk, cow's milk dilution with added carbohydrate, or whole soured milk with added carbohydrate, generally have stools more alkaline than pH 7.0. Very numerous small whitish masses are very common in the stools of infants apparently entirely healthy and thriving; and this is especially true of those artificially fed. A brownish color may depend upon a relatively large production of protein. Mucus is frequently visibly present even in the stools of healthy breast-fed infants.

The number of intestinal evacuations is at first from 2 to 4 in 24 hours, and after about the 6th week and up to the age of 2 years from 1 to 3. There may be, however, considerable variation from these figures within the bounds of health. The amount of fecal matter passed is estimated by Uffelmann[6] to be about 3 per cent. of the milk taken, or, in the case of feeding with cow's milk, 4.3 per cent.; averaging 3 grams per kilo (21 grains per lb.) of the body-weight, but with a wide range among individual cases.

The stools contain approximately 85 per cent. of water (85.13 per cent. Wegscheider).[7] The greater part of the residue consists of cellular elements, mucus and bacteria. The milk taken is never wholly absorbed. Fat is always present in the feces, both as neutral fat in the form of fatty acids, and as soaps from combination of these with the alkalies and

[1] Gerhardt's Handb. d. Kinderkrankh., 1877, 1, 118.

[2] Die Kinderernährung im Säuglingsalter, 1900, 58.

[3] Centralbl. f. Kinderheilk., 1906, XI, 237.

[4] *Loc. cit.*

[5] Amer. Jour. Dis. Child., 1924, XXVII, 312.

[6] Deutsch. Arch. f. klin. Med., 1881, XXVIII, 442.

[7] Ueber die normale Verdauung bei Säuglinge, Strassburger Dissertation, Berlin, 1875. Ref. Vierordt, Daten u. Tabellen, 1906, 306.

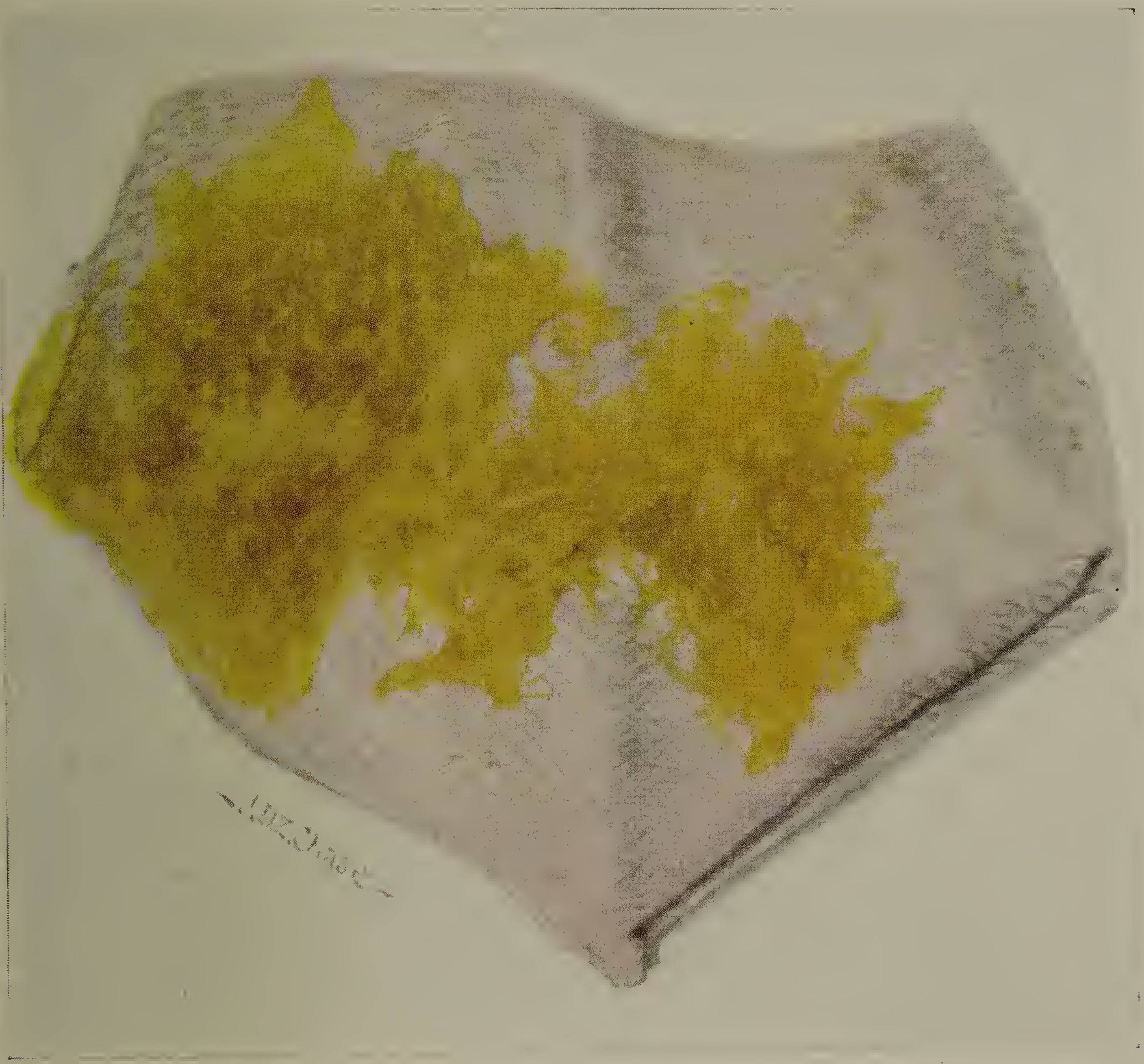

FIG. 12.—THOROUGHLY DIGESTED BREAST-MILK STOOL.

alkaline earths. With potassium and sodium an excess of fat forms soft, white curds; with calcium and magnesium insoluble soap-stools are produced. The fat forms from 9 per cent. to 25 per cent. or even more of the dried feces, according to various statistics collected by Biedert.[1] Holt, Courtney and Fales[2] found it averaging 34.5 per cent., and it was often even 50 per cent. of the dried feces in breast-fed infants. In the stools of infants taking the ordinary modifications of cow's milk the average fat equalled 36.2 per cent. The sugar of the food is entirely absorbed. Protein is present only in evry small amount in the stools of healthy infants. Knöpfelmacher[3] found that the greater portion of the nitrogen and phosphorus recovered from the feces of breast-fed infants is derived from the digestive secretions and not from the milk ingested. The protein of this latter has been in part absorbed, in part broken up by the action of bacteria. From 8 to 10 per cent. of the dried feces consists of mineral matter, chiefly calcium, derived partly from the food, partly secreted by the intestine. The proportion of mineral matter is higher in artificially fed infants (Blauberg;[4] Heubner[5]). The small, whitish masses very frequently present in the bowel-movements of healthy, thriving children consist principally of fat or its derivatives and of epithelial cells. The yellow color depends upon the occurrence of bilirubin which is present unchanged in part. The pale greenish tint so frequently appearing in the stools of healthy children a short time after they are passed, or even present at first, is the result of the oxidation of bilirubin to biliverdin. Such chemical combinations as phenol and skatol, which give the characteristic odor to the stools of adults, are not found in those of milk-fed infants. Various ferments are present in the feces, among them diastase, lactase, invertin, trypsin, rennin, a fat-splitting ferment, and others (Hecht).[6]

Under the microscope the bowel-movements of the infant exhibit fat-globules of various sizes; some molecular fat; needles of fatty acids; innumerable bacteria; cholesterin-plates; square and columnar epithelial cells; small round cells; some thin, granular, yellow, flake-like masses; lime-salts in crystalline form, and occasionally bilirubin-crystals, yeast-fungi, and proteid matter.

The evacuations become a somewhat darker yellow as the infant grows older; and, when the diet is more varied, and especially when the amount of milk is relatively diminished, they acquire more the characteristics of the stools of adults, both in color and odor. They are still soft, however, as a rule, and acid in reaction. It is only at about the age of 2 years that the stools become formed. This is, however, open to many exceptions, for even young infants may normally pass fully formed stools.

Bacteria of the Feces. (See also Bacteria of the Intestinal Tract p. 36.)—The meconium is at first sterile, but within 24 hours microörganisms enter by the mouth and anus, although not found in large numbers. After breast-feeding begins they are very numerous, but not in great variety. In fact a large part of the stools in infancy is composed of bacteria. Leschziner[7] found that the dried substance of the normal stool of breast-fed infants contains from 2 per cent. to over 28 per cent. of germs; and Strasburger[1] obtained as much as 42.3 per cent. in normal stools in artificially fed children. In the normal feces of the breast-fed infant are found especially the bacillus bifidus, as well as the bacillus coli, bacillus acidophilus, butyric acid bacillus and sometimes the bacillus lactis aërogenes and others. In the artificially fed infant the bacillus bifidus loses its predominance and is present in association with large numbers of the colon bacillus, bacillus acidophilus, bacillus lactis aërogenes, intestinal cocci, and others, no one type dominating the picture, and the number of varieties being greater than in breast-fed infants. The distinction is to be made between the fermentative and the putrefactive bacteria. Germs of the first class, such as the bacillus bifidus and the bacillus lactis aërogenes, are fermentative and break up milk-sugar into lactic acid and gases, giving an acid reaction to the stool; while the proteolytic bacteria produce, among other actions, decomposition of the protein and give rise to an alkaline reaction. Which variety predominates depends upon whether the diet is largely carbohydrate or largely protein. After a mixed diet is commenced there is a further change in the intestinal flora, with an increase in the variety of the microörganisms in the stools.

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[1] *Loc. cit.*, 61.
[2] Amer. Jour. Dis. Child., 1919, XVII, 241; 423.
[3] Jahrb. f. Kinderheilk., 1900, LII, 545.
[4] Zeitschr. f. Biol., 1900, XL, 1; 36.
[5] Verhandl. d. Gesellsch. f. Kinderheilk., 1901, XVIII, 230.
[6] Die Faeces des Säuglings und des Kindes, 1910, 148.
[7] Deutsch. Aerzte-Zeitung, 1903, V, 385.

ABSORPTION AND METABOLISM OF THE FOOD

In addition to and summarizing some of the statements already made under Digestion (p. 33) a brief résumé may be given of the physiological processes which attend and follow this, viewed from the point of view of the food-elements rather than of the organs, and as applicable especially to infancy. A very large amount of investigation in this direction has been made during recent years. For further consideration the reader is referred to the numerous journal-articles upon the subject, and especially to the publications of Tobler and Bessau,[2] Czerny and Keller,[3] Langstein and Meyer,[4] and Morse and Talbot.[5] (See also pp. 128–133.)

Fat.—The amount of fat in the food has a decided influence upon the time this remains in the stomach, as shown by the investigations of Tobler,[6] the larger the amount of fat, the slower being the discharge. This slow entrance into the small intestine permits of a readier digestion of it when it reaches this region. The fat is in no way affected by the saliva, but in the stomach the gastric lipase is able to break up a considerable portion of it. Comparatively little digestion of fat, however, occurs here, and no absorption at all. In the small intestine the fat is split by the pancreatic lipase in combination with the bile-salts and aided by the intestinal secretion, the fatty acids uniting with the alkalies and forming soaps, soluble and insoluble, and later an emulsion. It is in this form that the fat is absorbed by the small intestine, more than 90 per cent. of that ingested both in breast-fed and in normal bottle-fed infants, being utilized. Very little fat is absorbed by the large intestine. It is questionable whether the neutral fat as it occurs in the milk ingested is absorbed at all in this form. There is reason to believe, however, as claimed by

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[1] Zeitschr. f. klin. Med., 1902, XLVI, 433.

[2] Brüning u. Schwalbe, Handb. d. allgem. Path u. d. path. Anat. des Kindesalters, 1912, I, 650.

[3] Des Kindes Ernährung, etc., 1906.

[4] Säuglingsernährung u. Säuglingsstoffwechsel, 1914.

[5] Diseases of Nutrition and Infant Feeding, 1920.

[6] Ergebnisse der inn. Med. u. Kinderheilk., 1908, I, 514.

Kastle and Loevenhart[1] that the lipase in the intestinal mucous membrane may change the soaps back into neutral fat and that it enters the lacteals in this form. The fat appearing in the stools may possibly be derived in part from the intestinal secretion and from decomposition of the carbohydrates; but probably much the largest portion is from the food. The absorption of fat in artificially fed infants can be modified by altering the proportion of the other elements of the food. Thus in some cases an addition of carbohydrate apparently increases the absorption of the fat and changes a soap-stool to one of a more normal acid character; and, on the other hand, an increase of the protein may similarly change a highly acid, loose stool into a firmer, more alkaline one. Giffhorn[2] found soap-stools altered in character by the addition of whey to the diet. The fat appears to have little favorable influence upon the metabolism of protein, but is of importance in mineral metabolism, especially aiding in the absorption of calcium (Holt and Fales).[3]

Since the discovery of the vitamins the importance of sufficient fat in the diet has sprung into increased prominence, and a certain reaction has taken place in the opinions which had been prevailing. For a considerable time it had been regarded by many as a matter of indifference whether fat or carbohydrate was employed, provided only a sufficient number of calories was obtained. There is now a very noteworthy and most desirable tendency to recognize in fat an indispensable quality, not only on account of the fat-soluble vitamin contained, but for other reasons.

In the economy one of the chief purposes of the fat is to maintain the body-heat. It is an element of the food which it is very difficult to do without for any length of time, for since its caloric value is twice that of either protein or carbohydrate, to replace it an undue amount of one or the other of these must be given.

Carbohydrate.—The only carbohydrate present in milk is lactose; a disaccharide, resolvable into dextrose + galactose (Reuss).[4] In artificially fed infants the other disaccharides, saccharose (dextrose + levulose) and maltose (dextrose + dextrose), are often employed. Starch, too, a polysaccharide, is to be considered, and in older children cellulose. On the sugar the saliva has no action whatever, nor has the secretion of the pancreas, unless maltose is employed, when there may be a slight reduction by the maltase as found by Ibrahim.[5] The starch is converted by the salivary, pancreatic and intestinal secretions into sugar; although some part of it is probably destroyed in infancy by bacterial action within the intestinal canal. The power of digesting starch rapidly increases with an increase of the quantity and strength of the amylolytic ferments. The invertin, maltase and lactase of the intestinal canal act respectively upon the saccharose, maltose, and lactose, reducing them to monosaccharides, in which form alone are they absorbable under ordinary conditions. Only when given in very large amounts do the disaccharides pass the normal intestinal mucous membrane. They then appear unchanged in the urine, with the exception of maltose, which may be broken up by the maltase of the blood and utilized. The monosaccharides are absorbed rapidly by the small intestine, especially in the upper part, and carried by the portal circulation to the liver where they are changed into glycogen. The large intestine also possesses to some extent the power of the reduction of the disaccharides and of absorption.

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[1] Amer. Chem. Jour., 1900, XXIV, 491.
[2] Jahrb. f. Kinderheilk., 1913, LXXVIII, 531.
[3] Amer. Jour. Dis. Child., 1922, XXIII, 471.
[4] Wien. med. Wochenschr., 1910, LX, 1635.
[5] Verhand. d. Gesellsch. f. Kinderheilk., 1908, XXV, 32.

The different sugars are utilized by the infant to different degrees. All of them are fermentable, but lactose undergoes lactic acid fermentation, saccharose more readily alcoholic and less easily butyric acid fermentation, and maltose most easily butyric acid and next readily alcoholic fermentation. Maltose can be assimilated in larger amount than lactose; the latter is absorbed more slowly. All the sugars in large amount have a tendency to loosen the bowels, but lactose to a less degree than maltose. The normal infant can digest from 3.1 to 3.6 grams of lactose per kilogram of its body-weight (22 to 25 grains per lb.) and about 7 grams of maltose per kilogram (49 grains per lb.) (Hill).[1] Kahn[2] gives the tolerance for lactose as from 6 to 11 grams per kilogram of body-weight (42 to 77 grains per lb.). Feldmann[3] places the daily tolerance for saccharose at 7.2 grams per kilogram of body-weight (50.4 grains per lb.), and Schick[4] found that 17 per cent. of cane-sugar added to half milk was readily tolerated by new-born infants as a temporary substitute for human milk. It was even possible[5] to add the 17 per cent. of sugar to human milk, making the total sugar 24 per cent.

The assimilation, or at least the retention, of the nitrogen of the protein is rendered more complete by the presence of carbohydrates, as shown by Keller,[6] Orgler,[7] Talbot and Hill[8] and others. The carbohydrates would seem, too, when not in excess to favor the absorption of fat, perhaps by preventing the formation of calcium and magnesium soaps. In excess they interfere with the absorption of fat by producing diarrhea, by which the fat and other intestinal contents are rapidly removed from the body. After absorption the carbohydrates serve to maintain the heat of the body. In addition they have an action in preserving the proper metabolism of the fats, which without carbohydrates are productive of acetone bodies. In the stools the carbohydrates appear chiefly in the form of undigested starch, when this is given in unduly large amount to infants. Very little soluble carbohydrate is found. The gas in the intestine is dependent in part upon the decomposition of the carbohydrate by bacteria. This decomposition also aids in producing the acidity of the stools, which is in direct proportion to the amount of the carbohydrate ingested. The carbohydrate of the fetus is exhausted soon after birth, and acidosis may threaten. Hence undue crying is to be avoided in the new-born, or going without food for too long a period (Benedict and Talbot).[9]

Protein.—The digestion of the protein takes place, as stated, partly in the stomach, under the influence of rennin, pepsin and hydrochloric acid, but chiefly in the intestine by the trypsin of the pancreas and the erepsin of the intestinal secretion. The action of this latter is upon the casein and the peptones and albumoses, changing them into amino-acids, in which form they are absorbed and utilized in the body. It would not appear to be a matter of indifference in what form the protein is present in the food. The value of the large percentage of whey in human milk may depend upon the fact that amino-acids are present in the whey in large amount, but in quite small quantity in the casein, and that those in the whey are necessary to growth. According to Osborne and Mendel[1] lactalbumin is a more efficient protein than casein, 9 per cent. of the former being sufficient for growth, whereas from 15 to 18 per cent. of the latter is necessary. There is almost complete absorption of the protein in normal and normally fed infants, the dried stools showing only from 4 per cent. to 4.5 per cent. to be nitrogen (Orgler),[2] and even this is chiefly derived from the intestinal secretions and from dead bacteria. The curds in the stools are composed of casein to a very limited extent; the casein-curds being hard and tough, and being usually absent in the case of healthy infants. They are less liable to appear when the milk has been boiled (Brennemann).[3]

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[1] Bost. Med. and Surg. Jour., 1918, CLXXIX, 1.
[2] Jahrb. f. Kinderheilk., 1921, XCIV, 15.
[3] Zeitschr. f. Kinderheilk., 1921, XXVIII, 325.
[4] Zeitschr. f. Kinderheilk., 1918, XVII, 1.
[5] *Ibid.* Zeitschr. f. Kinderheilk., 1920, XXVII, 57.
[6] Czerny and Keller, *loc. cit.*, 1906, I, 305.
[7] Jahrb. f. Kinderheilk., 1908, LXVII, 390.
[8] Amer. Jour. Med. Sci., 1914, VIII, 218.
[9] Carnegie Instit. Washingt., 1915, Publ. No. 233

The needs of the infant in the matter of nitrogen derived from the protein are not great, and the giving of an excess of this is not required. Whether protein is capable of being harmful has been disputed. Certainly it can usually be tolerated in large amounts, and the excess is then utilized in the economy for the production of calories. (See also p. 132.) At least 1.5 grams per kilogram (10.5 grains per lb.) of the body-weight is required daily to maintain a positive nitrogen-balance in the system; or in other words 7 per cent. of the caloric requirements should come from the protein. This is approximately equivalent to 1½ oz. of breast-milk for each pound of the weight of the infant (98 c.c. per kilo). Probably 2 grams per kilogram (14 grains per lb.) would be a safe and a satisfactory figure. However, the production of calories by the consumption of an excess of protein is poor policy in healthy infants, since the work thrown upon the economy and the increase of consumption of heat-units in metabolizing the protein are very much greater than when fat or carbohydrate is employed for the purpose (Lusk).[4] While but 7 per cent. of the lactose ingested is consumed in making the remaining 93 per cent. ready for use, 20 per cent. of the protein is used up in making the other 80 per cent. available (Talbot).[5]

The maintaining of the nitrogen-balance does not depend solely upon the amount of protein ingested. Infants may retain nitrogen even when the number of calories in the food administered is insufficient. While, as stated, the giving of carbohydrate in proper amount favors the retention of nitrogen, the fat has little if any favorable action, and in excess may act unfavorably. An increase in the retention of nitrogen is not necessarily attended by a gain in the body-weight. Inasmuch as all proteins must be reduced to amino-acids before they can be absorbed, there would appear to be no difference in the effects produced by them in entirely healthy infants, except, as stated, in the greater amount and different character of the amino-acids present in some of them. Yet in spite of the large tolerance of proteins shown by most children, there is ample reason to believe that these, especially as seen in the case of the casein of cow's milk given in excess, are capable of producing decided nutritional disturbance. This has been emphasized by a number of investigators. The subject is reviewed, among others, by Benjamin[6] and by Talbot and Gamble.[7] (See also p. 132.)

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[1] Jour. Biol. Chem., 1924, LIX, 339.
[2] Ref. Thiemich in Feer's Lehrb. d. Kinderheilk., 1914, 14.
[3] Amer. Jour. Dis. Child., 1911, I, 341.
[4] Science of Nutrition, 1917, 3rd Edit., 238.
[5] Arch. of Pediat., 1919, XXXVI, 373.
[6] Zeitschr. f. Kinderheilk., Orig., 1914, X, 185.
[7] Amer. Jour. Dis. Child., 1916, XII, 333.

It has also been claimed that whereas breast-fed infants received but 1 or 1.5 per cent. of protein, as contained in human milk, bottle-fed infants need 3 per cent. or more (Holt and Fales)[1] on account of the fact that casein is the dominant protein of cow's milk, and that this substance is deficient in the necessary amino-acids required for growth, notably cystin, lysin, glycocoll and tryptophan. While this in theory would seem to be correct, the lack of sufficient accurate knowledge of the actual protein-needs is proven clinically by the vast numbers of infants who have thrived perfectly, as far as can be discovered, on cow's milk mixtures containing less than 2 per cent. of total protein. As a matter of fact we have as yet no actual knowledge of the quantity of the vital amino-acids required by an infant, and can therefore come to no conclusions regarding the total amount and the variety of protein necessary. Whether it is possible for the ingested protein to be absorbed unchanged by the mother, appear in this form in the breast-milk, and cause allergic phenomena in the infant is still an unsettled question. (See Allergy, p. 750.)

Mineral Matter.—The salts of human milk (see p. 95) are readily absorbed from the small intestine, but to a negligible degree from other regions. They may in part re-enter the intestine from the circulation; and in addition salts are contained in the digestive secretions. Consequently, although they are absolutely necessary for digestion and metabolism, their action is complicated and not as yet thoroughly understood. The greater quantity of salts, except iron, in cow's milk as compared with human milk results in the amount taken by the infant artificially fed being usually in excess of the needs. In general the absorption and retention of the mineral matter of the food goes hand in hand with that of the nitrogen. Under normal conditions 40 per cent. of the mineral of the ingested cow's milk is lost in the stools. This is chiefly calcium phosphate (Holt, Courtney, and Fales).[2] The presence of a sufficient amount of fat in the food increases the retention of the mineral matter (Hoobler).[3] An excess of it may increase the loss of calcium and of magnesium in the feces by the production of soaps in large amount. There should be ingested at least 0.13 gram of calcium oxide for each kilogram of body-weight (0.91 grain per lb.) (Holt, Courtney and Fales),[4] and there should be about 4 grams of fat per kilogram of body-weight (28 grains per lb.) to insure a proper metabolism of the calcium. Wang, Witt and Felcher,[5] however, claim that although the percentage of breast-milk calcium retained and utilized is relatively greater than is the case with that of cow's milk, with the latter the actual quantity absorbed and retained is greater. Sodium and potassium are absorbed well from the intestine. They are eliminated by the urine and feces. Phosphorus is absorbed better from human milk, since a greater proportion of it is in organic combination than is the case with cow's milk. According to Hoobler[6] an increase of the fat in the food favors its absorption. The retention of calcium is aided by carbohydrates unless they are in excess, when the diarrhea resulting causes a decided loss of all mineral matter, although chiefly the potassium and sodium. Calcium may be absorbed either from organic or inorganic combinations, and the part of it which is not retained is excreted through the urine or through the intestinal wall into the feces. The retention of magnesium is, according to Hoobler[1] better when the fat-percentage is low. With all these substances, as with sulphur, the absorption and retention appear to be better in infants receiving human milk.

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[1] Amer. Jour. Dis. Child., 1921, XXII, 371.
[2] Amer. Jour. Dis. Child., 1915, IX, 213.
[3] Amer. Jour. Dis. Child., 1911, II, 107.
[4] *Loc. cit.*, 1920, XIX, 97.
[5] Amer. Jour. Dis. Child., 1924, XXVII, 352.
[6] *Loc. cit.*

GASEOUS METABOLISM; ENERGY METABOLISM

An increasingly large amount of investigation has been directed to this subject for several years. Most of the results are too technical for inclusion here, and but a brief sketch can be given of some of the data obtained. In the section upon Substitute Feeding (p. 120) reference will again be made to the direct application of the physiological data to the preparation of the food for an artificially fed infant. The reader is referred to the journal-literature which will be mentioned, and especially to the chapter upon Energy Metabolism in the text-book of Morse and Talbot[2] and to the later journal-article by Talbot,[3] for a condensed review of the subject.

Inasmuch as the excretion of carbonic dioxide is, as shown by Rubner and Heubner,[4] on the one hand in proportion to the body-surface, and, on the other, an index of the amount of nourishment required, it is evident that the number of calories demanded is directly related to the body-surface of the infant. Since, however, the estimation of the surface is a matter difficult or impossible to compute by the clinician, it has become customary to make the weight of the infant the basis. This can very readily lead to considerable error, inasmuch as it is possible for an infant to lose many pounds of its body-weight, and yet to retain, of course, the same body-surface. Further, it is now doubtful whether even the body-surface is to be considered the guide, but rather the "actual mass of the protoplasmic tissue" (Benedict and Talbot).[5]

In calculating the caloric requirements of the infant it may be reckoned that the basal metabolism of an infant of any age, *i.e.*, the consumption of energy during a state of complete repose after taking food, is equivalent to from 52 to 63 calories per kilogram (24 to 29 calories per pound) of the body-weight. This constitutes the lowest number of calories on which the continuance of health is possible, and only under this condition of inactivity, and does not allow for growth. An exception to this is in new-born infants, as well as in older infants decidedly above the average weight for their age. Here the basal metabolism is usually from 40 to 52 calories per kilogram (18 to 24 calories per pound) of body-weight. Muscular exercise, crying, and the like may increase the energy-requirements occasionally as much as 100 per cent. (Benedict and Talbot).[6] The sick and inactive infant produces less heat than the healthy, lively one. The former may exhibit no greater heat-production than the basal metabolism; the latter decidedly more. The new-born requires a minimum of fewer calories probably on account of this inactivity; and the extra-fat child also, probably partly on this account and partly because its weight is out of proportion to its body-surface. Emaciated infants have a basal metabolism above the figures given, varying from 63 to 87 calories per kilogram (29 to 39 per pound) (Morse and Talbot). This is, however, provided they cry considerably. If very weak and quiet the figures are not so high.

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[1] *Loc. cit.*

[2] Diseases of Nutrition and Infant Feeding, 1920, 64.

[3] Phys. Rev., 1925, V, 477.

[4] Zeitschr. f. Biol., 1898, XXXVI, 1; 1899, XXXVIII, 315.

[5] Amer. Jour. Dis. Child., 1914, VIII, 1.

[6] Carnegie Instit. Wash., Pub. 201, 97. Ref. Morse and Talbot, Diseases of Nutrition and Infant Feeding, 1920, 69.

Heubner[1] determined that the average healthy breast-fed infant in its 1st half-year required 100 large calories per kilogram (45 per pound) of body-weight in order to gain properly, the number diminishing gradually in the 2d half-year. This he denominated the "energy-quotient." Premature infants and those under 3 months artificially fed have an energy-quotient of 120 calories (54 per pound). During the first 6 days, however, the normal breast-fed infant requires only 62 calories per kilogram (28 per pound) (Benedict and Talbot).[2] In premature infants the basal metabolism appears to be extremely low (Talbot and Sisson;[3] Murlin and Marsh[4]). The figures of Heubner have been largely followed by clinicians in determining the food-requirements of infants. They are, however, far from being universally accepted. Cramer[5] found that the energy-quotient for the new-born was less than 50 calories (23 per pound). Czerny and Keller[6] believe Heubner's figures too high, and reported an instance of satisfactory progress on an energy-quotient of 70 (32 per pound); and Ramsey and Alley[7] came to much the same conclusion; while Ladd[8] gives from 93 to 159 calories per kilogram (42 to 72 calories per pound) as the range of the energy-quotient in a series of cases studied by him. Other figures could be quoted varying from those of Heubner. The fact appears to be that the energy-quotient can be regarded as no more than an average one and at the best a rough estimate; and it will vary greatly with the individual child, depending largely upon its degree of activity and its age. Heubner's figures may be taken as a guide for the normal child in the 1st half-year; the number of calories required after this gradually decreasing to 70 or 80 per kilogram (30 or 36 per pound) by the end of the 1st year; but children under weight may need from 130 to 150 calories per kilogram (59 to 68 per pound) of body-weight (Morse and Talbot) in the 1st half-year.

Regarding the relationship of the elements of the milk to the caloric needs, although certain numbers of heat-units are produced in the calorimeter by the combustion of these, the figures do not correspond with the "utilizable" calories supplied when the milk is ingested. According to the values given by Rubner[9] 1 gram of protein ingested produces 4.1 utilizable large calories; 1 gram of fat 9.3 calories, and 1 gram of carbohydrate 4.1 calories. Knowing the percentage of each of these present in the milk-mixture selected and the amount of this taken, it is a simple procedure to calculate the total number of calories received by the infant during the day, and to determine whether the food meets the energy-requirement. There is, it is true, some difference between the energy-production of the different sugars respectively and of starch, but this is small enough to be disregarded. The further adaptation of the knowledge of the caloric value of the food to infant-feeding will be considered in discussing that subject (p. 122). The caloric needs of children older than 1 year are also considered later (p. 187).

[1] Jahrb. f. Kinderheilk., 1910, LXXII, 121. Lehrb. d. Kinderheilk., 1911, I, 50.
[2] Carnegie Instit. Washington, 1915, Publ. No. 233, 126.
[3] Proc. Soc. for Exper. Biol. and Med., 1922, XIX, 309.
[4] *Ibid.*, 431.
[5] Münch. med. Wochenschr., 1903, L, 1153.
[6] Des Kindes Ernährung, etc., 1906, I, 383.
[7] Amer. Jour. Dis. Child., 1918, XV, 408.
[8] Arch. of Pediat., 1908, XXV, 178.
[9] Zeitschr. f. Biol., 1885, XXI, 377.

ORGANS OF RESPIRATION

Upper Respiratory Passages.—The **nasal passages** in the infant are very narrow, and the **sinuses** are for the most part not yet fully developed. In careful studies Davis[1] found the maxillary sinus present at birth and pneumatization of the lateral ethmoidal mass usually complete. The sphenoidal sinus is very small in the 1st year, and the frontal sinus not present before the 3rd year. The **larynx** is situated high in the neck, the lower border of the cricoid being opposite the upper border of the 5th cervical vertebra, instead of opposite the 7th as in adults. By puberty it has descended to the adult position and in boys increases much in size. The space between the vocal cords of the infant is extremely narrow. The bifurcation of the **trachea** is opposite the 3d dorsal vertebra in the new-born, but opposite the 4th in adults.

Lungs.—The lungs of the infant at term lie in a collapsed condition at the back of the thorax. After air enters them they are still small in volume as compared with adult life, and continue so throughout childhood. They weigh at birth about 24 grams (0.85 oz.), the relative weight being about the same as in adult life. The lower level during infancy is not quite as low in relationship to the ribs as it is in adult life (Gittings, Fetterolf, and Mitchell).[2]

Respiration.—The type of respiration in the infant is generally described as abdominal. The careful experimental studies of Eckerlein[3] however, show that it is thoracic as well, neither type preponderating constantly, but sometimes one and sometimes the other being evident. The respiration in infancy, and especially in the new-born, is very irregular, and the rate is much influenced by the slightest causes. At times quite long pauses take place. This irregularity is perhaps the most striking feature. It is almost constantly present when the child is awake, and may occur even during sleep.

The average rate of respiration in early life can be determined only approximately, and the estimations of investigators differ widely. The following table of the rate at different ages contains average figures only:

Table 24.—Rate of the Respiration per Minute

New-born	30 to 50.	Average about 35 to 40.
Balance of 1st year	25 to 35.	Average about 30.
1 to 2 years	About 28.	
3 to 4 years	About 25.	
4 to 15 years	20 to 25.	
Adult life	16 to 18.	

The rate of respiration is from 1/5 to 1/4 less during sleep. Only with the beginning of childhood does the irregularity largely disappear, but even then the rate may be much increased by comparatively slight causes. It is not until the 10th year that the predominating costal type of breathing begins to develop in girls. The vital capacity is constantly greater in boys than in girls.

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[1] Development and Anatomy of the Nasal Accessory Sinuses in Man, 1914; Annals of Otol., Rhinol. and Laryngol., 1919, XXVII, 940.

[2] Amer. Jour. Dis. Child., 1916, XII, 579.

[3] Zeitschr. f. Geburtsh. u. Gynäk., 1890, XIX, 120.

ORGANS OF CIRCULATION

Heart. **Size.**—The heart in the new-born weighs about 24 grams (0.85 oz.) according to the statistics of H. Vierordt[1] as compared with the weight of between 260 and 300 grams (9.17 and 10.58 oz.) in the adult. It is thus proportionately larger in the infant, equalling 0.76 per cent. of the body-weight in the new-born, and 0.46 per cent. in the adult. This disparity is, however, not so marked after the 1st month of life, from which time the heart grows nearly in proportion to the increase in the body-weight, except that at puberty there is a physiological hypertrophy. The right ventricle is comparatively large and strong in early infancy, its walls being almost as thick as those of the left ventricle (v. Starck).[2] From the 2d year onward, however, the muscle-mass of the right ventricle is not more than ½ that of the left. The foramen ovale—the oval opening between the auricles—exists still at birth. It is situated at the lower posterior portion of the auricular septum. From the anterior border of the inferior vena cava arises a thin membrane, the Eustachian valve, which during fetal life diverted the blood from this vessel through the foramen ovale into the left auricle. With the beginning of respiration at birth, and the cessation of circulation in the umbilical vein, blood ceases to pass through this foramen to any extent, and it gradually becomes closed entirely by about the 10th day of life, or often not for some months. (See Vol. II, p. 376.)

Position.—The position of the heart is rather more horizontal in the infant. This previously accepted view has been confirmed by the x-ray studies of Bamberg and Putzig.[3] Regarding its percussion-boundaries the opinions of writers vary considerably. Von Starck's[4] review of these, with his personal observations, make the relative dullness—which is the most important—begin in the 1st year at the 2d left interspace or, oftener, the 2d rib, extend to the right parasternal line, and reach as far as from 1 to 2 cm. (0.4 to 0.8 inch) beyond the left nipple-line. The width at the position of the nipple is 6.6 to 8 cm. (2.6 to 3 inches). At the age of 6 years the highest extent in percussion-dullness is to the 2d intercostal space. It reaches to the right scarcely as far as the parasternal line, and to the left it extends to the nipple-line or slightly beyond it. The greatest average breadth is 10.2 cm. (4.02 inches). At 12 years the relative dullness is bounded by the 3d rib, the right edge of the sternum and the left nipple-line. Its greatest breadth is 11.5 cm. (4.5 inches). The absolute dullness in the 1st year is bounded by the lower edge of the 3d rib, the left border of the sternum and the left nipple-line.

Apex.—The apex-beat in the 1st year is generally found in or oftener beyond the mammillary line, but quite frequently its position cannot be determined. After this period, according to von Starck, although it may lie in various positions with regard to the mammillary line, it is oftenest outside of it up to the 4th year; on the line from this time up to the 7th year, and within it after the 9th year. It is nearly always in the 4th interspace in the 1st year, generally so in the 2d year, in the 4th and 5th interspaces from the 3d to the 6th years, and generally in the 5th interspace after the 7th year. The position of the apex is, however, subject to great variations within physiological limits. A distance of 2 cm (0.8 inch) outside the nipple line is to be looked upon, however, with suspicion.

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[1] Arch. f. Anat. u. Entwickelungsgesch., 1890, Suppl. Bd., 62.
[2] Arch. f. Kinderheilk., 1888, IX, 247.
[3] Zeitschr. f. Kinderheilk., 1919, XX, 195.
[4] *Loc. cit.*, 241.

Auscultation.—This shows all sounds to be loud, sharp and distinct, and more widely diffused than in adults. This is in part owing to the thinness of the chest wall. In infancy and up to the age of 4 or 5 years the second sound is weak over the aortic cartilage, and loudest over the pulmonary cartilage, but is everywhere weaker than the first sound of the heart. These conditions are entirely different from those obtaining in adult life (Hochsinger).[1]

Blood-vessels.—The blood-vessels in the child are relatively of somewhat greater capacity than in the adult. The pulmonary artery is decidedly larger than the ascending aorta, while in adult life they are of nearly the same size (Beneke.)[2] The relative size of the heart as compared with the diameter of the ascending aorta is much less in children than in adults (Beneke). As a consequence of these two relations the general arterial tension is less than in adult life (see p. 50), and the blood-pressure in the lungs in childhood is greater than that of the general arterial system.

At the point where the pulmonary artery divides into its two main branches springs in the fetus the ductus arteriosus Botalli. Although the largest branch, this vessel is but a short trunk about ½ inch (1.3 cm.) in length at birth. It passes obliquely upward and joins the aorta just below the origin of the left subclavian artery. Since with the beginning of respiration the blood is diverted from the ductus arteriosus into the lungs, this canal closes in from 1 to 2 weeks, persisting only as a small fibrous cord in later life. Among other vessels characteristic of antenatal life, and still present at birth, are the hypogastric arteries and their continuation, the umbilical. These are stout trunks arising from the internal iliac arteries and passing upward beside the bladder to and through the navel, whence, twisted around the umbilical vein, they reach the placenta. The distal portions of the arteries within the body of the child close completely in from 2 to 5 days after birth, forming fibrous bands, the anterior ligaments of the bladder. The proximal portions remain pervious for only a short distance from their origin, as the superior vesical arteries. The umbilical vein after entering the body from the navel passes upward along the free margin of the suspensory ligament of the liver. After giving off small branches to the hepatic substance it divides in the transverse fissure into two main branches, of which, the larger and shorter, joins the portal vein. The other, the ductus venosus, continues along the posterior longitudinal fissure of the liver and joins the hepatic vein where this empties into the ascending cava. Both the umbilical vein and the ductus venosus close completely in from 2 to 5 days after birth, the part of the former within the body finally becoming the round ligament of the liver.

The activity of the circulation is greater in the child. K. Vierordt[3] estimates that the time elapsing from the moment the blood leaves the heart until it returns to it is in the new-born 12.1 seconds, at 3 years 15.0 seconds, at 14 years 18.6 seconds and in the adult 22.1 seconds.

The circulation during fetal life and the changes which take place at birth have so intimate a relation to congenital diseases of the heart that they will be described in connection with cardiac disorders.

Pulse.—Even in health the pulse tends to be somewhat irregular in force and frequency. It has been the general impression that this was true especially of infancy, but the electrocardiographic studies of Krumbhaar and Jenks[1] and of Seham[2] would indicate that arrhythmia is uncommon in infancy, but becomes increasingly frequent from the age of 6 years to puberty, 47 per cent. of school-children showing an arrhythmia, according to Seham. It cannot be called pathological. Trifling causes, such as crying, nursing, or excitement or exercise of any other kind increase the pulse-rate from 20 to 40 beats per minute. It is often not easy to feel the radial pulse in the first months. The rate is less when the child is lying than when sitting, and sitting than when standing, and is 16 to 20 beats less during sleep. It diminishes also with increasing age, but the figures of different observers vary much. An approximation would be expressed by the following:

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[1] Die Auscultation des kindlichen Herzens, 1890.

[2] Constitution u. constit. Kranksein, 1881. Ref. Vierordt, Daten u. Tabellen, 1906, 171.

[3] Gerhardt's Handb. d. Kinderkrh., I, 107.

Table 25.—Pulse-Rate During Infancy and Childhood

Birth	130 to 150
1st month	120 to 140
1 to 6 months	About 130
6 to 8 months	About 120
1 to 2 years	110 to 120
2 to 4 years	90 to 110
6 to 10 years	90 to 100
10 to 14 years	80 to 90

The rate is slightly greater in females except in the first few months. Townsend[3] has shown that at birth sex has no influence on the rapidity. Dicrotism is absent or imperfectly developed in the pulse of children, and does not appear until the age of from 10 to 14 years (Keating and Edwards).[4]

Blood-pressure.—The blood-pressure is lowest in children, and exhibits little if any increase until puberty is approached, when a more abrupt increase occurs. Probably the most extensive and carefully conducted studies are those by Judson and Nicholson,[5] and their results may be taken as the most reliable. These are shown in comparison with the investigations of others in Table 26, an extension of that given by them. The authors[6] found it impossible to determine any reliable average normal readings for subjects under 3 years of age. More recently, however, Rucker and Connell[7] using the oscillatory method with an aneroid sphygmomanometer observed that the mean systolic pressure at birth was 55 mm. of mercury and the mean diastolic 40 mm., while Seitz and Becker[8] obtained an average systolic blood-pressure of 43 mm. on the 1st day after birth, by the end of the 1st week 60 mm., and at the end of the 1st month 82 mm. The figures in the table indicate measures recorded in millimeters of mercury.

BLOOD

Blood. Amount, Specific Gravity and Volume.—The blood in the new-born constitutes about 1/15 of the body-weight against 1/13 in adult life. If tying of the cord is deferred the percentage is temporarily

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[1] Heart, 1917, VI, 189.
[2] Amer. Jour. Dis. Child., 1921, XXI, 247.
[3] Bost. Med. and Surg. Jour., 1896, CXXXIV, 484.
[4] Arch. of Pediat., 1888, Dec.
[5] Amer. Jour. Dis. Child., 1914, VIII, 257.
[6] Personal communication.
[7] Amer. Jour. Dis. Child., 1924, XXVII, 6
[8] Zentralb. f. Gynäkol., 1920, XLIV, 1338.

TABLE 26.—BLOOD-PRESSURE IN NORMAL CHILDREN

Age	L. Gordon[1]		Stowell[2]		M. Leitão[3]		W. Kaupe[4]		W. Kriss[5]		A. Katzenberger[6]		Judson and Nicholson[7]		Faber and James[8]	
	S.	D.	S.	D.	S.	D.	S.	D.	S.	D.	S.	D.	S.	D.	S.	D.
3 years	81	..	91	..	91	65	90	81.8	80	74	.....	....	91.8	65.6	...	..
4 years	83	..	89	..	99	71	86.4	78.3	83	76	93	54	91.6	64.9	89	60
5 years	86.5	..	95	..	99	71	82.2	74	83	76	94.5	54	91.3	64.4	92	62
6 years	88.5	..	96	..	..	..	97.5	82.1	90	82	82	53	92.6	67.3	94	64
7 years	85	..	102	..	..	..	91	78	90	82	.....	....	94	66.3	97	65
8 years	93	..	101	..	..	..	90.5	80.2	90	88	103	55	93.6	64.7	100	67
9 years	100	..	102	..	..	..	90	81.5	90	88	100.6	58.6	94.3	71	101	68
10 years	95	..	112	..	..	..	97.7	82.6	98	90	104.6	69.5	99.2	67.1	103	69
11 years	104	..	102	..	..	..	96	87.2	98	90	99.8	78.8	97.1	65.5	104	70
12 years	105	..	111	..	..	..	92	83.1	99	95	108.2	68.2	102.3	65.2	106	71
13 years	.....	..	107	..	..	..	96.6	88	99	95	107.7	70.2	103.6	70.5	108	72
14 years	.....	..	110	..	..	..	107	96	101	96	119.2	71.2	106.1	67.4	110	73
15 years	.....	..	109	..	..	..	.....	....	101	96	.....	....	105.6	67.5	112	75

[1] Arch. of Pediat., 1911, XXVIII, 343.
[2] Arch. of Pediat., 1908, XXV, 91.
[3] Arch. d. méd. des enf., 1913, XVI, 102.
[4] Monatsschr. f. Kinderheilk., Orig., 1910–11, IX, 257.
[5] Arch. f. Kinderheilk., 1910, LIII, 332.
[6] Zeitschr. f. Kinderheilk., Orig., 1913, IX, 167.
[7] Amer. Jour. Dis. Child., 1914, VIII, 257.
[8] Amer. Jour. Dis. Child., 1921, XXII, 7.

increased to ⅑ of the body-weight (Schücking).[1] Bakwin and Rivkin[2] and Lucas and Dearing[3] estimated the blood-volume in infants by the vital-dye-method. The latter found the average from 15 days to 1 year to be 10.9 per cent. of the body-weight. In the new-born there was great variation with an average of 14.7 per cent. of the body-weight. The specific gravity in the new-born is estimated by Lloyd Jones[4] to be highest at birth, equalling 1066, but by the end of the 2d week it has fallen rapidly and continues to decrease until the age of 2 or 3 years when it equals 1048 or 1050. These figures are practically those for the adult's blood, which, according to Macleod,[5] varied from 1040 to 1065. The normal blood-concentration of the new-born, as demonstrated by Lust,[6] gave 22.3 per cent. of dry substance during the period of from 1 to 30 days after birth, and 18 per cent. from the age of 1 to 10 months.

Hemoglobin.—The percentage of hemoglobin is generally conceded to be higher in the new-born, being then decidedly over 100 per cent. It diminishes rapidly and reaches its minimum in the first 3 months, falling almost to 50 per cent. This low percentage continues for the first 6 months or 1st year, after which it rises slowly. Throughout childhood a percentage of 75 has not been considered low enough to indicate anaemia. Appleton,[7] who has studied the haemoglobin percentage by an accurate method (Palmer),[8] gives the following figures: 1st day 164; 1st week 135; 2nd month 102; 5th month 88; 1st year 87; 2nd year 85. These figures are certainly higher than those obtained with the relatively inaccurate clinical methods in common use, but correspond quite well with the actual amounts of haemoglobin found by Williamson.[9]

Erythrocytes (see also Vol. II, p. 757 and Fig. 412).—The number of red blood-corpuscles varies greatly according to different statistics. It is relatively high in the new-born, exceeding the proportion in adult life. Stengel and White[10] give 5,742,080 per c.mm. as the average at birth obtained from the statistics of a number of observers, and Mayers[11] 7,630,000. A decrease in number begins after the 2d day and goes on rapidly, about 500,000 being lost in the first 2 weeks (Schiff).[12] The diminished number continues to exist during the 1st year, and then there follows a gradual increase up to the age of from 8 to 12 years, when the number normal for adults is attained (Stengel and White). The average number of red blood-cells in early and later childhood is 4,000,000 to 4,500,000. Nucleated red blood-corpuscles (normoblasts) occur in the fetus, and in small numbers during the early days of life, in the proportion of about 6 to every 100 leucocytes (Slawik),[13] but after the 1st week their presence is unusual. The size of the red corpuscles varies very much in the new-born, pale corpuscles deprived of their hemoglobin (shadow-corpuscles) are present in considerable numbers (Silbermann)[14] and poikilocytes may be seen. When the fragility is tested in normal children it is found that hemolysis begins in 0.425 per cent. sodium chloride solution and is complete in one of 0.325 per cent. Reticulated red cells, which normally appear in the adult's blood in the proportion of 0.5 to 2.0 per cent. of the total red cells are present in larger numbers in the blood of new-born infants.

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[1] Berl. klin. Wochenschr., 1879, XVI, 582.
[2] Amer. Jour. Dis. Child., 1924, XXVII, 340.
[3] Amer. Jour. Dis. Child., 1921, XXI, 96.
[4] Jour. of Physiol., 1887, VIII, 4; 1891, XII, 299.
[5] Physiol. and Biochem. in Modern Med., 1918, 87.
[6] Jahrb. f. Kinderheilk., 1911, LXXIII, 85.
[7] Jour. Biol. Chem., 1918, XXXIV, 369.
[8] Jour. Biol. Chem., 1918, XXXIII, 119.
[9] Arch. Int. Med., 1916, XVIII, 505.
[10] University of Pennsylvania Medical Bulletin, 1901, November, 310.
[11] Arch. Int. Med., 1922, XXX, 478.
[12] Zeitschr. f. Heilk., 1890, XI, 17.
[13] Zeitschr. f. Kinderheilk., Orig., 1920, XXV, 212.
[14] Jahrb. f. Kinderheilk., 1887, XXVI, 252.

Leucocytes (see also Vol. II, p. 758 and Fig. 412).—The leucocytes are present in relatively large numbers in the new-born, equalling 16,000 to 20,000 or more to the c.mm. in the 1st days of life, but diminishing to 12,000 or 13,000 by the age of 2 weeks (Schiff).[1] There is little change from these figures during the 1st year of life (Gundobin),[2] but after this period the number steadily falls, averaging according to Karnizki[3] approximately 9500 at from 1 to 6 years, and 8000 at from 6 to 15 years. Rabinowitsch,[4] however, found the total average number of leucocytes in the first 15 years from 6000 to 7000 as in adults. There may be a very considerable variation within physiological limits. Perlin[5] gives approximately 8000 to 13,000 as the number at from 4 to 8 years of age, and 7000 to 9000 at 16 years of age. The proportion among the different forms of leucocytes is very variable in infancy and childhood. At birth the polymorphonuclear neutrophiles constitute nearly 75 per cent. (Carstanjen)[6] but within 48 hours the lymphocytes begin to increase in number. Gundobin gives the proportion of lymphocytes and allied forms in infancy as 50 to 60 per cent. and that of polymorphonuclear neutrophiles as 28 to 40 per cent., and these figures may be assumed as approximately correct. In adults they are 24 to 38 per cent. and 62 to 70 per cent. respectively. These proportions alter gradually, and by the age of 3 years they approach to, and by the age of 8 or 10 years do not differ materially from, those of adult life. The number of eosinophiles is much more variable in the blood of infants and children, and may be considerably increased even up to 6 per cent. (Carstanjen) without having any pathological significance. In the first weeks of life a few myelocytes may be found and even after this period they may occur in a large variety of pathological conditions (Zelenski and Cybulski).[7] A description of the different varieties of red and of white cells and their significance will be given under Diseases of the Blood (Vol. II, p. 757).

Blood-platelets.—The normal number of platelets ranges from 300,000 to 450,000 to the c.mm. Even in the new-born these counts are usual, although the platelets may occasionally fall as low as 100,000 (Morse).[8] Keilmann[9] thinks that the number in infancy must be below 40,000 or above 200,000 to be considered pathological.

Bleeding-time.—The bleeding-time normally varies from 1½ to 3 minutes (Lucas et al.).[10] In the new-born it may be prolonged to 25 minutes (Sherman and Lohnes).[11] The method of determining the bleeding-time is described later (p. 213).

Coagulation-time.—This varies, according to the method used, from 3 to 9 minutes, with an average of about 5 minutes. From the 1st to the

[1] *Loc. cit.*
[2] Jahrb. f. Kinderheilk., 1893, XXXV, 191.
[3] Arch. f. Kinderheilk., 1903, XXXVI, 42.
[4] Arch. f. Kinderheilk., 1912, LIX, 161.
[5] Jahrb. f. Kinderheilk., 1903, LVIII, 549.
[6] Jahrb. f. Kinderheilk., 1900, LII, 215.
[7] Jahrb. f. Kinderheilk., 1904, LX, 884.
[8] Bost. Med. and Surg. Jour., 1912, CLXVI, 448.
[9] Monatsschr. f. Kinderheilk., 1922, XXIII, 383.
[10] Amer. Jour. Dis. Child., 1921, XXII, 525.
[11] New York State Med. Jour., 1923, XXIII, 146.

5th or even the 10th day of life the coagulation-time is normally somewhat prolonged, even as much as from 10 to 16 minutes according to Sherman and Lohnes.[1] Rodda,[2] however, from a study of 126 new-born infants concludes that the coagulation-time at this period normally ranges from about 5 to 9 minutes, with an average of 7 minutes. A figure of more than 10 minutes indicates a pathological prolongation, as in the haemorrhagic diathesis. The method of determining the coagulation-time is described later (p. 213).

Iso-agglutinins and Iso-hemolysins.—Both iso-agglutinins and iso-hemolysins are present in the blood of even the youngest infant, thus making it advisable to type the blood in the selection of a donor for transfusion (Jones;[3] McQuarrie;[4] Happ and Zeiler[5]). DeBiasi,[6] however, claims that mothers may act as donors for their new-born infants without compatibility tests for agglutination and hemolysis (p. 214).

Blood-chemistry.—Blood-chemistry has become an important factor in diagnosis, prognosis and the direct treatment of such diseased conditions as nephritis, diabetes, rickets, tetany, and dehydrated and acidotic states in infancy. The accompanying table has been compiled from many sources.

TABLE 27.—CHEMISTRY OF THE BLOOD

	Infants	Children
Blood protein	6.0 – 6.5 %[7]	7.5 – 8.5 %[7]
Total solids (by weight)	16. –18. %[8,9]	16. –18. %[8,9]
Total nitrogen (per 100 c.c. serum)	0.911 – 1.198 gm.[9]	
Nonprotein nitrogen (per 100 c.c. blood)	0.02 – 0.04 gm.[9,10,11,12]	0.02 – 0.04 gm.[9,10,11,12]
Urea nitrogen (per 100 c.c. blood)	0.005 – 0.015 gm.[9]	0.012 – 0.02 gm.[5 13]
Uric acid (per 100 c.c. blood)	0.0006– 0.0038 gm.[11]	0.0006– 0.0048 gm.[11]
	0.0013– 0.0068 gm.[10]	0.0026– 0.0059 gm.[10]
Amino-acid nitrogen (per 100 c.c. blood)	0.0044– 0.0069 gm.[10]	0.004 – 0.007 gm.[10]
Creatinin (per 100 c.c. blood)	0.0012– 0.002 gm.[11]	0.0012– 0.002 gm.[11]
Creatinin + creatin (per 100 c.c. blood)		0.005 – 0.008 gm.[11]
Cholesterol (per 100 c.c. blood)	0.150 – 0.175 gm.[13]	0.150 – 0.175 gm.[13]
Chlorides (per 100 c.c. blood)	0.500 – 0.600 gm.[13]	0.500 – 0.600 gm.[13]
Carbon dioxide (per 100 c.c. plasma)	46– 76 vol. %[14]	60 vol. %[13]
Sugar	0.08 – 0.13 %[13,15,16]	0.08 – 0.13 %[13,15,16]
Hydrogen ion concentration	7.4 – 7.6[13]	7.4 – 7.6[13]
Calcium (per 100 c.c. serum)	0.010 – 0.011 gm.[17]	0.010 – 0.011 gm.[17]
Inorganic phosphorus (per 100 c.c. serum)	0.0045– 0.0055 gm.[18]	0.0045– 0.0055 gm.[18]
Potassium (per 100 c.c. serum)	0.018 – 0.022 gm.[19]	0.018 – 0.022 gm.[19]

Practically all these figures are somewhat higher in the first few days of life, due probably to blood-concentration.

The following tabular arrangement shows in convenient form the approximate normal number of the formed elements of the blood and the variations with increasing age.

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[1] *Loc. cit.*
[2] Amer. Jour. Dis. Child., 1920, XIX, 269.
[3] Amer. Jour. Dis. Child., 1921, XXII, 586; 598.
[4] Johns Hopk. Hosp. Bull., 1923, XXXIV, 51.
[5] Jour. Amer. Med. Assoc., 1924, LXXXII, 227.
[6] Jour. Amer. Med. Assoc., 1923, LXXXI, 1776.
[7] Utheim, Amer. Jour. Dis. Child., 1920, XX, 366.
[8] Courtney and Fales, Amer. Jour. Dis. Child., 1917, XIV, 202.
[9] Schloss, Amer. Jour. Dis. Child., 1918, XV, 165.
[10] Hoefel and Moriarity, Amer. Jour. Dis. Child., 1924, XXVII, 64.
[11] Schlutz, Trans. Amer. Ped. Soc., 1922, XXXIV, 283.
[12] Leopold and Bernhard, Amer. Jour. Dis. Child., 1916, XI, 432.
[13] Levinson, Jour. Lab. and Clin. Med., 1923, VIII, 525.
[14] Schloss, Amer. Jour. Dis. Child., 1917, XIV, 218.
[15] Mertz and Rominger, Arch. f. Kinderheilk., 1921, LXIX, 81.
[16] Muggia, Rivista di Clin. Pediat., 1924, XXII, 1.
[17] Tisdall and Howland, Amer. Jour. Dis. Child., 1921, XXII, 560.
[18] Howland and Kramer, Amer. Jour. Dis. Child., 1921, XXII, 105.
[19] Wilkins and Kramer, Arch. Int. Med., 1923, XXXI, 916.

Table 28.—Approximate Constituent Formed Elements of Normal Blood

	Birth	2nd–3rd day	2nd week	3 months–1 year	1–6 years	Puberty
Hemoglobin, per cent	100–110	100	55–60	50–65	65–75	85
R. B. C. number	5,500,000	5,000,000–5,500,000	5,000,000	4,500,000	4,500,000	5,000,000
W. B. C. number	15,000–20,000	12,000–15,000	12,000–14,000	12,000	10,000–12,000	7,000–9,000
Polymorphonuclears, per cent	70–75	60–65	35–40	30–35	40–45	65–70
Lymphocytes, per cent	20–25	30–35	40–45	50–60	40–45	20–30
Large mononuclears and transitionals, per cent	4– 8	5–10	10–12	8–12	3– 5	3– 5
Eosinophiles, per cent	5	5	4– 6	2– 3	2– 3	1– 2
Basophiles, per cent	0.5	0.5	0.5	0.5	0.5	0.5

GENITO-URINARY SYSTEM

Organs.—The **kidneys** are distinctly lobulated and comparatively large at birth and generally extend lower than the crest of the ilium, especially upon the right side. Their weight at birth is estimated by H. Vierordt[1] at about 23 grams (0.81 oz.) or 0.75 per cent. of the total body-weight, against 0.46 per cent. of the body-weight in adult life. A few years after birth the kidneys assume the position occupied in later life. The renal structure in the new-born often exhibits on section reddish-yellow streaks toward the apices of the papillæ. These consist of deposits of urates in the tubules, the uric acid infarcts described by Virchow.

The **bladder** in the infant is practically an abdominal organ, as the small pelvis is not capable of containing it. The **prepuce** has normally an extremely narrow orifice and is often more or less adherent to the glans. The cervix of the **uterus** is relatively long and the body small. The organ grows but little until puberty. It is more anteverted than in adult life. The **ovaries** lie in the abdominal cavity at birth and are relatively large (0.8 grams (0.03 oz.)). (Adult 7.5 grams (0.25 oz.) H. Vierordt.)[2] The **testicles** are comparatively small (0.8 grams (0.03 oz.)). They are generally found in the scrotum at birth. The **mammary glands** very frequently become somewhat swollen and begin to secrete a milk-like fluid when the infant is 3 or 4 days old. Lindig[3] found secretion in the glands in 82 of 100 infants examined. This secretion is most abundant about the 10th day of life, and may continue for several weeks. This condition of the breasts may be considered physiological. It occurs in either sex with equal frequency, and is independent of the general state of health. The fluid sometimes called "witch's milk," chiefly resembles in appearance and composition the colostrum secreted by the mother before the birth of the child (Genser).[4] Colostrum corpuscles are always present. The amount secreted is generally very small, unless the gland has been repeatedly irritated, as by pressure, when even as much as 1 or 2 c.c. (16 to 32 minims) may be obtained at a time.

Urine. **Amount.**—Urine is, as a rule, present in the bladder at birth, but it is in small quantity, and but little is secreted during the first 2 or 3 days of life. The average amount at the first passage is 9.6 c.c. (0.326 fl.oz.), according to the observations of Martin and Ruge.[5] The total daily quantity secreted in the first 2 days is inconstant, but averages much less than later. As soon as the child begins to obtain milk from the mother the secretion of urine is much increased, and continues throughout childhood to be proportionately greater than in adult life. The amount is exceedingly variable, and the statements of different investigators differ widely. It is influenced by many causes, among them the amount of liquid ingested, the temperature of the air, and the state of the digestion or of the nervous system.

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[1] Arch. f. Anat. u. Entwickelungsgeschichte, 1890, Suppl. Band, 62.

[2] Daten u. Tabellen, 1893, 29.

[3] Monatsschr. f. Geburtsh. u. Gynäk., 1918, XLVII, 534.

[4] Jahrb. f. Kinderheilk., 1876, IX, 160.

[5] Zeitschr. f. Geburtsh. u. Frauenkr., 1876, I, 279.

The following figures, based upon many estimations, are fairly representative approximations:

Table 29.—The Daily Secretion of Urine

1st and 2d days	15–60 c.c.	(0.5– 2.0 fl.oz.)
3d to 10th day	100–300 c.c.	(3.4–10.1 fl.oz.)
10th day to 2 months	250–450 c.c.	(8.5–15.2 fl.oz.)
2 months to 1 year	400–500 c.c.	(13.5–16.9 fl.oz.)
1 to 3 years	500–600 c.c.	(16.9–20.3 fl.oz.)
3 to 5 years	600–700 c.c.	(20.3–23.7 fl.oz.)
5 to 8 years	650–1000 c.c.	(22.0–33.8 fl.oz.)
8 to 14 years	800–1400 c.c.	(27.0–47.3 fl.oz.)

The studies of Churchill[1] gave amounts decidedly less than these. Fuller details may be found in the writings of Martin and Ruge,[2] Cruse,[3] Herz,[4] Schiff,[5] Camerer,[6] Lesné and Merklen[7] and others.

Frequency of Micturition.—This varies from 2 or 3 up to 6 times on the 1st and 2d days of life. Quite commonly the evacuation does not take place until more than 12 hours after birth, and not infrequently not until on the 2d or even the 3d day of life. After this excretion is very frequent during infancy, varying anywhere from 5 or 6 to even sometimes 30 or 40 times in the twenty-four hours, the urine often being retained several hours during sleep. Engel[8] studied the frequency of micturition by an automatically registering electrical apparatus, and found it varying from 10 to 30 times in twenty-four hours, with a normal average of 25. Pfeifer[9] estimated the average number at about 18 in twenty-four hours. After control of the bladder is obtained the frequency of urination varies from 6 to 8 times in twenty-four hours.

Physical and Chemical Characteristics of the Urine.—The *specific gravity* of the urine during the first few days of life is high as compared with that of later periods (1012, Martin and Ruge).[10] After the ingestion of milk begins it rapidly falls to 1002–1006, but when a mixed diet is commenced it gradually increases, and when the child is 5 or 6 years old the specific gravity is about the same as in the adult.

In *appearance* the secretion is at first highly colored and slightly turbid, owing to the concentration and to the presence of urates and mucus. Later, even during childhood, it is generally of a paler yellow than in adult life. Sometimes in infancy, particularly in the new-born, it stains the diaper a faintly reddish color through the deposition of urates. The *reaction*, at first decidedly acid, usually soon becomes neutral. *Odor* is almost absent in infancy and even in childhood, unless the urine is high-colored. The ammoniacal odor often noted in the nursery usually is due to lack of care in changing the diapers, the urine decomposing after it has been passed. This is especially true if indigestion is present. (See Vol. II, p. 435.)

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[1] Arch. of Pediat., 1898, XV, 646.
[2] *Loc. cit.*
[3] Jahrb. f. Kinderheilk., 1877, XI, 393.
[4] Wien. med. Wochenschr., 1888, XXXVIII, 1510.
[5] Jahrb. f. Kinderheilk., 1893, XXXV, 21.
[6] Würtemb. Correspondbl., 1876, XLVI, No. 11.
[7] Rev. mens. des mal. de l'enf., 1901, XIX, 61.
[8] Deutsch. med. Wochenschr., 1914 XL, 1960.
[9] Jahrb. f. Kinderheilk., 1917, LXXXVI, 220.
[10] *Loc. cit.*

As regards the *chemical constituents* there is very little or no urea in the urine at birth. The proportion is much increased by the 3d day, but is still relatively low during infancy. Phosphates, chlorides and sulphates are also present in relatively small amounts. The proportions of all of these are increased when a mixed diet is commenced, but are still less than in adults. The amount of urea, however, as compared with the body-weight is greater in childhood than in adult life. The percentage of uric acid is especially large in the new-born, and, though diminishing after this, still remains throughout childhood in excess of that of adult life. The relation of uric acid to urea is 1:14 in the new-born, and but about 1:70 in the adult. Creatin and creatinin are normal constituents.

The urine of healthy breast-fed infants usually contains no indican, but in those fed artificially it is generally present in small quantity. Older children on mixed diet exhibit indican to the same extent as do adults.

Albumin in small amount may very often be found in the urine of healthy new-born infants. Out of 61 subjects in this class, Ewald[1] found albumin in nearly all. It disappeared by the 12th day. Sugar may occur in healthy infants, according to the statements of a number of investigators, the amount depending upon the quantity of sugar ingested (Greenthal).[2] Reuss[3] found glycocol a normal constituent in the new-born, and Ostrowski[4] observed urobilinuria frequently in healthy infants. Rennin and pepsin are said by Pechstein[5] to be always discoverable in the urine of children. Small amounts of the acetone-bodies are normally present (Veeder and Johnston);[6] and phenol is constantly found (Moore).[7] *Microscopically* nothing characteristic is noticed except that the presence of hyaline casts is not unusual in the case of young infants.

LYMPHATIC GLANDS

Entirely apart from the occurrence of infectious processes in the glands, or even of the existence of the lymphatic diathesis, both of which are so common in children, the lymphatic glands are relatively numerous and well-developed in early life, and large enough even in infancy to be detected by careful palpation. Jensen[8] examined 240 infants of 9 days old or less, and found palpable glands in the neck in 43 per cent. Coerper[9] made a study of 1000 children up to the age of 2 years. In 100 new-born the axillary glands were palpable in 95 per cent. The cervical, inguinal, occipital and other glands were also often discoverable. As age advanced through the first 2 years the frequency of occurrence and the number of palpable glands increased; but some of this is to be considered as pathological.

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[1] Monatsschr. f. Geburts. u. Gynec., 1916, XLIII, 347.

[2] Amer. Jour. Dis. Child., 1920, XX, 556.

[3] Zeitschr. f. Kinderheilk., Orig., 1911–12, III, 12; 286.

[4] Przeglad lekarski, 1912, No. 17. Ref. Monatsschr. f. Kinderheilk., Ref., 1913, XII, 172.

[5] Zeitschr. f. Kinderheilk., Orig., 1911, I, 357.

[6] Amer. Jour. Dis. Child., 1916, XI, 291.

[7] Amer. Jour. Dis. Child., 1917, XIII, 15.

[8] Zeitschr. f. Kinderheilk., Orig., 1916, XIV, 176.

[9] Monatsschr. f. Kinderheilk., Orig., 1915, XIII, 458.

THYMUS GLAND

The thymus gland is essentially an organ of early life. Its size is subject to great variation. According to Friedleben,[1] whose figures have been much quoted, the length from birth to the 9th month averages approximately 6.9 cm. (2.71 inches); from 9 months to puberty 8.4 cm. (3.30 inches), and in adult life from 10 to 13 cm. (3.94 to 5.12 inches), the glandular tissue being then largely replaced by fat. At birth the gland fills up much of the space in the lower anterior portion of the neck and behind the upper part of the sternum. Its weight, according to his figures,[2] are:

Table 30

Birth	14.3 grams (0.51 oz.)
1–9 months	20.7 grams (0.73 oz.)
9–24 months	27.3 grams (0.96 oz.)
2–14 years	27.0 grams (0.95 oz.)
15–25 years	22.1 grams (0.78 oz.)
25–35 years	3.1 grams (0.11 oz.)

Some more recent investigations gave reason to believe, however, that these estimations are too high. The careful studies of Bovaird and Nicoll,[3] based upon the examination of 495 glands, furnish figures for weight decidedly less. The birth-weight was found to average 7.7 grams (0.27 oz.), with a decrease to 5.9 grams (0.21 oz.) for the first 5 years of life, and a still further dimunition after this period. Dudgeon[4] found the normal weight in infants up to 2 years of age to be from 7 to 10 grams (0.25 to 0.35 oz.). Sokolow[5] gives still lower figures for the first 2 years. Vierordt,[6] from his comparison of different statistics, placed the average birth-weight as 8.15 grams (0.29 oz.). On the other hand, Hammar,[7] in a study upon the thymus glands of 126 well-nourished individuals dying suddenly or after very acute illness, and consequently with presumably normal glands, found that the weight at birth averaged 13.26 grams (0.47 oz.); from 1 to 5 years 22.98 grams (0.81 oz.); from 6 to 10 years 26.10 grams (0.92 oz.), and from 11 to 15 years 37.52 grams (1.32 oz.). His later publications[8] confirm his claim that the gland is not a transitory organ, but that it functions through life, although diminishing in size after puberty. Yamanoi,[9] too, after the examination of 363 cadavers, observed more or less persistence of the thymus gland to adult age in 25.89 per cent. These views oppose the older ones that there is always a disappearance of the gland with increasing age. It is evident that a decided difference of opinion still exists which has yet to be settled; but it would seem safe to assume that any gland weighing more than from 10 to 15 grams (0.35 to 0.53 oz.) in the early months of life is above the average weight, although the possible range in normal weights is to be borne in mind (from 5 to 20 grams) (0.18 to 0.70 oz.) (Schridde).[10]

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[1] Die Physiologie der Thymusdrüse, 1858.

[2] As Friedleben's figures are given in the old grains of Cologne, I have used the equivalents as quoted by Friedjung (Pfaundler und Schlossmann, Handb. der Kinderheilk., 1906, II, 1, 394).

[3] Arch. of Pediat., 1906, XXIII, 641.

[4] Path. Soc. Transac., London, 1904, LV, 151.

[5] Jahrb. f. Kinderheilk., 1923, CIII, 157.

[6] Daten und Tabellen, 1906, 44.

[7] Ergebn. d. Anat. u. Entwickelungesch., 1909, XIX, 253.

[8] Endocrinology, 1921, V, 543.

[9] Schweizer med. Wochenschr., 1921, LI, 557.

[10] Münch. med. Wochenschr., 1914, LXI, 2161.

The weight of the thymus gland appears to be to a degree in proportion to the weight and general nutrition of the child. The gland is loosely attached to the sternum but firmly to the pericardium. Its position may be recognized by radiography as well as by percussion. (See Diseases of the Thymus Gland, Vol. II, p. 836.)

SUPRARENAL BODIES

The suprarenal bodies are relatively very large in infancy, weighing about as much as in adult life.

SPLEEN

The spleen weighs approximately 10 grams (0.35 oz.) at birth (Vierordt).[1] Its relative weight is slightly greater at this period than in adult life. (Adult 163 grams (5.75 oz.) Vierordt.) It is frequently palpable at or below the costal margin in early life. Even as late as the 10th year it may be felt in about 10 per cent. of normal children Zamkin).[2]

THYROID GLAND

The thyroid gland is comparatively large in infancy, weighing, according to the statistics of Vierordt,[3] 4.85 to 9.75 grams (0.17 to 0.34 oz.). This makes its size as compared with the body-weight 3 times as great as in adult life. The isthmus is small in children, but can be discovered by palpation, if the subcutaneous fatty tissue of the neck is not too abundant at the point where it crosses the trachea below the cricoid cartilage. The lateral lobes, however, cannot be felt, and even a somewhat enlarged gland may be impossible of recognition during life.

TEMPERATURE

The temperature of the body at birth, taken in the rectum, averages in the neighborhood of 37.8°C. (100.4°F.) (Vierordt).[4] Within an hour or two it falls temporarily about 1.7°C. (3.1°F.) and then, before or by the end of the 1st day of life, rises again to 37.6°C. (99.7°F.) (37.59°C. Förster).[5] Throughout infancy a daily rise in temperature begins at 2 or 3 A. M., which gradually increases and reaches its maximum in the early afternoon, to be followed by a fall which commences toward evening and continues until after midnight. The very extensive investigations of Jundell,[6] upon over 3000 records, show that the daily fluctuation in the second half of the 1st week in perfectly healthy infants does not amount to more than 0.1°C. (0.18°F.), by the age of 1 month averages about 0.25°C. (0.45°F.), and by 6 months reaches a range of 0.5°C. (0.9°F.). In early childhood the daily fluctuation amounts to nearly 1°C. (1.8°F.), which is somewhat greater than in adult life. The average temperature of childhood is rather higher than in adults (0.3°C. Vierordt) (0.54°F.), and elevations are more readily produced by slighter causes. After the ingestion of nourishment infants show a slight fall of temperature, followed soon by a slight temporary rise above that existing before nursing (Demme).[1] The temperature during sleep is slightly lower than when the infant is awake, and somewhat higher after exercise or crying. External influences, too, affect it. Thus variations in the temperature of the surroundings may elevate or depress that of the infant. It is to be noted also that the axillary temperature in normal children is from 0.3 to 0.9°C. (0.54° to 1.6°F.) less than the rectal (Demme), and in sick children from 0.5 to 1.1°C. (0.9 to 2°F.). The rectal temperature is, of course, to be considered the representative one.

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[1] Daten u. Tabellen, 1906, 29.
[2] Arch. of Pediat., 1926, XLIII, 169.
[3] Daten u. Tabellen, 1906, 43.
[4] Daten u. Tabellen, 1906, 360.
[5] Journ. f. Kinderkr., 1862, XXXIX, 1.
[6] Jahrb. f. Kinderheilk., 1904, LIX, 521.

NERVOUS SYSTEM

Brain.—The brain of the new-born is proportionately very heavy, equalling about 380 grams (13.4 oz.) or from 12 to 13 per cent. of the body-weight, while in adults it is only about 2 per cent. Growth is rapid, especially in the 1st year, the weight increasing nearly 2½ times. After about the 5th year, however, the rate of increase in weight is very slow.

The following table, after Vierordt,[2] shows the weight at different ages:

TABLE 31.—WEIGHT OF THE BRAIN AT DIFFERENT AGES

	Male	Female
Birth	381 grams (13.4 oz.)	384 grams (13.5 oz.)
6 months	632 grams (22.3 oz.)	575 grams (20.3 oz.)
1 year	945 grams (33.3 oz.)	872 grams (30.8 oz.)
2 years	1025 grams (36.2 oz.)	961 grams (34.0 oz.)
5 years	1263 grams (44.6 oz.)	1221 grams (43.1 oz.)
10 years	1408 grams (49.7 oz.)	1284 grams (45.3 oz.)
25 years	1431 grams (50.5 oz.)	1224 grams (43.2 oz.)

The cerebellum is relatively smaller than the cerebrum as compared with adult life. The brain-substance is very soft at birth, and the grey matter is not sharply differentiated from the white. Although the convolutions are less evident than in adult life they all become visible by the age of 5 weeks. The dura mater is adherent during the 1st and often also during the 2d year. There is a greater amount of fluid in the subdural space than later in life.

Spinal Cord.—The spinal cord at birth is comparatively heavy, weighing 5.5 grams (0.19 oz.) or 0.18 per cent. of the body-weight (Vierordt) against 0.06 per cent. in adults. At birth it extends downward sometimes to the 3d lumbar vertebra, but in other cases only to the 1st, as in adults.

DEVELOPMENT OF MUSCULAR AND NERVOUS FUNCTIONS

For the first few weeks of life the infant lies very still wherever placed unable to change its position, and sleeping most of the twenty-four hours. The action of the flexor muscles preponderates to some extent, and the hands are usually clinched much of the time, the head sunken forward, the back convex, and the forearms, thighs and legs flexed and drawn to the body. The head cannot be held erect, the alterations in the expression of the face are meaningless, and the apparent smile sometimes seen is not expressive of comfort. Any movements which occur are automatic or reflex. Sucking at the nipple, for instance, and the grasping by the hand of an object placed in it are done purely unconsciously. This automatic grasp of the new-born is very powerful; so much so that the child can sometimes be raised entirely from the bed before it relaxes. Soon a very distinct increase in general power takes place. The motions of the legs and arms become very active, but uncontrolled and still purposeless. In the 2d month the head can be held upright to some extent, and by the 3d or 4th month very well. By the age of 3 months, or sometimes a little sooner, purposeful efforts at grasping objects begin, but without any idea whatever of distance being shown. By 6 months, although the motions are still largely impulsive, the child can make many well-directed movements and can grasp for and play with its toys. At this age it can sit supported very well, and unsupported to some extent, although frequently falling backward until the age of 9 or 10 months. The age when the infant is able to roll over varies greatly. A few can accomplish this by 3 months, but the majority not until much later.

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[1] 14. med. Bericht. über d. Thätigkeit Jennersch. Kindersp. in Bern, 1877, 7. Ref. Vierordt, *loc. cit.*, 364.

[2] Arch. f. Anat. u. Entwickelungsgeschichte; Suppl. Band, 1890, 62.

At about the age of 6 months the infant will often try to stand if held on its feet in the lap. At 7 or 8 months it makes attempts at creeping or at moving along the floor or bed in some other manner. Some children, however, never creep. When a year old, or sometimes even when 9 or 10 months, it will stand, holding to objects. Walking while supporting itself by the wall or by furniture begins soon after 1 year of age, the time for this, as for standing, varying greatly, and the power to walk without support is gained in a few months more. Falls are, of course, very frequent, and these are nearly always backward, bringing the child into a sitting position. This is due to the comparative weakness of the extensor muscles. The toes are always turned in when walking, and this condition is overcome only very gradually.

The time for the acquisition of muscular and nervous control of the passage of urine depends largely upon training. With care it is sometimes possible to teach it to an infant of even 3 or 4 months, at least during the day. Usually, however, the control by day is not acquired until sometime in the 2d year. The age of 2 years is an extreme limit for children with whom any effort at instruction has been made. The same statements apply to the control of the fecal evacuations.

Reflex Action.—This is, for the most part, well-developed in the new-born, but subject to great variation. Many of its forms are entirely uncontrolled by the inhibitory influence which develops later; as, for instance, reflex evacuation of the bowels and the bladder. Some of the reflex movements which persist throughout life, as the plantar, Achilles, and patellar reflexes, are, on the whole, not so uniformly well-shown in the 1st and 2d years as later. Others, such as the abdominal, cremasteric, corneal and pupillary reflexes, are claimed to be fully developed from birth. The abdominal reflex is present only irregularly, according to Burr.[1] The lip-reflex is present in the new-born and gradually disappears with increasing age. The investigations of Engstler[2] and of Léri[3] showed that the plantar reflex in the new-born is characterized by *dorsal* flexion of the toes (Babinski reflex). Gradually this condition changes, but it is not until the 3d year that plantar flexion is the rule. This existence of early dorsal flexion is, however, not universally admitted (Feldman),[4] (De Angelis).[5]

[1] Amer. Jour. Dis. Child., 1921, XXI, 529.

[2] Wien. klin. Wochenschr., 1905, XVIII, 567.

[3] Gaz. des malad. infant., 1903, V, 277.

[4] Amer. Jour. Dis. Child., 1922, XXIII, 1.

[5] Amer. Jour. Dis. Child., 1923, XXVI, 211.

DEVELOPMENT OF SPECIAL SENSES AND MENTAL POWERS

Sight.—In the first weeks the child probably cannot see, except to distinguish light from darkness, and will not wink when the finger is brought near the eye. The perception of light is decided, and sometimes evidently unpleasant, since the infant closes its lids whenever the light is too bright. The eyes are expressionless and move slowly, and more or less of lack of coördination persists until the age of 3 months. The pupils react promptly to light at once after birth. Between the ages of 3 and 6 weeks the baby can fix its eyes upon objects, but even by the 6th day it may turn its face to the light. By 7 weeks the reflex closing of the lids on the approach of an object close to the eyes is well developed. Colors probably cannot be distinguished until the age of a year and the ability increases very slowly with many children. Yellow, white and red appear to be recognized before green and blue. In later childhood the power of sight is unusually strong, and light can be endured better than in adult life. Hyperopia seems to be the normal condition in the new-born.

Hearing.—This is absent on the day of birth, due probably in part to the filling of the tympanic cavity by mucus and swollen mucous membrane, in part to the approximation of the walls of the meatus. In a few days, however, air begins to enter the cavity and infants can then be awakened by loud noises. In the early months the sense of hearing is very acute and sleeping children are very easily awakened by noise, and they are especially sensitive to high and shrill tones. Infants of 3 months can generally recognize the direction from which sound comes and may turn the head toward it.

Musical tones are sometimes recognized between the ages of 1 and 2 years, and a child of 2 years may distinctly prefer one tune to another and may even know it by name. Very often, however, the ability to recognize tunes does not come until later in childhood, and sometimes never.

In later childhood the hearing is particularly acute, and very weak or very high tones are detected which an adult cannot hear at all.

Smell.—The sense of smell probably exists in the new-born, but is certainly slight, although infants born blind are said to be able to recognize the odor of milk. Except for the ability to differentiate pleasant from distinctly unpleasant odors, the sense of smell develops, on the whole, slowly, and is not fully present until later childhood.

Taste.—This is present at birth and the new-born can distinguish pleasant and unpleasant substances, such as sweet and bitter. Although the sense of taste during infancy does not always appear very keen, many infants taking without objection medicine which is generally considered decidedly unpleasant, this probably depends, as Preyer[1] has pointed out, on the fact that the taste for sweet is so remarkably developed that the infant often willingly takes anything to which sugar has been added in considerable quantity.

Touch.—This is present at birth, and the touching of the eyelashes, the lips or the hands promptly causes reflex movements. It is, however, much less strongly developed than later. Preyer found the forehead and the external auditory meatus especially sensitive. In older children the tactile sensibility is very acute. The sensibility to *pain* is quite distinct in young infants, but comparatively poorly developed if the area affected is small. Thus the pricking of the finger or toe during a blood-examination, or the scratching of the skin in vaccination frequently produces no crying.

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[1] Die Seele des Kindes, 90.

The *temperature-sense* in general, though not well developed, is still probably present in the new-born, as is shown by the comfort a warm bath gives, and the crying produced by chilling of the surface. In the mouth the temperature-sense is active from birth, as evidenced by the refusal of many infants to take cold milk, while warm is readily accepted.

Mental Powers.—The infant at birth is largely in a vegetative state, and its mental powers are dormant. On the whole its sensations are probably pleasurable, or at least not disagreeable, and those which are not are evidenced by a cry. Hunger, pain, cold, lack of sleep and the like are expressed in this manner without the infant being actually conscious that anything ails it. In the 2d month it expresses pleasure by smiling, as when tickled; but smiles before this age are merely reflex, and often indicative of pain. It is not until the age of 5 or 6 months that the average baby really laughs. In the 3d month there is distinct evidence of mind and thought. At this age the first signs of memory are witnessed, the child clearly recognizing its mother by smiling at her approach, or by ceasing to cry from hunger when preparations for nursing are witnessed. In the 3d or 4th month certain tones awaken its attention and it is interested in bright and especially in moving objects. It also shows its mental activity by grasping after objects, and by attempting, if it reaches them, to put them into its mouth. Before the age of 6 months the infant indicates its recognition of other persons as well as the mother by smiling at them, and realizes the difference between strange and familiar places. When 9 months old it will stretch out both hands intelligently, or will give its hand when told to do so, and enjoys a game of "peep-bo." It clearly understands many things spoken to it, even before it is able to speak any words itself. By the completion of the 1st year it has learned distinctly to indicate by expression of face and by gestures its likes and dislikes for the persons and acts of others.

In the 2d year the baby has some idea of numbers. Sensations of joy, anger, fear and the like are well-shown, but none of these make more than a most transient impression, and the child quickly passes from one to the other. In fact, memory in infancy and early childhood is but weak. Later in childhood, however, it is at its highest point.

Speech.—All early sounds made by the child are impulsive. In the 2d month the child often begins to use certain tones of voice, frequently of a "cooing" character, to express comfort, but these are still automatic in nature. About the age of 3 or 4 months the infant commences to utter a few different vowel-sounds preceded by certain consonants, especially m, and b; then d, p, n, and j. These sounds are still not in any way imitative or even selected; but by the age of 8 or 10 months several such syllables are pronounced with some evidence of intent, and by the end of the 1st year "mamma," "papa," and even some other words may be spoken intelligently. At 18 months the infant can express by gestures and a few words many of its desires, and by 2 years it employs very short incomplete sentences of two or three words, using nouns and verbs. Qualifying words of speech are learned later. The time at which speech is first acquired is, however, open to great variation within entirely normal limits.

CHAPTER II

HYGIENE

Prenatal Hygiene.—The health and manner of life of the prospective mother exercise enormous influence upon the well-being of the future child. Constitutional diseases, such as syphilis, affecting her and all acute or chronic maladies require treatment. The general hygiene must be overseen, and especially must the amount and nature of amusements and of exercise taken be carefully supervised. Thus the diet must be generous and digestible; the dress one which does not constrict; the condition of the breasts and nipples carefully attended to before the birth of the child; violent or sudden movements avoided; sufficient outdoor life obtained; the condition of the kidneys carefully watched; the nervous system maintained in a quiet state, and, in general, the hygienic instructions followed which are better detailed in works upon obstetrics.

First Care of the New-Born.—In from 5 to 10 minutes after birth, as soon as pulsation has ceased in the cord, a ligature of sterilized surgeon's silk is applied about 1½ inches (3.8 cm.) from the abdomen, and the cord then cut. (For further dressing of the cord, see p. 68.) The child is then wrapped in a soft and warmed blanket and laid in some warm and safe place for a short time until its toilet can be commenced. When ready for this the nurse seats herself on a low chair beside the baby's bath-tub, taking the child, still in its blanket, into her lap, and having the vessels of hot and cool water, the bath-thermometer, and other required articles close at hand. All draughts should be cut off by the use of a screen and by closing doors and windows, and the bathing done before an open fire or other source of heat, unless the weather is very warm. The eyes also should be protected against bright lights. The surface of the body is now rubbed with white petrolatum or olive oil to soften the vernix caseosa, particular attention being given to all the creases and folds in the skin. Next the eyes are washed with a saturated solution of boric acid in boiled water squeezed into them from absorbent cotton after separating the lids. Should the mother have had a suspicious vaginal discharge, a few drops of a 1 per cent. solution of nitrate of silver should be instilled with a dropper and this washed out later by normal salt solution; and it is a safer plan to use this or to instill a stronger solution of argyrol in every case. The interior of the nostrils is now gently cleansed, as far as possible, with absorbent cotton, and the mouth *very* gently with absorbent cotton wrapped around the nurse's finger or, better, around a small stick like a wooden tooth-pick, and moistened with warm sterilized water. So important is it that the mouth-washing be done gently that many pediatrists insist, with much reason, that it should not be done at all; since the slightest trauma of the mucous membrane produces an area ready to develop stomatitis. The face is washed with warm water applied with a sponge or wash-cloth, but without soap, and is then dried with a soft towel. The scalp is next soaped, washed and dried. The toilet of the head being now completed, the rest of the body is rubbed with soap and water, and the baby then placed in the tub filled with water at 100°F. (37.8°C.), kept there for a minute or two, and finally removed to the nurse's lap, where it is wrapped in a fresh flannel blanket, or in the flannel apron which it is advisable she should wear. Here it is patted thoroughly dry with soft towels, particular attention being given to all the folds and creases of the body, these parts being finally powdered slightly with an unscented talcum powder. The child is then dressed and placed in its bed. Should it seem chilled, as shown by blueness and coldness of the extremities and nose, it should have hot bottles put about it, using great caution against burning it. During the toilet it is important to keep the temperature of the bath uniform by adding hot water as required.

In the case of premature or weakly children it is best to omit bathing entirely until the vitality has become greater, and to substitute rubbing every two or three days with warm oil or petrolatum. (See p. 289.)

Certain matters appertaining to the child's toilet must be considered more in detail:

Bathing.—Succeeding baths resemble the first, except that the oiling is omitted. In place of the tub-bath, however, only a daily sponging is given until the cord has separated, in order that the dryness of its dressing shall not be disturbed. Throughout infancy and childhood the bath is given daily, either as soon as the child wakens in the morning or before the morning nap, but never soon after eating. The duration of immersion varies from 1 or 2 to 5 minutes, enough water being used to cover to the neck when the baby is in a semi-reclining position. The nurse, sitting on the right side of the infant, holds it in the tub by grasping its left shoulder and arm with her left hand, thus supporting its head and back on her left forearm. In some cases the reaction after the bath is unsatisfactory. It is then better to employ sponging only.

The temperature of 100°F. (37.8°C.) of the first full bath may be diminished gradually, until, when the age of 6 months or a year has been attained, it is from 90° to 95°F. (32.2° to 35°C.) in winter, or 85° to 90°F. (29.4° to 32.2°C.) in hot summer weather, the reaction of the child always being the guide. In the 2d year the temperature may be from 85° to 90°F. (29.4° to 32.2°C.), according to the effect on the child. After the 4th year the morning bath may be from 75° to 80°F. (23.9° to 26.7°C.) given as a sponge, shower, or tub-bath, with the duration brief, the room warm, and the drying vigorous. In this way it is generally a useful tonic. The temperature of the water should always be determined by a bath-thermometer; not guessed at, as is too often the case.

The *bath-tub* is commonly of painted metal, oval in shape. For the sake of greater convenience to the nurse or mother it may be placed upon a low stand when the bath is given. A very convenient tub is a folding one of rubber, but it is not as easily kept clean.

The *baby's basket* is employed to contain many of the articles commonly used in the toilet. It holds soap, hair-brushes, sponges, powder, and the like, and a certain amount of clothing. A *wash-cloth* is best suited for applying soap. It should be very soft, of flannel, diaper cloth, or cotton stockinet All folds and hollows of the surface should be thoroughly washed, but no effort made to cleanse the auditory canal.

For the removal of the soap a *sponge* is to be preferred, as water can be more easily squeezed from it upon the body. It should be of fine texture and free from all silicious particles. The sponge and wash-cloth may be kept in the pockets of the baby's basket, but only after they have been thoroughly dried.

The *soap* employed should be unirritating, and free from excess of alkali. Some of the unmedicated superfatted soaps are serviceable. Imported castile soap is an old favorite. All soap must be used cautiously, lest the skin become irritated.

Towels should be soft and absorbent. Well-washed and, preferably, old diaper-cloth constitutes one of the best materials. Later in life Turkish towelling is excellent. Young infants should be patted dry and then rubbed with the palm of the hand. Later, more vigorous drying with the towel may be employed. As the skin of the infant is extremely sensitive, dusting it with some absorbent *powder* after bathing is advisable, especially in all the folds and hollows. For this purpose talcum or starch is useful. The addition of any perfume is unnecessary and not advisable. Occasionally the application of a very small amount of petrolatum is advantageous if the skin seems too dry.

Local Toilets.—The *eyes* of the young infant should be washed daily with boric acid solution during the first days of life. They should be carefully protected against excess of light on account of the sensitiveness to it which exists especially in the new-born and even into the 2d month of life and longer. Later in infancy and childhood care must still be taken that the child does not injure its eyes by light too intense, insufficient, or badly placed, and the possibility of errors of refraction existing must be borne in mind, lest serious trouble arise. The *mouth* of the young infant may be very gently washed once or twice a day, with absorbent cotton wrapped around the little finger or a wooden toothpick or applicator and moistened with sterilized water, but this should be done with the greatest caution, inasmuch as the mucous membrane of the mouth in infancy is extremely sensitive. The advisability of omitting the mouth-washing entirely has already been alluded to (p. 64).

After the first teeth appear, the mouth should be washed and the teeth themselves rubbed with a moistened cloth morning and evening. When most of the temporary set are cut, a small tooth-brush of softened bristles is to be preferred to the cloth. Occasionally the use of a carbonate of lime tooth-powder on the brush, or even of powdered pumice-stone applied with a match-stick, is required if stains appear on the teeth. As early as possible older children should be taught to use the tooth-brush, and to draw floss-silk between the teeth after each meal. All decay, even of the first set, must be watched for, and the services of a dentist obtained at once, since not only are the carious teeth unsightly, but they cause toothache, occasion indigestion, and even interfere with the eruption of the second set in the proper position. It is especially to be remembered that the permanent anterior molars may appear and even decay, the mother meantime mistaking them for the teeth of the primary set. (See Dentition, p. 27.)

The *scalp* should be soaped daily for some months at the time of the general bath. After the age of 6 months, however, it is not desirable to use soap so frequently, lest the hair be made dry and brittle. In childhood soap must be used occasionally, and water daily, even with the longer hair of girls. The first brush employed should be of camel's hair. Later, when the hair grows coarser, a stiffer brush is required, in order to remove all scaliness. Combs should always be used with great care, and only for parting the hair. In infancy they are not needed. Hair should be trimmed frequently, and even in the case of girls be kept short until well into early childhood.

The *nails* of the fingers should be cut often even in young infants, and be kept clean with a soft nail-brush. The toe-nails should never have the corners rounded off, lest ingrowing follow.

In addition to the daily general bath, the region of the *anus* and *genitals* should be washed with water, without soap, after every movement of the bowels during infancy. If there is much irritation of the skin, starch-water may be substituted with advantage. Daily, too, the prepuce ought to be fully retracted and the glans cleansed carefully with soap and water. Adhesions between the prepuce and glans are very common. These are usually readily broken by "stripping," if this procedure is done early. (See Adherent Prepuce, Vol. II, p. 483.) The labia majora should be separated at the morning-bath and the vulva washed carefully, the direction of the washing being *toward* the anus in order to avoid as far as possible the forcing of feces into the vagina.

CLOTHING[1]

The great requisites for the clothing of infancy are softness, lightness, warmth, looseness, and simplicity. With the exception of the diapers, woollen or partly woollen stuff is the best for use next the skin, except in hot summer weather, or for infants whose skin is especially sensitive, when cotton, silk or linen is to be preferred. The weight of the garments must, of course, vary with the season of the year, but it is important even in winter not to have these of such a weight that perspiration is readily produced. Extra warmth is easily obtained by the use of sacks and the like, which can readily be slipped on or off as required. More children are dressed too warmly than the reverse. The underclothing should cover the whole body except the head and the hands in order to prevent sudden chilling after perspiration. There should be no pressure which can be avoided. All petticoats should be suspended from the shoulders, and should be simple in construction, fastening with but few buttons or with a narrow ribbon. The old-fashioned "pinning blanket" is to be condemned.

First Clothing for Infancy.—Considering the garments more in detail, those required for infancy are as follows:

1. An *abdominal band*, which is of flannel, and is wrapped about the abdomen next to the skin. It holds the dressing of the cord in place. After the falling of the cord the wearing of any band by healthy infants may be omitted entirely, but it is generally advised to replace the flannel band by a knitted circular one of wool or of wool and silk, which is pinned to the diaper and supported by shoulder straps.
2. A *diaper*, which should be of cotton or linen diaper-cloth or, still better, of cotton stockinet. A small diaper-square may be placed within the outside diaper in such a position that it will receive the urine and feces. This greatly lessens the thickness and consequent heating. A rubber or other impervious cover should never be employed. The diapers should be changed as soon as wet by urine, and should never be used again until after they have been washed. If this precaution is not observed chafing is likely to result. No soda should be employed in washing them.
3. High crocheted or knitted *socks* of silk or woollen yarn.
4. A loose *shirt*, long sleeved and extending below to the band, with the opening the whole length of the front and fastened by small flat buttons, or overlapping well in front and secured by tape. This should

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[1] Fuller details concerning the clothing, the layette, etc., may be found in the Author's "The Care of the Baby," published by W. B. Saunders Co., Phila.

be made of all wool, wool and cotton, or wool and silk, and be of thickness varying with the season. In the hottest summer weather it may need to be entirely of cotton.

5. A sleeveless *petticoat* of white flannel throughout, made in one piece from neck to hem, fastened at the back with one or two flat buttons, and extending not more than from 6 to 10 inches (15 to 25 cm.) below the feet. For summer the petticoat may consist of a flannel skirt attached to a loose cambric waist. A second petticoat of cambric is often worn over the first to prevent the flannel showing through, but this is unnecessary. In very warm weather the flannel petticoat may be discarded.

6. A *slip* or *dress* of nainsook or lawn, loose, with long sleeves, and opening at the back like the petticoat and of the same length, or only a trifle longer.

A once popular costume known by various proprietary names is similar to that described except that the knitted, close-fitting shirt is replaced by a long loose garment very similar to, and almost as long as, the petticoat described, but with long sleeves. This shirt, the petticoat and the slip may be fitted together, one within the other, and then all three slipped on at once, thus saving considerable turning of the baby back and forth while being dressed. This style is only suitable for long clothing, as the short clothing of later infancy allows too much air to enter under the loose inner shirt.

Besides the garments described the infant needs a *shawl* or a *shoulder blanket* of flannel, to be used as a protection when taken out of the room. Sometimes a thin, knitted worsted *sack* is useful if the room happens to be cooler than usual. This allows for movement of the arms. A flannelette *wrapper* is also often serviceable to put on before the child has its bath. For use out-of-doors there is required for winter a warm long *cloak*, warm *hood*, and a *veil*. In hot summer weather only a cambric or thin silk cap is required.

At night the child should be dressed in a fresh diaper, band and shirt, and then have put on a long, roomy *nightgown* of flannel, canton flannel, or stockinet, closing with a drawing string at the bottom. In summer it may be of muslin, and need not be fastened below. No socks are required at night.

Dressing the Cord.—Before the clothes are put on for the first time the stump of the umbilical cord must be dressed. It is dried as far as possible, powdered with bismuth, boric acid, or a mixture of salicylic acid and starch and wrapped thickly in an antiseptic absorbent cotton. It is then laid against the abdomen, a thin compress put over the navel, and the binder applied. The wrappings of the cord should not be changed until the cord falls, provided there is no evidence of putrefaction. The greatest care should be taken to prevent the soiling or wetting of the dressing. The ulcer remaining after the separation of the cord should be dressed with powdered boric acid applied on a pad of absorbent cotton.

Method of Dressing.—After the morning bathing and drying, the nurse, still holding the infant lying on its back in her lap, puts the band about it, or, if this is a knitted one, slips it on over the feet. The diaper is next placed under the buttocks and the ends brought around in front and fastened with a large safety pin to each other and to the tab of the band. Neither band nor diaper must ever be so tight that the hand cannot readily be slipped between it and the skin. The socks are then drawn on. The petticoat is next adjusted inside of the dress, and the two slipped on together over the feet. The baby is now laid upon its abdomen and these garments buttoned. After the child is old enough to sit alone they may conveniently be slipped on over the head.

Short Clothes.—At about the age of 6 months, the choice of the time depending upon the season of the year, the infant is put into *short clothes.* Its costume then consists of a band, diaper, shirt, petticoats and slip similar to those described, except that the skirts reach only nearly to the ankle, and that it is very customary now to make the flannel petticoat always with a muslin waist and to have a second white petticoat over this. In addition, the child requires *stockings* and *shoes.* The stockings should be white, in order to avoid any action of irritating dyes, and made of silk, woollen or partly woollen goods, or, in hot weather, of cotton. They should always be long enough to reach to the diaper, to which they may be pinned, and should be loose and with broad toes, in order to prevent undue constriction of the feet.

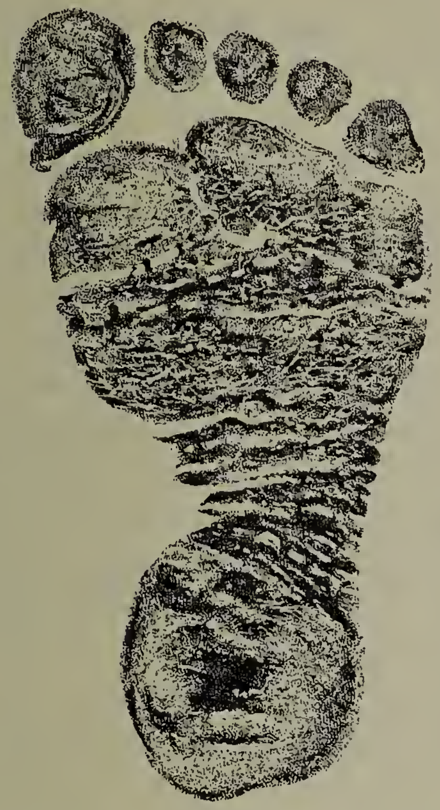

Fig. 13.—Imprint of Foot. From life, three-quarters natural size.

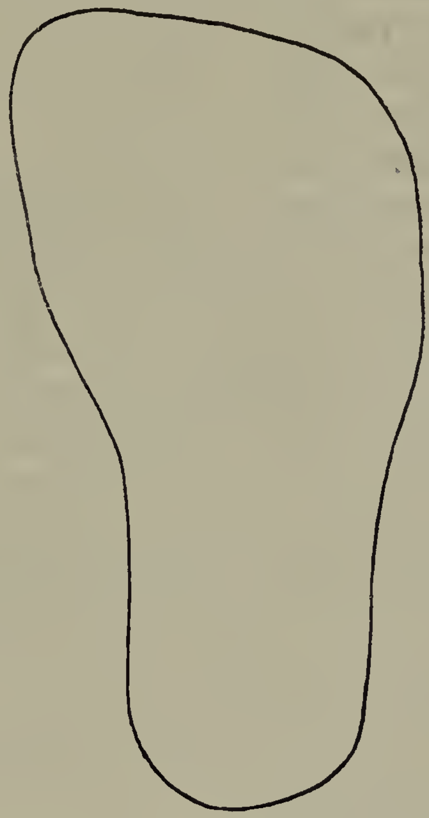

Fig. 14.—Outline of Sole of Shoe to Cover Fig. 13.

As the child becomes more active there is often great difficulty in keeping the diaper from falling off. Careful pinning to the band will obviate this, or the infant may wear *diaper-suspenders*, or a small light waist to which both diaper and stockings can be attached, the latter by means of elastics or tapes.

The first foot-covering may be moccasins of kid, chamois-leather or felt. When the child begins to stand and creep true shoes are required. These may be of soft kid, with soles of kid or thin leather. From the beginning they should be rights and lefts, conformed to the natural shape of the foot and with broad toes and without heels (Figs. 13 and 14).

The clothes for the night are the same as in earlier infancy. For use out-of-doors the coat must be shorter after the child has learned to walk, and warm knitted or Jersey *leggings* are useful in winter.

In addition to the articles mentioned the infant now needs quilted *bibs* to catch the overflowing saliva. Later, a serviceable article is a *creeping apron.* This should be large and roomy, opening at the back but closed at the bottom, except for the apertures for the legs which fasten just below the knees. *Rompers* may be used in place of the apron. Creeping aprons which go over the clothes and fasten under them around the waist are to be avoided, as they allow too free access of air to the legs.

Clothing of Childhood.—At 2 years of age, or less, when the diaper can be dispensed with, the clothes of *childhood* replace those of infancy. The binder is abandoned, if it has been worn hitherto, and the clothing consists of the following articles:

1. An *undershirt*, long-sleeved and high-necked, of material as before and of thickness varying with the time of year, but never too heavy.
2. *Drawers*, close-fitting merino for winter, looser and of muslin for hot summer weather.
3. *Stockings*, long at all seasons. Nothing is gained and much damage may be done by dressing little children in short stockings and leaving the legs bare.
4. A white *muslin skirt* without a waist.
5. A loose, high-necked, sleeveless *waist* provided with buttons, to which the stocking-supporters, drawers, and skirt can be attached. If desired, the white skirt can be made attached to a waist of its own, but this offers no advantage.
6. A *flannel skirt* with muslin waist, to be worn in winter.
7. A *dress*, which commonly indicates by its style the sex of the wearer.
8. *Shoes*, which should have the posterior portion of the sole made slightly thicker—*i.e.*, with a "spring"—when the child is 3 or 4 years old, but which should have no true heel until the age of 10 or 11 years.

The clothes for the night consist of a *shirt* and *night-drawers*, the latter having closed feet if the child sleeps restlessly and displaces the bed-clothes. The material may be cotton-flannel or stockinet for winter, and muslin for summer. Only in later childhood does the girl begin to use a night-gown and the boy a night-shirt; or pajamas may be worn by either sex. When out of doors the hood should be worn in winter until the age of 2 years at least.

At the age of about 2 years or earlier some distinction of sex is made in the style of the clothing, and by 3 or 4 years the boy assumes the ordinary boy's clothes.

SLEEP

Hours for Sleeping.—A healthy infant in the first few weeks of life sleeps nearly all the time; in all from 19 to 21 hours, rousing only when being nursed, washed and dressed, or when it is hungry or uncomfortable. As it grows it requires less and less sleep, and at the age of 2 months will often lie awake quietly for an hour or so at a time, and show a tendency to some regularity in the hours of sleeping. By the age of 6 months, 16 to 18 hours are required daily; by 1 year, 14 to 16 hours; at 2 to 3 years, 12 or 13 hours; at 4 to 5 years, 11 or 12 hours; at 12 to 13 years, 9 or 10 hours. Sleep during infancy is always easily disturbed by light, noise, and handling. In children it is deeper. From the beginning the child should be taught to sleep at regular times, and to be put to sleep in proper ways. Walking the floor, rocking, singing to sleep, and the like, are entirely unnecessary. They establish the child in a bad habit, and make a slave of the mother. If the infant is certainly well, it should be put in its bed at the time for sleep and left alone in the room. If it wakes in the night it must not be taken from its bed unless it is time to nurse it, or the diaper requires changing. Knowing no other method it will soon content itself with this. Allowing a child to go to sleep while nursing at the breast or bottle and before it has finished should be prevented as far as possible. Before the age of 3 or 4 months the baby is made ready for bed at 5.30 to 6 P.M., and should rouse but once or twice during the night. During the day it may sleep at first all it will, but must be wakened for feeding when this is due. This is a matter of importance, as otherwise there can be no regularity in the feeding hours. After this age it may be put to bed at from 6 to 7 P.M., be wakened at 9 or 10 for feeding, as long as this is required, and be trained, as soon as possible, to sleep without further rousing until 6 or 7 A.M. It will now be awake for longer periods in the day time, and by the age of 6 months or earlier will content itself with a nap in the morning of from 1½ to 2 hours or more, and perhaps a shorter one in the afternoon. The nap in the day should be of regular length, and the child not allowed to sleep over the time for feeding. At the age of from 1 to 2 years the afternoon nap may be omitted, unless the child seems to require it; the morning nap lasting usually 1½ to 2 hours. After 2 years the morning nap may be shortened to 1 or 1½ hours. The child should continue to take it up to the age of 4 years, if possible. Children of 4 years should go to bed at 8 P.M. or earlier, and the time be gradually changed to not later than 9 P.M. by the age of 10 to 12 years.

When the infant begins to take a regular morning nap, it is best that it should be undressed for it, and be put to bed as though it were night. Often the morning dressing may conveniently be delayed until after the nap, and the morning bath then given. The hour for the morning nap will depend partly on the disposition of the child, and partly on the season of the year. A portion of the day should be selected which will not interfere with the daily outing. A certain degree of latitude is therefore to be allowed in the fixing of the time and in the necessary duration of sleep. Sometimes it is better to transfer it to the early afternoon. The two great desiderata sought for are, first, regularity, and, second, the obtaining of the long quiet sleep at night.

When asleep, the child may assume any position most comfortable to it. In the case of young infants the necessity of the position being changed from time to time must not be forgotten. (See Disorders of Sleep, Vol. II, p. 557.)

Place to Sleep.—The infant should not sleep in the bed with its mother. There is danger of her overlying it, to say nothing of the constant temptation to nurse the child too often. It is liable, too, to receive too little fresh air, as a result of getting its head under the bedclothes. The first bed generally used is the *bassinette*. This is made of enamelled wicker or wood, with high sides, lined, best unprovided with curtains, and with or without a hood at one end. It should be high enough above the floor to escape the draughts. A *cradle* may be used instead, but should be one not capable of being rocked. The bassinette is preferable to the crib for the early months of life, as it gives the child more support and keeps it warmer. At the age of 8 or 9 months and up to that of 5 years a *crib* is used. This should have sides which let down on hinges or slides and are high enough to prevent the child falling out, and be provided with a woven-wire mattress. A very serviceable device, especially for sleeping out-of-doors in summer, is the screened nursery crib (Fig. 15).

The *bedding* for the bassinet and crib is the same. There should be, namely, a thin, hair-mattress, a rubber cloth, in the case of infants, to go over this, and a doubled sheet. Sometimes a quilted bed-cover may be put under the sheet to increase the warmth in winter time. The pillow should be small; a soft, thin, feather pillow with a linen pillow-slip. A curled-hair pillow may be substituted in summer if desired.

The *coverings* of the child when in bed should consist of a muslin sheet, as many light blankets as needed, and a light spread. A down quilt is an advantage in winter. Owing to the restless sleep of so many children, some form of bed-clothes-fastener is desirable. A great many children are covered far too warmly at night, with the result that sleep is rendered restless, free perspiration occurs and cold is very easily taken.

It is theoretically better that the infant from the beginning sleep in a separate room from its mother, under the care of a competent nurse. Often this cannot be arranged for many reasons. After the age of a year, it certainly should have, if possible, a separate room at night. Older children are preferably placed in individual beds.

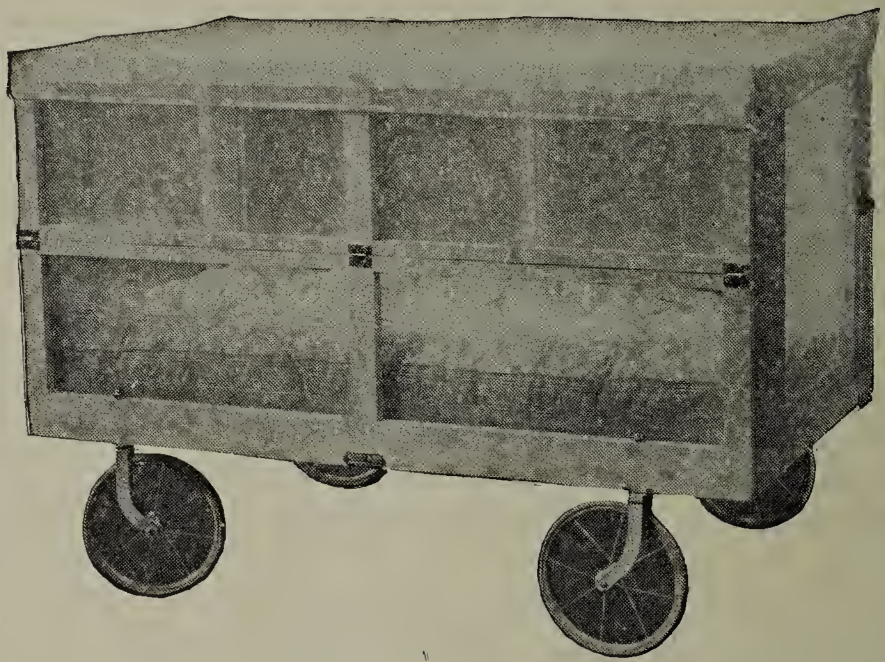

Fig. 15.—Screened Nursery-crib. *(Courtesy of Best and Co.)*

AIRING, EXERCISE, AMUSEMENTS, AND TRAINING

Airing.—With regard to airing, no absolute rule can be formulated. At 2 weeks of age the nurse may take the infant into another room of a somewhat cooler temperature, proper protection being given by dressing it in its out-door clothing. Before this age it should not be exposed to a temperature of less than 70°F. (21.11°C.). By the age of 1 month it may be taken into the open air for 10 or 20 minutes, and longer on subsequent visits, until finally it is out of doors 2 or 3 hours or more daily. This applies to the spring and autumn, if the temperature is not lower than 60°F. (15.55°C.) but in warm summer weather it may go out even at an earlier age, while in midwinter it cannot do so until it is 3 or 4 months old or possibly even later. In place of this it may have its daily airing in a room, the windows of which have been open for a short time, but closed just before the child enters it. Later, when accustomed gradually to the outer air, the windows may be left open during its presence. When possible, autumn babies should be gotten out of doors before the winter sets in. If this cannot be managed, the use of the room with open windows may often be advantageously continued until spring. The important matter is that the infant shall be accustomed *gradually* to the cool air, and that as soon as possible it shall have an abundance of fresh out-door air.

It is a mistake to take an infant into the open air every day no matter what the character of the weather. Very damp, windy, or intensely cold days are to be avoided and the outing in the room with open windows employed instead. A temperature of 20°F. (6.66°C.) is an indication that the infant had better stay in the house. Always the condition of the hands and feet and the color of the face are to be watched, and the airing stopped if the infant shows the least chilliness.

Although the bed is usually a better place, there is no reason why a child may not sleep out of doors in the day-time in warm weather, or even in cold if it is well protected and its condition carefully watched. Some children invariably fall asleep when taken out. In the night-time, however, the child should sleep in a room where it can be under better supervision. This does not mean that the room should not have fresh air in abundance.

The first going out should be in the nurse's arms, as this gives greater protection and warmth. After the age of 3 or 4 months, depending upon the season, a perambulator should be used. This should have a sunshade or hood which is lined with some dark color, such as green or brown, to avoid injury to the eyes by the reflected or transmitted light. The infant should recline in the coach, but after the age of 7 or 8 months may sit supported a portion of the time, care being taken that the back does not become fatigued.

Exercise.—After the first 2 weeks of life the infant begins to move its arms and legs freely. It can be assisted in this by having the clothing loose and not too long. At 2 weeks it should be exercised by being systematically carried about in the arms several times a day, lying on a pillow in order to give support to the spine. At the age of 1 month the pillow may be discarded. At 3 or 4 months the infant should be carried about, seated upright in the nurse's arm with her hand supporting its back. It should not be supported invariably on the right or the left arm or lateral curvature of the spine is liable to develop. The carrying about by the nurse is a matter of vital importance. Infants who lie too much in their cribs do not thrive as they should. At about 6 months of age it may be propped with pillows in a sitting position, but only for a little while at a time. From the age of 3 or 4 months on the child may be placed at times upon a thick blanket or mattress, spread upon the floor or elsewhere, securely out of draughts. Here it can make all movements of which it is capable, and has also a good chance later to creep as soon as it is able. The great likelihood of the existence of draughts upon the floor, especially in cold weather, must never be forgotten. Children who have learned to creep or walk will take all the exercise they need. The baby should take its own time to learn to walk, and no appliances to aid it in this are advisable. The legs should be carefully watched for evidence of bowing. (For the age for creeping and walking, see p. 61.) The perambulator or some form of baby-coach must still be used for most of the airing until the age of 2½ or even 3 years, the child being allowed to walk when it desires, but for not too long at a time, lest fatigue ensue or the arch of the foot be overstrained.

Various different forms of exercise will be advisable as the child grows older, in order to bring into play different muscles of the body and to preserve symmetry in development. Little restraint need now be imposed unless the child is delicate, or is of the nervous and excitable nature which disposes it to exercise to the point of exhaustion. All out-door sports should be encouraged for both sexes. The tricycle and similar apparatus can be used early, and later the bicycle. Swimming, skating, riding, tennis, and the like are all excellent. Jumping rope is harmless unless much overdone. In winter, especially in cities, it is often difficult to obtain sufficient exercise. Dancing and work in a gymnasium now become the best substitutes.

Training and Amusements.—Teaching the control of the bowels and bladder should be commenced early, certainly by the age of 3 months. If the infant is systematically supported on a receptacle at the time when the evacuation of the bowels is found most likely to occur, generally after a meal, much may be accomplished. The control of the bladder is more difficult to acquire, but even before the end of the 1st year some infants can be taught it, at least during the waking hours. Patience and perseverance are required, and punishment at any age is out of the question.

Amusements begin early, and those may gradually be added which educate the mind to a certain extent. Yet the training of the mind must always hold a secondary place, lest overstimulation of it result. In fact all amusements which cause much excitement are to be avoided, and this is especially true if they have been permitted just before bedtime. Insomnia is a natural result. By the age of 5 or 6 months a rattle or a rubber doll will be enjoyed. Later more toys are needed for the house, and others to be used out of doors. Mental and moral training can soon be commenced by using judgment in the selection of the amusements. Thus the constant providing of new toys teaches a child discontent, lack of valuation and lack of care of those which it possesses, as well as lack of neatness. Pernicious, too, is the constant effort of the mother to amuse the child, since it should be taught to amuse itself. Many toys may be chosen which instruct, such as picture-books, Noah's arks, picture-blocks, and, later, lettered blocks.

The inculcating of obedience, unselfishness, absence of self-consciousness, fearlessness, and general kindliness to all created things can hardly be commenced too early. There is no hurry about teaching a child to talk, but the use of "baby-talk" in addressing it is always to be avoided.

Girls and boys should play together and at the same games as long as disposed. They have a natural disposition to do this until the age of 9 or 10 years is reached unless they have been taught to do differently. Yet a careful, unobserved supervision should early be kept over the play, since sexual innocence in early years is by no means so common as is often assumed.

School Life.—Most important is it that the mental powers shall not be forced. Precocity is not desirable. The kindergarten is excellent for young children, and much instruction is gained in this way. Yet even this is sometimes too stimulating. There is nothing gained in having a child learn to read before the age of 6 years. Even at this period the hours spent in school are generally far too long. From 3 to 4 hours daily from the age of 6 or 7 up to that of 10 years are quite enough. Ocular defects, lateral curvature of the spine, loss of appetite, poor general health and nervous irritability are common results of improper school life.

As to the fittings of the school-room, the chairs employed should be low enough to allow the feet to rest comfortably on the floor, and should be so constructed that they support the *lower* part of the back. The desks must not be high, or the child's eyes are brought too close to its work, and the spine is liable to be distorted when he writes. The light must be so disposed that there is no glare in front, and that trying cross-lights are done away with. There should be at least 300 cubic feet (8.49 cubic metres) of air space for each individual, and, in addition, the air should be constantly and completely changed by ventilation several times an hour.

NURSERY

Day=Nursery.—Inasmuch as the baby spends so much of its time in the nursery, the *position* of this in the house is of the greatest importance. It should be selected with reference to winter rather than summer, since the child will be out of doors so much of the time in the latter season. It ought to be, if possible, the brightest, airiest room in the house. The preferable exposure is south, or, if this is not obtainable, east rather than west on account of the morning sun. A corner room with south and west windows is ideal. The windows should not extend to the floor, since the children readily take cold as a result of getting too close to them. Other things being equal, the nursery should be on the third floor, if this is not directly under the roof, as it is more out of the way here. It should have at least 500 cubic feet (14.16 cubic metres), and preferably 1000 cubic feet (28.32 cubic metres) of *air space* for every person occupying it, and in addition should be properly ventilated by a constant and abundant supply of fresh air. Window-ventilation of some form accomplishes this fairly well yet with some danger of draughts. Probably the best of all ventilation in houses not especially built to secure it in other ways, is obtained by an open fireplace with a fire burning. Sometimes it is safer to ventilate from an adjoining room. In addition to the constant ventilation the room should be aired thoroughly with wide-open windows once or twice a day at times when it is unoccupied.

Draughts upon the floor nearly always exist in cold weather even when the windows are closed, the air being chilled by the cold panes. Double sashes will prevent this to a great degree, but in very cold weather it is better to keep the child away from the windows and even off of the floor.

In the line of *heating* probably nothing equals a hot-air furnace, especially if the air comes from over hot-water pipes, as it is easily controlled and supplies fresh warmed air from without. A fireplace, or a properly constructed coal stove, is effective in the same way, but gives an uneven heat and is less controllable. Gas-stoves or oil-stoves should never be used unless provided with smoke-pipes connected with the chimney. The only exception is for the rapid heating of the room before the bath, when this can be accomplished in no other way. Steam and hot water radiators, though serviceable heating apparatus, afford no ventilation whatever.

All hot registers, fireplaces and the like, and all lights, should be so guarded that a child cannot possibly burn itself. A couple of thermometers should be placed in different parts of the nursery, one of them near the floor, since it is in here that so much of the time is passed. The temperature of the nursery should be as uniform as possible at from 66° to 70°F. (18.9° to 21.1°C.).

The *furnishings* of the nursery may be attractive, yet simple and arranged with a view to cleanliness. The floor should be well-made, the cracks stopped, and the whole painted or varnished, but not with a slippery finish. Carpeting in the form of one or several rugs is required. These should not be tacked down. The walls and ceiling are best painted.

If paper is used one must be sure it contains no poisonous coloring matter. Windows should have no heavy curtains, and should be fitted with bars to avoid danger of falling out. The doorway should have a swinging or sliding gate to keep the child from the stairway. The furniture of the nursery is better if not upholstered. Chairs with projecting rockers and furniture with sharp corners are to be avoided. Among the articles of furniture is the "nursery-chair" on which the infant is placed when it is to empty its bladder or bowels, and only then. Portable screens are very useful. A nursery refrigerator is a great convenience.

The nursery must be kept scrupulously clean and neat. No wet diapers are to be kept hanging about, and no empty nursing bottles or empty dishes left in it. A stationary washstand may be in the room, provided that one is sure that the trapping is perfect, and that there is no danger of the entrance of sewer-gas.

Night=Nursery.—Although a room set apart especially for the night is not an essential, yet it is a great convenience when there are several small children in the family. The requirements are the same whether this separate room or the day-nursery is used, and the infant should sleep here whether at night or during its daily naps unless the sleeping in the daytime is done out of doors. The heating and ventilation are provided as in the day-nursery, and the room should be thoroughly aired and then warmed again after each occasion on which it is used. There must always be some arrangement for heating food at night, such as an alcohol-stove or small gas or electric heater. The temperature of the room should be about 65° or 70°F. (18.3° or 21.1°C.) during the early weeks and later from 50° to 65°F. (10° to 18.3°C.). A window should be partly open as a rule, but it is not wise to have it widely open during very cold weather. Light should not be allowed to burn all night. If it is necessary to have this on occasions, a small wax night-light or a candle is excellent. Nothing equals electric light, as it consumes none of the oxygen. Small shaded electric lamps can be purchased. If oil-lamps are used for lighting before the child is put to bed they must be out of harm's way where they cannot be upset. The beds must be carefully placed out of the way of draughts. The use of folding screens is an aid to this end. If the child is bathed and dressed in the night-nursery the temperature of the room should be elevated to 70°F. (21.1°C.) before the toilet begins.

Sick=Room.—In the case of ordinary slight ailments no special room is required, one of the nurseries being generally employed; but in more serious illnesses, or where special quiet is demanded, the room selected should be away from sources of disturbance. Numerous bottles of medicine should not be allowed to stand about. Not only is this unsightly, but there is danger of giving the wrong drug. Ventilation is very important, yet sometimes difficult to obtain satisfactorily on account of the danger of draughts in certain diseases. Sometimes it is necessary to ventilate entirely from an adjoining room where the air is kept fresh and warmed.

The special requirements of the sick-room for infectious diseases will be described in considering that subject. (See Infectious Diseases, p. 345.)

NURSES

The Nurse=maid.—Two of the greatest requisites in a nurse-maid are intelligence and docility. Consequently the nurse-maid should be preferably in middle life, but better young than old. The old nursemaids are commonly so opinionated that their methods, generally bad ones, cannot be changed. There are, of course, exceptions to this. The maid should be neat, strong, healthy and of loving and patient disposition. No matter how faithful and efficient she may seem, no mother, even the inexperienced, dare give up her own constant supervision.

The Trained Nurse.—The nurse trained specially for the care of infants and children during illness will be of the greatest service to the patient, the family, and the physician, if with a proper knowledge of nursing she combine a special recognition of the responsibilities and relationships which the nursing of asick child creates. If she is not so qualified she may do more harm than good. It is the physician's duty to select carefully the nurse adapted for this service and to supervise the nursing constantly. A few of the qualifications required may be passed in review:

1. The nurse should be accustomed to and have a fondness for little children. Nothing is more exacting and trying than the nursing of sick children, and, unless there exists a special native fitness and liking for it, a nurse should not undertake it.

2. She should be quick to recognize and prompt to report important symptoms. Only careful training and inborn acuteness of observation, combined with good judgment, can give a nurse this invaluable quality.

3. She should be quiet, gentle, firm, resourceful, and comforting. The influence of such a nurse upon the mental state of a nervous infant and nervous mother is often most remarkable.

4. The nurse must carry out the physician's directions implicitly and without criticism before the family; yet know when emergency justifies and compels a failure to do this. Unless she has this quality her actions are too much those of an automaton.

5. She must not be imposed upon by the family. No nurse can do good work who is exhausted by the nursing of a fretful, ill infant, in which she is given no opportunity for systematic rest. Families are prone to forget this, and for the sake of the patient the physician should see that matters are properly arranged.

6. On the other hand, the nurse must be willing and helpful, and avoid making her presence a burden. The installing of a nurse necessarily adds to the responsibilities, expenses, and cares of the manager of the household, and mothers often dread it with reason. If the nurse seems to need some one specially to wait upon her, as is often the case; stands constantly upon her rights and privileges without regard to the effect upon the patient; is not quick to offer to prepare and serve special food for the child; is unwilling to take the ordinary care of the room, or to take the place of the mother in giving a convalescent child its outdoor airing in its coach; is displeased if she is not taken fully into the social life of the family, as at meals or in the evenings, she fails to recognize the dignity and duties of her profession, and is not a suitable caretaker for a sick child. Inasmuch as these faults are all too common, the physician must acquaint himself with conditions and correct those which are wrong.

CHAPTER III

BREAST-FEEDING

Advantages.—The general superiority of breast-feeding can hardly be questioned. Statistics show that the mortality of breast-fed children in the 1st year is much less than that of those fed on the bottle. (See Mortality, p. 239.) In fact the average probability of death in the 1st year in artificially fed infants as compared with the breast-fed may be placed at 5 : 1. The figures of Camerer[1] and others demonstrate, too, that infants fed artificially do not as a class gain as rapidly in the 1st year as those breast-fed. Further, the interesting studies of Rose[2] upon 164,000 individuals showed that the deleterious influence of bottle-feeding could often be detected even in adult life. Although artificial feeding carefully carried on will undoubtedly give much better results than statistics indicate, yet the superiority of breast-feeding, as a rule, remains unquestionable. This is a natural and unavoidable result of the fact that other mammalian milks are in a sense foreign substances and cannot be made otherwise. It is probable, too, that breast-fed infants are less susceptible to general infection and to many specific ones (McClanahan).[3] Certainly, then, every effort should be put forth to have the infant breast-fed for the first 3 months of life at least, by which time a fair start has been obtained, and success with artificial feeding is more likely to follow.

Ability of Mothers to Nurse.—The long-continued propaganda for artificial feeding, especially with proprietary foods, had in the course of years the natural sequence that breast-feeding in many localities became comparatively uncommon, or at least continued for a very short time. I[4] have reviewed the subject rather extensively elsewhere, and a few illustrative statistics may be quoted from those publications. Conditions vary greatly with the locality. In Japan breast-feeding is the rule. In Greenland artificial feeding is scarcely known, and among the Esquimo of Alaska there is no cow's milk available, and infants are often nursed for from 2 to 3 years. On the other hand, Nordheim[5] found that only 3.6 per cent. of the women studied in Munich nursed for over 3 months, and only 0.8 per cent. for over 6 months. Neumann[6] stated that 55.2 per cent. of the children seen by him in Berlin were nursed in 1885 and only 31.4 per cent. in 1890; and Koplik[7] in New York found only 10 per cent. in private practice fed on the breast alone.

Much of this apparent inability is probably unwillingness and a lack of teaching by the profession, and the indications are that with the increasing urging by physicians upon mothers regarding the importance of breast-feeding the frequency is now again increasing. An investigation by Mitchell[1] upon 2819 mothers in Philadelphia showed that the average duration of lactation was 6 months. Nigris[2] in Graz found a physical disability in only 10 per cent. of the women in the obstetrical clinic. Mme. Dluski[3] observed that 99 per cent. of the women seen in the obstetrical clinic of Pinard in Paris could nurse at least for a time; and Blacker[4] in London found only 2.5 per cent. of the mothers with a physical inability to nurse. Among the better classes in the United States the statistics of Manning[5] for Seattle in 1000 cases in private practice gave 64 per cent. of the mothers still nursing at 3 months or over; 41 per cent. at 6 months or more, and 26.8 per cent. at 9 months or over; while in Dietrich's[6] 1000 private cases in Los Angeles 37 per cent. of the mothers stopped nursing by 3 months or less, and 40 per cent. were still nursing at 8 months or more. That the general ability can increase through the training of the people is indicated by the experience of Jaschke[7] who observed that while in 1904, 64.01 per cent. of the infants in Heidelberg were breast fed, in 1907, 86.14 per cent. were so fed, and in some years 97.22 per cent. The frequency of maternal nursing is, however, far from what could be desired; and it is undoubtedly true that the nervously organized mother of the upper classes often is unable to nurse her infant in spite of her strong desire to do so. Yet in numerous cases it is the ill-considered advice of the nurse, or still oftener of the physician, which is the cause of early weaning or of failure to nurse at all. Many a woman whose breast-milk has ceased entirely for a day or two, or in whom it has been slowly diminishing, will still be able to nurse her baby if given proper treatment and encouragement. So, too, the failure of the secretion to appear in the first few days after parturition is no reason for abandoning efforts at maternal nursing, inasmuch as it is not at all infrequent to have the full secretion delayed for a number of days. A scanty supply of milk is no excuse for weaning, since even the small amount, helped out by artificial feeding, is better for the baby than no breast-milk at all. Again, the apparent fact that the milk disagrees with the infant should be regarded with suspicion, especially in the early weeks after birth, inasmuch as it often happens, when the mother gets out of bed and resumes her ordinary method of life, that both the quantity and the quality of the secretion will change in a satisfactory manner. What can be done by means of careful and persistent instruction of mothers by their physicians has been conclusively demonstrated by Sedgwick[8] in Minneapolis, from 66 per cent. to 71 per cent. of the infants being still at the breast at from 8 to 9 months after birth as a result of this influence.

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[1] Jahrb. f. Kinderheilk., 1893, XXXVI, 249.

[2] Deutsch. Monatsschr. f. Zahnheilk, 1905, XXIII, 3. Ref. Ziegenspeck, Verhandl. der deutsch. Gesellsch. f. Gynäk., 1907, XII, 829.

[3] Arch. of Pediat., 1918, XXXV, 652.

[4] New York Med. Jour., 1909, Dec. 4; Journ. Amer. Med. Assoc., 1912, LIX, 1874.

[5] Arch. f. Kinderheilk., 1901, XXXI, 89.

[6] Deutsch. med. Wochenschr., 1902, XXVIII, 795.

[7] Jour. Amer. Med. Assoc., 1912, LVIII, 75.

Probably one of the chief causes for the early disappearance of the secretion is the failure to obtain a satisfactory emptying of the breast. This is especially liable to occur when the infant is weakly and grows easily fatigued by sucking. Another cause often assigned for failure to nurse is painful fissuring of the nipples. This is a real problem, but generally can be solved with care and patience. Indeed the causes which are assigned for the failure to nurse are often of the most trivial nature. This has been interestingly studied especially by Keller.[1] In addition in the poorest classes is the very real difficulty that nursing women are often obliged to go to work. This frequently necessitates early weaning. It is a matter which can best be dealt with from a civic point of view, aid being given to mothers in these circumstances.

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[1] Jour. Amer. Med. Assoc., 1916, LXVI, 1690.
[2] Wien. klin. Wochenschr., 1905, XVIII, 459.
[3] Thèse de Paris, 1894. Ref. Marfan, Rev. mens. des mal. de l'enf., 1902, XX, 11.
[4] Manchester Med. Chron., 1900, Series 3, II, 323.
[5] Arch. of Pediat., 1920, XXXVII, 214.
[6] Jour. Amer. Med. Assoc., 1922, LXXIX, 268.
[7] Med. Klinik, 1908, IV, 257.
[8] Amer. Jour. Dis. Child., 1921, XXI, 455.

Before abandoning breast-feeding on account of a refusal of a weakly infant to nurse, the effort should be made to empty the breast artificially and then to feed the milk. This is nearly always necessary in premature infants. Pumping can be employed, but is frequently very unsatisfactory with the ordinary apparatus. Abt's[2] electric breast pump we have found very satisfactory. Better than the use of the ordinary breast pump is the expression of the milk. To accomplish this the breast should be taken between the thumb above and the fingers beneath. The thumb is then pressed downward toward the fingers, and at the same time carried forward to the base of the nipple, and finally a forward pull given, which makes the milk flow from the nipple.[3]

Preparation of the Prospective Mother.—In view of the importance of breast-feeding the preparation of the prospective mother for this is of great importance. The general hygiene has already been briefly referred to (p. 64). The diet should be rather more abundant than usual, the bowels should be kept regular, gentle exercise in the fresh air should be taken, and simple amusements enjoyed, without much strain of the emotions. Undue pressure of clothing upon any part of the body, and especially upon the breasts, should be avoided during pregnancy. If the nipples are retracted nipple-protectors may be worn. Useful, too, to develop the nipples is gentle traction by the fingers, or, still better in some cases, by the application of the breast-pump several times daily. Neither procedure, however, should be practised until the last 4 weeks of pregnancy. At the same time the nipples must be hardened by applying twice a day equal parts of glycerine, tannic acid and water, or a saturated solution of boric acid in equal parts of alcohol and water.

Hygiene of the Nursing Mother.—The care of the nipples during the period of lactation is even more important than before the birth of the child. After nursing they should at once be washed and dried gently and then smeared with a little sweet oil. If they become excoriated or fissured, the application of a small amount of a bismuth-ointment (equal parts of bismuth and ol. ricini) or of compound tincture of benzoin is often efficient. Sometimes an initial soreness will pass away entirely in a few weeks. In other cases, where the pain of nursing is intense, the use of an artificial nipple must be tried. Great care must be observed to keep this scrupulously clean. It has been suggested (Moll)[4] that fissures are largely due to faulty position of the nipple in the infant's mouth. The nipple should be vertical in the mouth from the overhanging breast.

During the first few days after childbirth the nursing mother should eat rather cautiously, taking small quantities of easily assimilated food frequently, lest overfeeding produce indigestion. There is no need, however, for any great restriction of diet. Toward the end of the 1st week she may have meat and later may, as a rule, eat plentifully of any nutritious, digestible food. In addition to this milk, cocoa, or some other light liquid nourishment should be taken at bed-time and possibly between meals. The ingestion of excessive amounts of milk, such as 2 or 3 quarts a day, should be avoided, as this will usually cause indigestion in the nursing mother, or, if tolerated, will result in gain in weight without influencing the breast-milk. Green vegetables should be given every day because of their vitamin-content and their value as roughage. The cereals used should be of the cooked variety and it is often of advantage to give them twice a day. Weak tea and coffee are permissible. With regard to stimulants, the nursing mother, if in good health, needs nothing of the sort. Delicate mothers are sometimes much benefited by one of the malt liquors, and the quality and strength of the milk increased thereby. Alcohol, however, is to be prescribed with caution lest habits of intemperance be established.

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[1] Wien. klin. Wochenschr., 1909, XXII, 635.

[2] Jour. Amer. Med. Assoc., 1923, LXXXI, 391.

[3] U. S. Dep't. of Labor, Children's Bureau. Care of Children Series No. 5, Publ. 83.

[4] Med. Klinik, 1922, XVIII, 403.

A proper amount of exercise in the open air, taken with regularity, favors greatly the production of a healthy, nourishing milk. Carriage-riding is beneficial, but does not take the place of walking begun as soon after parturition as the state of the patient permits. Late hours should be avoided and plenty of rest and sleep obtained. All causes of nervous excitement and of worry should be carefully shunned.

Rules for Nursing.—The infant should be put to the breast after it has been washed and dressed, and as soon as the mother is sufficiently rested to allow it. It lies upon its left side to nurse from the right breast, and *vice versa,* its head being supported by the arm of the mother, who rests upon her side or is propped up slightly in bed. When the mother is convalescent and able to sit up, she should lean slightly forward while nursing, partly supporting and steadying the breast with the fingers of one hand, in order to keep its weight from pressing against the infant's nose and interfering with its breathing. If the milk flows too freely it may be restrained by compressing the base of the nipple slightly with the finger and thumb. One breast should be sufficient for one nursing. The giving of both breasts at a nursing is inadvisable, unless a scanty secretion makes this necessary. A good secretion is stimulated by a thorough emptying of the breast, and the giving of both of them is liable to leave neither empty. An infant should nurse for not more than 15 or 20 minutes, and should not be allowed to go to sleep until it has finished its meal. Many infants are uniformly contented with a shorter period, because they have nursed with vigorous rapidity; others stop nursing in a few minutes because of feebleness or because the breast is empty; others from habit or from an insufficient milk-supply wish to nurse longer. The weighing of the infant before and after nursing will determine the actual condition present.

Intervals.—During the first 2 days but little secretion of any kind is in the breast, the needs of the infant are slight, and it is not necessary to nurse the child oftener than from 4 to 6 times in the 24 hours. The employment of some substitute such as a solution of sugar or a starchy decoction is generally entirely unnecessary, but water should be given freely. If there is no milk by the 3d day feeding with a very weak substitute-milk-mixture must be commenced while efforts at nursing are still continued. Only in exceptional cases, as in prematurity or where the infant seems particularly hungry, is earlier feeding required. As has been pointed out elsewhere (p. 6) no real benefit is gained by feeding an infant even with breast-milk of another woman from the 1st day of birth. The infant should be kept very quiet after nursing. A little turning about, jogging on the knee, or other motion may very readily cause vomiting. From the beginning the greatest regularity should be observed in the times for nursing the infant, keeping in mind, too, the fact that at night both mother and child should have as much undisturbed sleep as possible. The duration of the intervals between feedings has been a subject of much discussion. The fact that the healthy, breast-fed infant empties its stomach often within 1 hour after nursing, and practically always in not more than 2 or 2½ hours (see p. 34) is an indication that an interval as long as 4 hours, as recommended by many, may be too great, and that the shorter intervals, which have so long been in vogue, should not be too readily abandoned. The fact is that there can be no absolute rule applying to all infants, although there should be regular intervals for each individual. In the case of a hearty infant taking a large amount of food at each nursing the intervals will necessarily be longer. A more delicate infant with less appetite, or one with a limited gastric capacity, will need more frequent feedings. The following statements can therefore apply only in a general way, being viewed as average figures for average babies: After the first 2 days, in which the child is fed only every 4 or 6 hours, the interval between nursings, *i.e.*, the *beginning* of each nursing, should be of 2 or oftener 2½ hours duration during the daytime, with only 2 nursings after 9 or 10 P.M. until morning, there being in all from 8 to 10 nursings. This holds good for the first 4 weeks. From 4 weeks to 3 months the interval should be 2½ or, preferably, 3 hours in the daytime, with only 1 nursing after 10 P.M., the total number being 7 or 8. From 3 months until the age of 4 or 5 months the interval should be 3 to 3½ hours by day, perhaps with 1 nursing after 10 o'clock at night in case the child wakens for it. The total number of nursings is, therefore, 6 or 7 in 24 hours. From this time until the age of 1 year the intervals should be 3 to 4 hours, but no night-nursing after 10 P.M. is required, and the total number equals 5 or 6. The following tabular arrangement shows in convenient form the intervals and number of nursings, not including the first 2 days of life:

TABLE 32.—FREQUENCY OF NURSINGS

Age	Intervals, hours	No. of feedings in 24 hours	No. of feedings between 10 P.M. and 6 A.M.
Birth to 4 weeks	2–2½	8–10	2
4 weeks to 3 months	2½–3	7–8	1
3 months to 4 or 5 months	3–3½	6–7	1 (?)
4 or 5 months to 12 months	3–4	5–6	0

It may be again emphasized that these figures are only a guide, some infants requiring fewer and some more frequent nursings at certain ages. The great principle is, that a rule, once established, and which has been found satisfactory, must be followed with uniformity, unless some very special reason arises for modifying it. The determining of the exact hours of the day for the nursings will depend in part on the time the infant regularly wakens in the morning, and in part upon the hours of its regular daytime naps. The child should be awakened for its food when the appointed time comes during the daytime and at the 9 or 10 P.M. feeding. This will soon train it to waken of its own accord at the proper time. During the night it may sleep as long as it will, the hours for nursing being movable ones.

Amount of Food.—This can be determined with even less fixity than in the case of the intervals, owing to the very different demands of different infants. It varies also from time to time in the same infant. If an unusually large amount is ingested at one nursing, the child will naturally require less at the next one, or perhaps even refuse it altogether. This occurrence need, therefore, be no cause of alarm in the case of a healthy infant. The important consideration is that a sufficient total quantity of breast-milk be taken in the 24 hours. Reference to the table upon page 90 will show what may be regarded as the average amount taken at each nursing and during the whole day. The matter is referred to again in considering Artificial Feeding (p. 135). It is a point of interest, as pointed out by Feer,[1] that in any event the greater part of the milk is ingested by the infant in the first 5 minutes of nursing. Smith and Merritt[2] found that from 40 to 60 per cent. of the milk was obtained in the 1st 2 minutes and from 60 to 85 per cent. in the first 4 minutes; while after 8 minutes very few babies received any milk.

Causes Making Nursing Inadvisable or Impossible.—Many causes may render nursing impossible or harmful. (See p. 102.) Pregnancy is one of the chief of these, the milk becoming insufficient or indigestible, or the secretion disappearing entirely. The return of menstruation is quite frequently regarded by mothers as a necessary cause for weaning. This is a mistake. The milk may be temporarily altered to a slight degree or may exhibit no change whatever. It is true that such an occurrence sometimes heralds a permanent cessation of secretion, but there is no need by weaning to anticipate this. The development of an acute brief illness may render a temporary cessation necessary, this depending altogether on the individual case. The effect upon the mother is that to be considered; and a decision against nursing should not be made too hastily. If it is determined to give breast-feeding under these circumstances, every precaution should be taken, such as the wearing of masks and the like, to prevent infection being carried to the baby. If diphtheria is the disease present preventive serotherapy may be employed. The occurrence of a long-continued illness, such as typhoid fever, generally prohibits nursing, as the milk is liable to be poor and the demands upon the strength of the mother too great. The presence of healed tuberculosis is a contra-indication, chiefly because the drain upon the mother is too severe. A woman with active tuberculosis, especially of the lungs, must not nurse, both for her own sake, and on account of the danger of infecting the infant through the close contact. The likelihood, however, of this infection occurring through the milk during its secretion is very slight indeed. Sepsis, nephritis, puerperal eclampsia, diabetes, insanity, and frequently repeated epileptic seizures, are contra-indications against nursing. Other chronic illnesses or the existence of a delicate state of health may contra-indicate nursing, both because of the harmful effect upon the mother, and because the milk is liable to be of poor quality. However important breast-feeding may be for the infant, the mother's health should be the first consideration. One must be sure, however, that nursing will undoubtedly be harmful to her. It is true that the strain of nursing often does cause loss of weight and of strength in an anemic or neurotic mother; but it is generally a condition which can be tolerated without any permanent harm, certainly for a time at least, and the effort at nursing ought to be made. Syphilis existing in the mother is usually no contra-indication to her nursing, since the child is almost invariably syphilitic, even though it appears healthy and without manifestations of the disease. In a small percentage of cases, however, the infant may escape being syphilitic when the mother is diseased. The Wassermann reaction of the infant's blood within its limitations may be depended upon to settle the status, and if this is negative it is better that the infant be not nursed. On the other hand, the mother of the syphilitic infant is always infected, and even though she may be in a latent stage and have a negative Wassermann reaction she may nurse her infant without danger to herself. (See Syphilis, pp. 658, 675.) Mastitis prohibits nursing from the diseased breast as long as the suppuration continues. This, however, is disputed by many (see Moldenhauer).[1] Retraction of the nipple which cannot be overcome, and fissures which will not heal, causing unbearable pain, often make the continuance of nursing impossible. Sometimes an artificial nipple will overcome the difficulty, and in any case thorough trial of nursing should be made before this is abandoned. Sheets[2] reports a case of congenital occlusion of the lacteal openings in the nipples. Puncture through the nipples allowed nursing to be established. The secretion of milk on which the infant persistently does not thrive makes weaning imperative. There exists, however, a far too great readiness on the part of physicians to advise weaning under these circumstances, without sufficiently long-continued efforts to remove the difficulty.

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[1] Jahrb. f. Kinderheilk., 1896, XLII, 225.

[2] Amer. Jour. Dis. Child., 1922, XXIV, 413.

On the part of the infant nursing may be temporarily or permanently interfered with by severe coryza, cleft palate, or conditions of great debility. The last is seen especially in premature infants, or those with congenital asthenia from other causes, the baby being too weak to suck properly. In this event the breast-milk must be fed from a dropper or a Breck feeder until the child is stronger.

Signs that Breast-feeding is Not Satisfactory.—The breast-milk may be insufficient in quantity; the child may for various reasons fail to get enough of what is in the breast; or the milk may be inferior in quality. Not every breast-milk is suitable for the infant even when sufficiently abundant. It is important to determine as promptly as possible whether nursing is satisfactory, as otherwise valuable time may be lost and damage done. The normal infant shows a steady gain in weight according to the rates already given (p. 9). This does not mean, however, that the gain must be regular and unbroken from day to day, for such is by no means the rule; nor does it indicate that a gain which is not quite up to the normal figures necessitates a change in the diet. A baby who is receiving a supply of milk defective in quality or quantity constantly falls behind in its weight-curve and in its appearance of well-being; is liable to become hungry too soon; to cry much of the time when or before nursing is properly over; to sleep very little; and often to have too few bowel-movements in the twenty-four hours, and these are frequently constipated. Weighing the baby or the mother before and after each nursing will show whether the absolute daily quantity of milk ingested is insufficient (p. 90). Sometimes a weight-curve which has been entirely satisfactory perhaps for some months makes a decided deviation from the normal during 1 or 2 weeks, yet without there being any other noticeable disturbance in the condition of the child. This is very liable to mean that the maternal secretion is unsatisfactory. In other cases it is evident that the milk produces symptoms of indigestion. Actual vomiting should not occur in the normal infant, unless as a result of its being disturbed in some way. It may not infrequently be on account of indigestion having developed, either from the milk being too abundant or too rich in some respect. For the same reason the stools may be too frequent or not well digested; and the occurrence of colic is a very common evidence of indigestion. If with these symptoms the baby is still thriving normally in other respects, there can be no thought of weaning, although an effort at a modification of the secretion (p. 106), or of some of the details of the method of feeding should be made, and treatment for the indigestion given. Sometimes, if the failure to gain has been of but short duration, the giving of a purgative such as castor oil, or the temporary cessation of breast-feeding for a day or two, will permit the infant to resume nursing, and a normal gain in weight may be hoped for. Again, a child may fail to gain on account of the existence of some disease other than a digestive one, and a careful study of the case must be made for some concealed ailment before concluding that the breast-milk is unsatisfactory. The special symptoms produced by an excess of the different elements of the food are referred to more conveniently under Artificial Feeding (p. 128). Idiosyncrasy to breast-milk has been described by a few authors (Sztark),[1] (Kreutzer and Mendilaharzu).[2] In these cases there occurs fetid diarrhoea, colic and vomiting following breast-feeding, and a cessation of these symptoms when other food is substituted. Hypodermic injections of from 1 to 5 c.c. (0.03 to 0.17 fl. oz.) of mother's milk repeated 2 or 3 times are said to desensitize the infant and allow the continuance of breast-feedings without digestive disturbance. (See also Allergy, p. 754.)

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[1] Zeitschr. f. Kinderheilk., 1921, XXIX., 1.

[2] Jour. Amer. Med. Assoc., 1920, LXXX, 551.

MIXED FEEDING

Very many women secrete only enough milk to nourish the child in part. Such circumstances render it necessary either to wean, to employ a wet-nurse, or to supplement with artificial food. The last plan is greatly to be preferred to complete weaning, provided the mother's health is not suffering and her milk is of good quality. To obviate the former it is important to see to the general health of the mother, and especially so to manage that she shall have her sleep at night undisturbed. To determine the latter the milk should be analyzed and then, if necessary, an attempt be made to improve its quantity or quality. If efforts to improve it fail, substitute feedings should be commenced, at first a little weaker than normal human milk and then, when tolerance of the new food is established, of such percentage-strength as the gain in weight shows to be requisite. So, too, even though the mother's milk may be sufficient, partial substitute feeding may well be instituted if her health is distinctly suffering. The number of substitute feedings, and the time in the twenty-four hours for them, depend always on individual circumstances. They may either replace entirely some of the feedings, the mother nursing at other times from both breasts if necessary, or a small bottle; *i.e.*, a *supplemental* or *complemental* feeding, may be given after each nursing. The latter is in many respects the better plan, so far as the baby is concerned, since the more frequent stimulation of the breast by nursing tends to maintain the supply of milk. In fact, the giving of two

[1] Arch. de méd. des enf., 1921, XXIV, 555.
[2] Semana Med., 1922, II, 242.

or more bottles daily without nursing immediately preceding this is extremely liable to hasten an entire disappearance of the maternal secretion. From the side of the mother, however, the giving of one or two bottles daily without nursing has the advantage that it furnishes her with an increased degree of freedom. Systematic weighing, at least twice a week, will be the indication of the success of the mother and of any necessity to wean entirely and quickly. As to the amount to be given at each feeding, this depends upon the age of the child, and the quantity it obtains from the breast. To determine this the baby may be weighed before and after each nursing during one or two days, and then given enough of a supplemental feeding to make up the total quantity which the age would naturally require in the average infant. (See p. 135.)

It is frequently advised to give a baby one bottle of artificial food from the beginning, in order that it may become accustomed to this in case of the maternal supply failing. This seems like meeting a possible danger by undergoing a real one; and it is not necessary if the infant is early trained to take water from a bottle. The only advantage is the freedom for the mother already mentioned, and this may often be obtained by pumping or expressing the milk, keeping it carefully on ice, and feeding it from a bottle at the proper time. It is of common occurrence in attempting to employ mixed feeding to find that the infant soon will refuse the breast and take only the bottle, finding nursing from it easier. Judicious starving will generally overcome the difficulty. Sometimes putting a little sugar or some of the bottle-food on the maternal nipple, or the employment of an artificial rubber-nipple, will persuade the infant to nurse. Always the breast should be given first, and then the supplemental feeding.

It does not necessarily follow that mixed feeding, once commenced, shall continue indefinitely. The mother may be suffering from a temporary impairment of health, or the milk may have diminished as a result of maternal anxiety or other psychic cause; or the difficulty may exist only in the first few weeks of the infant's life. It will then be found, with the increasing supply of milk, that the infant has no desire for the bottle and is yet gaining weight normally, and a purely maternal nursing is thus reinstated. During the period of mixed feeding, however, it is very important to see that the mother's breasts are frequently emptied as thoroughly as possible in order to stimulate the secretion.

WEANING

With very many women the supply of milk is liable to diminish greatly by the time the infant is 8 or 9 months old, or even, as previously stated, before this. Weaning is thus rendered necessary early. When, however, nothing of this sort occurs, the child should be fed on the breast alone during the first 9 or 10 months of life. We should certainly attempt to have maternal nursing continue until the infant is 5 or 6 months of age, after which period substitute feeding will be better tolerated. Nursing longer than 12 months generally offers no possible benefit and is often deleterious to the infant. It is not infrequently practised by mothers in the hope of preventing conception.

Weaning should not be done in hot weather if it can be avoided. At this season, if the child is clearly thriving on the breast-milk and the mother is not suffering, it is well to prolong breast-feeding beyond the usual time for weaning and until the hot weather is over. Exceptionally the reverse is true, and weaning, or at least mixed feeding, may well be hastened before hot weather sets in. This reason is, however, not often adequate, and the step must be taken cautiously. With proper guarding against the employment of an impure milk, a baby may readily be weaned in the summer-time if necessity arises.

Some of the causes which make weaning necessary have already been discussed (p. 83). The sudden cessation of the maternal supply, the secretion of milk which is harmful to the child, or decided illness on the part of the mother, may compel a sudden complete weaning. So, too, the absolute refusal of the child to take anything but the breast may require it. This last is a very real and not infrequent factor in cases where the mother's milk is not entirely sufficient. It is sometimes necessary to withdraw the breast absolutely and almost to starve the child for several days before it will take its artificial nourishment. This can often be avoided by teaching the breast-fed child, early in life, to suck water from a bottle. When, however, such an emergency arises, the bottle should be offered to the child in the mother's absence, as the infant is more prone to be content with it under these circumstances.

Whenever possible, however, weaning should be done gradually. The child should be started at the age of 9 or 10 months with the mixed feeding described. At first only one bottle is given daily, using a formula much weaker than the needs actually demand, and gradually increasing the strength of the food until one sufficiently nourishing is found to be well tolerated. In a few days another breast-feeding should be omitted and two feedings of milk-mixture given. This process is continued until the infant is entirely on artificial food. After this the strength and quality of the food is to be increased gradually in the manner to be considered later (p. 133).

FEEDING BY A WET-NURSE

Advantages.—The milk of a wet-nurse is beyond question generally the best substitute for mother's milk. In fact in many cases it may be better than the milk of the mother. Many infants have been saved by wet-nursing who would unquestionably have perished if artificial feeding had been continued. This is particularly true of marantic infants with serious digestive disturbances. So too, the rearing of young infants, normal at the start, with artificial food in the heat of summer, and especially in the cities, is often of such difficulty that wet-nursing is greatly to be desired, and this should certainly be urged by the physician, if a brief trial with artificial food shows any deterioration of health.

Yet, on the other hand, it is a mistake to regard wet-nursing as an unqualified good. Very many wet-nurses are prone to take advantage of their important position, become overbearing and insolent, and be a trial in the household which words cannot describe. The nurse often turns out unreliable and may abandon her position at some most inopportune time. It does not follow, too, that the milk of the wet-nurse will agree with her foster-child. Sometimes milk, at first abundant and good, becomes altered by the changed methods of living which the nurse experiences in her new position or by anxiety or nervousness, although this may be only a temporary matter. For all these reasons mothers dislike the introduction of a wet-nurse into the household. Very many of them, too, have so strong a prejudice against having their babies nursed by another woman, that they prefer seeing them die rather than consent to it, and I have heard them frankly admit this. Others draw the line at the engaging of a woman other than white. All this is naturally wrong and distinctly selfish, since the baby's good should be the first consideration. Equally wrong, and often fatal in its results, is the deferring of the employment of a wet-nurse until it becomes a last resort.

It is sometimes urged that the employment of a wet-nurse is inhuman and selfish on the part of the employer, because it forces the nurse to deprive her own infant for the sake of the foster-child. This is altruistic in theory, but illogical in fact. The wet-nurse's own child is probably in a healthy and flourishing condition, or the woman would not have been selected as a nurse. The foster-baby is probably having dangerous digestive trouble existing or threatening, or a wet-nurse would not have been sought. By employing a wet-nurse there is much greater chance of saving two lives instead of one. Still more important is the fact that the wet-nurse takes this position to obtain the livelihood of herself and child. If she did not do this, she would be obliged to take up some other and less well-paid work, and in this event she could not nurse her baby.

The Wet-nurse's Baby.—There is no objection to the wet-nurss taking her own baby with her into her new position. In fact, she is less liable to be anxious about it, and this conduces to the maintenance of her supply of milk. On the other hand, it is bad policy, in my experience, to make any arrangement whereby she shall try to supply milk for both babies. The foster-baby is the one likely to suffer in this event. Indeed, the only vital objection against the presence of the nurse's baby in the house is the difficulty in preventing the nurse from a surreptitious defrauding of the needy foster-child. Unless such a procedure can be absolutely controlled it is better that the nurse leave her own baby behind. There are instances, however, when the opportunity for nursing both children is an advantage. This is especially true when the supply of milk is very abundant, since the foster-baby may be overfed, or if too weakly to empty the breast properly the secretion is not maintained and may dry up. The employment of a breast-pump, or expression of the milk, is by no means as satisfactory for this emptying as is the direct sucking by a vigorous infant. Whether or not the wet-nurse's child accompany her, it is only humane that the employer see to it that the infant is well cared for.

Selection of a Wet-nurse.—The wet-nurse selected should be strong, between 20 and 30 years of age, of quiet disposition, and not nervous. Other things being equal, her general health should be good, although delicate looking wet-nurses not infrequently supply excellent milk. Particularly she should be not anemic, not too fat, and free from any evidence of syphilis or tuberculosis, or of parasitic or infectious disease of the skin. The presence of a positive tuberculin-reaction is, of itself, not a contra-indication to her employment. The existence of apparent health in the mother, and especially in her own child, makes it probable that syphilis is absent; but whenever possible a Wassermann test should be made. Conversely it may be said that a syphilitic foster-baby should not nurse from a healthy wet-nurse. The nipples must be prominent enough to be grasped easily by the child, and be free from fissures. The mammæ should be firm and hard before nursing, and should become flabby to some extent when emptied. If they do not, their size and shape before nursing probably depend on the fact that they contain more fat than glandular tissue. The best test, however, of the amount of secretion is the weighing of the infant before and after nursing, since the appearance of the breast is so often deceptive. The weighing should be done after each nursing for a day or more. A single weighing is not satisfactory. In connection with the weighing it may be said that this is a check, too, upon a young infant receiving far more milk than it can properly digest. (See Quantity of Milk, p. 90.)

The nurse's baby should by preference be some weeks old in order to show that the milk has been nutritious, and that a condition of equilibrium has been reached. If her child is healthy and well-nourished it is an indication that the milk will probably agree with the foster-child. There is no necessity of having the nurse's own child and her foster-child of very nearly the same age, inasmuch as there is comparatively little change in a woman's milk after the 1st month. (See p. 102.) Yet, on the other hand, the difference should not be too great, especially if her own child is much older, lest the quality deteriorate and the secretion finally cease before the younger foster-child is of the age to wean.

When practicable the nurse's milk should be analyzed before she is employed. Should her milk not seem sufficiently abundant during the first days after she is engaged, one must not at once despair of her, for the scantiness may be remediable by hygienic treatment, or may be the result merely of nervous impressions such as excitement from assuming the new position, worry at weaning her child, and the like.

The moral character of the wet-nurse has no effect whatever upon the child; that is to say, traits of character are not transmitted with the food. It is similarly a matter of indifference whether her color is white or black. Yet her character cannot be entirely disregarded for other reasons. If she is intemperate, vicious, and irresponsible, an infant cannot be safely entrusted to her charge, however normal her milk-secretion may be. A wet-nurse who is a primipara should certainly not be rejected because she is unmarried. If she has had more than one illegitimate child she is probably depraved in other respects and should not be trusted.

Hygiene of the Wet=nurse.—The rules for the diet and hygiene of the wet-nurse are those already given for the nursing mother (p. 80). Yet it is important to remember that the diet of the household is perhaps not that to which the nurse has been accustomed, and that no sudden change to a richer and more delicate diet should be made, lest some alteration in the character or quantity of the milk due to indigestion or other causes result. It is important, too, that a woman who has followed an active working life, be not forced to sit about and do nothing. Such a course is sure to affect her milk. She should as far as possible do the kind of work, eat the sort of food, and in general live the life to which she has been accustomed.

Breast=milk Dairy.—It is difficult at times to procure a wet-nurse, and this becomes a problem not only in private practice but in institutions where large amounts of human milk could be used to advantage. The attempt to solve this has been made by Talbot[1] in Boston and Hoobler[2] in Detroit by collecting and pooling the milk from many nursing mothers; and Chapin[3] describes the operation of a breast-milk dairy in the management of which the mothers come to stations and the milk is extracted under supervision, in this way avoiding contamination and dilution. Emerson[1] has succeeded in drying human milk, and it may be found that this product will be helpful under certain conditions where fresh human milk cannot be obtained.

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[1] Jour. Amer. Med. Assoc., 1911, LVI, 1715. Bost. Med. & Surg. Jour., 1913; CLXIX, 760.

[2] Trans. Amer. Ped. Soc., 1924, XXXVI, 55.

[3] Jour. Amer. Med. Assoc., 1923, LXXXI, 200.

HUMAN MILK

Quantity.—The quantity secreted depends largely upon the demands made by the infant. Consequently there is less milk supplied in the early period of infancy than later. The estimation of the average secretion can therefore be only approximate. Systematic studies extending over some months, made by weighing infants before and after each nursing, have been carried on by very few investigators. Feer[2] has combined a number previously published with experiments of his own. The following figures deduced from his table represent the average total daily amounts taken by the infants studied, as well as the average at each nursing during the 1st half year:

Table 33.—Secretion of Breast Milk

Age	Average amount taken daily		Average amount at each nursing	
	Grams	Fluidounces	Grams	Fluidounces
1st week..........	310	10.5	66	2.2
2d week..........	558	18.9	90	3.0
3d week..........	601	20.3	97	3.3
4th week..........	666	22.5	111	3.7
5 to 8 weeks.......	725–818	24.5–27.6	125–141	4.2–4.8
9 to 12 weeks......	800–832	27.0–28.1	138–146	4.7–4.9
13 to 16 weeks.....	847–879	28.6–29.7	154–157	5.2–5.3
17 to 20 weeks.....	842–922	28.5–31.2	153–174	5.2–5.9

During the first 2 days of the 1st week the amount taken by the child is necessarily much less than later, only 10 to 20 c.c. (0.34 to 0.68 fl.oz.), since secretion has not been fully established. The figures obtained by Selter[3] in 3 systematically studied cases are somewhat lower than those of Feer, and Ramsey and Alley,[4] in a study upon 300 new-born infants of normal weight, showed that in the first 10 days of life infants will thrive on amounts distinctly less than those in the table. The average daily quantity taken ranged from 39 c.c. (1.33 fl.oz.) on the 2nd day to 365 c.c. (12.41 fl.oz.) on the 10th day of life. All estimates made in this way, however, show only the amount of milk taken by the child, and not that which the breast is capable of secreting. An average breast should give an average secretion of 1200 grams (40.6 fl.oz.) a day during the nursing period (Schlossmann).[5] A wet-nurse feeding several infants may not infrequently secrete daily in the neighborhood of 2 litres (67.6 fl.oz.), or occasionally even 3 litres (101.4 fl.oz.). Laurentius[6] observed an instance where 3450 grams (116.66 fl.oz.) were produced in a single day; Rommel[1] one with 4125 grams (139.48 fl.oz.), and Brodsky[2] a secretion of 5400 c.c. (5.7 quarts) in one day.[3] In the last case there was a total secretion of 1193.279 litres (1260.10 quarts) in 339 days.

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[1] Jour. Amer. Med. Assoc., 1922, LXXVIII, 641.

[2] Jahrb. f. Kinderheilk., 1896, XLII, 195; 1902, LVI, 421.

[3] Arch. f. Kinderheilk., 1903, XXXVII, 91.

[4] Amer. Jour. Dis. Child., 1918, XV, 408.

[5] Arch. f. Kinderheilk., 1902, XXXIII, 194.

[6] Arch. f. Kinderheilk., 1911, LVI, 275.

Colostrum.—The secretion occurring during the first 3 or 4 days after the birth of the child, and to some extent before its birth, is called "colostrum." It differs from milk not only to some degree in chemical composition, but in the presence of the "colostrum corpuscles." These are cells probably, of lymphoid nature as Czerny[4] believes, or derived in part from the "mast" cells (Unger).[5] They are 4 or 5 times larger than the average leucocyte, are phagocytic, contain a nucleus, are filled with fat-globules, and are at first very numerous (Fig. 16). They persist in constantly decreasing numbers until the end of the 2d week. A longer continuation indicates that the milk is not in a healthy state. The corpuscles reappear also when nursing is intermitted for a day or two, or when the breasts are not sufficiently emptied. Any breast-milk in which colostrum-corpuscles are present in decided numbers is liable to disagree with the infant. In addition to the corpuscles and fat-globules, mononuclear and polymorphonuclear leucocytes may be found. The fat-globules of colostrum are very unequal in size.

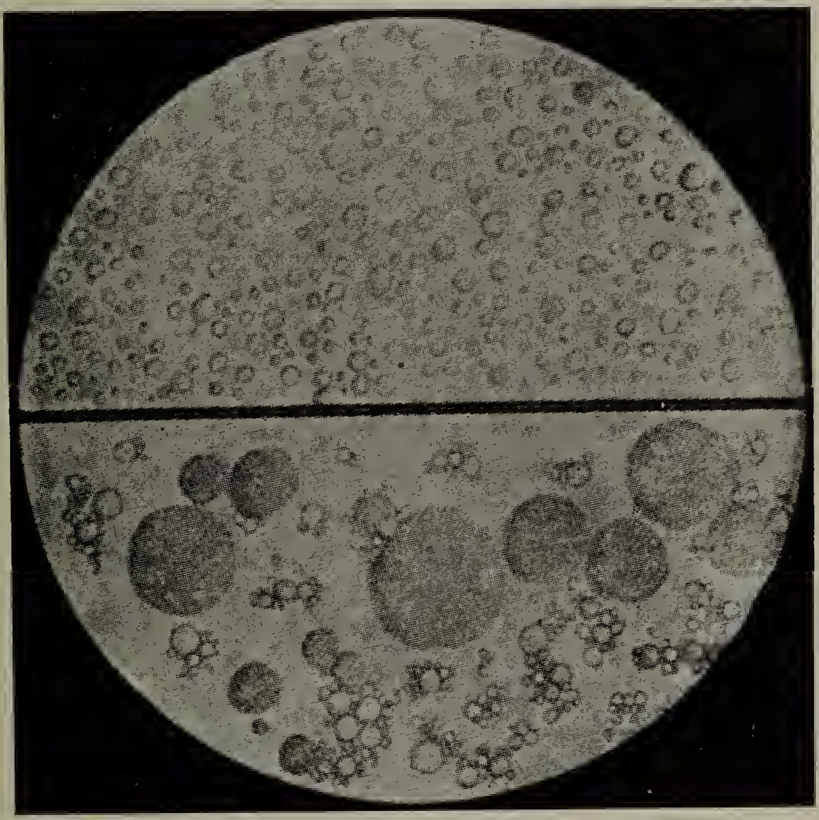

Fig. 16.—Milk and Colostrum.

Upper half human milk; lower colostrum. Magnified. (*Thiemich, Feer's Lehrbuch der Kinderheilkunde,* 1914, 6.)

Most investigators agree that the colostrum of the first few days before milk begins to be secreted is richer in protein and salts and poorer in sugar and fat than the fully-developed milk. The results from analyses made by different investigators is shown in the following table:

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[1] Münch. med. Wochenschr., 1905, LII, 444.

[2] Arch. f. Kinderheilk., 1914, LXIII, 166.

[3] The calculations of the English measures as given above are on the assumption that grams were taken as the equivalent of cubic centimeters. If the milk was weighed, the volumes secreted will be slightly less than the figures given.

[4] Henoch's Festschrift, 1890, 194.

[5] Virchow's Archiv., 1898, CLI, 159.

TABLE 34.—COMPOSITION OF COLOSTRUM

	Adriance[1]	Woodward[2]	Söldner[3]	König[4]
Fat	3.46	4.00	3.26	3.34
Protein	2.05	1.90	1.79	3.07
Lactose	6.35	6.50	5.83	5.27
Ash	0.23	0.20	0.30	0.40
Total solids	12.10	12.50	12.09	12.08
Water	87.89	87.50	87.91	86.70

The difference in these figures depends largely upon the material analyzed. The analysis of König is of the colostrum of the first 3 days; the others of that of the first 5 or 6 days or longer.

The color of colostrum is deep lemon-yellow due to the presence of the corpuscles, the reaction decidedly alkaline, and the specific gravity 1040 to 1060. It is coagulated firmly by heat and by acids. The excess of protein and of salts is perhaps the cause of the slightly laxative action which colostrum is believed to possess. The fat of colostrum is richer in oleic acid than is that of human milk (Engel);[5] and according to Bauer[6] the protein is of the nature of a direct transudate, in contradistinction to that of fully developed milk, and is analogous to that found in blood-serum. The mineral matter differs from that of milk in that magnesium and phosphorus are in relatively greater amounts and possibly the calcium also, while the proportion of sodium is smaller.

The chemical characteristics of colostrum persist to some extent, although in a constantly diminishing degree, during the first 2 weeks after childbirth, by which time the "colostrum period" may be said normally to have closed, and the "equilibrium milk" or "mature milk" is secreted.

Composition of Human Milk.—Human milk, like that of other animals, is an emulsion consisting chiefly of water, containing suspended or dissolved in it certain amounts of fat, sugar, protein and salts. It is the product of actual secretion by the breast, the epithelial cells not undergoing destruction in the process of its formation. It is not a transudate, although to a limited extent substances, such as certain drugs, may simply pass through the gland and be excreted in the milk. It begins to be secreted by about the 3d day after birth, or sometimes later, the characteristics of colostrum being still present to some extent. It is of a bluish-white color and has a slightly sweetish taste and an amphoteric or faintly alkaline reaction with litmus paper. Kerley, Gieschen and Myers[7] have shown that with phenolphthalein it is always faintly acid. The white color is not due solely to the existence of fat in emulsion, but probably also to the presence of casein and calcium phosphate, since it is to be seen as well in fat-free milk. The specific gravity averages about 1030 to 1031 with a normal range of 1028 to 1034. Greater variations occur, and König[8] gives a range of 1020 to 1036. Coagulation is caused by acid, but only to a slight degree as compared with cow's milk; and that by rennet does not take place except in the presence of acid, and then less firmly than in the case of cow's milk. The casein is changed to paracasein under the influence of rennet. Under the microscope are found crowded oil-globules varying in size (Fig. 16). In addition to these are a limited number of leucocytes, and a very large number of extremely minute ultramicroscopic particles consisting of casein (Alexander and Bullowa).[1]

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[1] Arch. of Pediat., 1897, XIV, 22.

[2] Jour. Exper. Med., 1897, II, 217.

[3] Zeitschr. f. Biol., 1898, XXXVI, 280.

[4] Chemie des menschl. Nahrungsmittel, 1903, I, 100.

[5] Sommerfeld's Handb. der Milchkunde, 1909, 810. Ref. Morse and Talbot, Diseases of Nutrition and Infant Feeding, 1915, 96.

[6] Deutsch. med. Wochenschr., 1909, XXXV, 1657.

[7] New York Med. Rec., 1903, LXIV, 201.

[8] *Loc. cit.*, 110.

Numerous and careful analyses of the composition of milk have been made. There is necessarily a considerable variation among individual women and those of different races and conditions of life, and even of the same woman at different times of the day. The following table, after Czerny and Keller,[2] shows some of the variations according to the analyses of different investigators:

TABLE 35

Investigators	Fat, per cent.	Sugar, per cent.	Protein, per cent.	Ash, per cent.	Solids, per cent.
Pfeiffer[3]	0.76–9.05	4.22–7.65	1.049–3.04	0.104–0.446	8.23–15.56
Johannessen and Wang[4]	2. 7–4.6	5. 9–7.8	0. 9–1.3		
V. and J. S. Adriance[5]	1.31–7.61	5.35–7.95	0.23–2.60	0.09–0.28	9.19–15.31
Guiraud[6]	1.75–6.18	6. 7–7.7	0.85–1.4	0. 1–0.27	11. 2–16.3
Camerer and Söldner[7]	1.27–5.77	5.35–7.52	0.83–1.87	0.11–0.36	9.41–14.11
Schlossmann[8]	1.65–9.46	5.2–10.90	0.56–3.4		

For an approximate analysis of average human milk the following may be assumed as a working guide:

TABLE 36.—AVERAGE COMPOSITION OF HUMAN MILK

Fat	3.5– 4.0 per cent.
Sugar	6.5– 7.0 per cent.
Protein	0.1– 1.5 per cent.
Ash	0.2 per cent.
Water	87.0–88.0 per cent.
Total solids	12.0–13.0 per cent.

There are also present in small amounts certain nitrogenous substances not of the protein class; citric acid; and a number of other bodies little understood.

THE DIFFERENT CONSTITUENTS OF MILK AND THEIR NORMAL VARIATIONS

Fat.—The fat in milk is in the form of many globules of size varying from 0.0033 to 0.01 mm. (0.00013 to 0.00039 inches) in diameter, and in number averaging 1,026,000 to the c.mm (Bouchut).[9] These form an emulsion with the milk-plasma. It is disputed whether the globules are surrounded by a distinct albuminous membrane. There is clearly some form of coating which prevents their coalition one with another, but the exact nature of this is not known. Abderhalden and Völtz[1] maintain that it is not casein, and perhaps is a mixture of different proteins. Chemically the fat consists principally of the oleate, myristate, palmatate and stearate of glycerole, the first being especially abundant. The others, as well as the volatile acids—butyric, capric, caproic, and caprylic—are present in small amount as compared with cow's milk (Ruppel);[2] viz., 2.5 per cent. of the total fat in human milk; 27 per cent. in cow's milk (Morse and Talbot).[3] The proportion of the total fat in the milk is capable of great variation, more than that of any other constituent. The percentages may certainly range from 2 up to 5 or even more in milk which may yet be called healthy for practical purposes. Not infrequently these limits are much exceeded, the percentage dropping occasionally to 1.5 or even less, while in one instance I observed it exceed 11 in milk otherwise normal. There has even been noted a decided variation in the percentages of fat dependent upon the time of day (Denis and Talbot).[4]

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[1] Jour. Amer. Med. Assoc., 1910, LV, 1196.

[2] Des Kindes Ernährung, Ernährungsstörungen und Ernährungstherapie, 1906, I, 416.

[3] Verhandl. XI, Versamml. d. Gesellsch. f. Kinderheilk., 1894, 126.

[4] Zeitschr. f. physiol. Chem., 1898, XXIV, 499.

[5] Arch. of Pediat., 1897, XIV, 27.

[6] Thèse de Bordeaux, 1897.

[7] Zeitschr. f. Biol., 1898, XXXVI, 280.

[8] Archiv f. Kinderheilk., 1900, XXX, 324.

[9] Gaz. des hôpiteaux, 1878, LI, 66; 75.

Sugar.—The sugar present in human milk is identical with, or closely allied to, the lactose occurring in cow's milk. Its proportion is less liable to vary than that of fat and protein, and is fairly fixed at from 6.5 to 7 per cent. in healthy milk. Variations decidedly below or above these figures sometimes occur.

Protein.—The protein-constituent of human milk is far from being a simple one and its chemistry is not yet fully understood. The total protein-matter, as studied by later and more accurate methods, may be given as from 1 per cent. to 2 per cent. within entirely normal limits, and with a decided capability of variation from these figures. A percentage of 3.5 or even more is not uncommon, and is sometimes digested well, though oftener not. Two principal protein bodies are uniformly recognized: casein and lactalbumin. There exists in addition the protein substance frequently described by some writers as "lactoglobulin," and still others have been reported. Wroblewski[5] described one rich in sulphur which he called "opalisin." Nuclein and lacto-protein are also reported. For practical purposes, however, only the first two need be considered. One of the principal characteristics of human milk as compared with cow's milk is the relatively large quantity of lactalbumin present, the relation which the amount bears to that of casein being, according to Lehmann[6] as 5 to 12; albumin with lactoglobulin 0.5 per cent.; casein 1.2 per cent. König[7] gives lactalbumin 1.21 per cent., casein 0.80 per cent., while in cow's milk, according to the same authority, the figures are 0.51 per cent. and 2.88 per cent. respectively. Ciccarelli[8] found lactalbumin from 62.1 to 73.1 per cent. and casein from 26.9 to 37.9 per cent. of the total protein. The results obtained by different investigators are clearly by no means uniform, but all agree that in human milk the percentage of lactalbumin as compared with that of casein is greater than obtains in cow's milk. Average figures would probably give lactalbumin and globulin 39 to 44 per cent.; casein 41 per cent.; residual nitrogen 15 to 20 per cent. (Talbot).[9] It is the

[1] Zeitschr. f. phys. Chem., 1909, LIX, 13.
[2] Zeitschr. f. Biol., 1895, XXXI, 1.
[3] Diseases of Nutrition and Infant Feeding, 1915, 107.
[4] Amer. Jour. Dis. Child., 1919, XVIII, 93.
[5] Zeitschr. f. phys. Chem., 1898–99, XXVI, 308.
[6] Arch. f. d. gesammte Physiol., 1894, LVI, 577.
[7] Chem. d. menschl. Nahrungs-u. Genussmittel, 1903, 110.
[8] La Pediat., 1908, VI, 912..
[9] Amer. Jour. Dis. Child., 1914, VII, 445

lactalbumin which contains certain essential growth-promoting amino-acids not present in casein.

The casein is a nucleoalbumin which is held in suspension in the milk-plasma through the action of the calcium phosphate. It is believed that it is not entirely identical with the casein of cow's milk, being not so readily coagulated by acids, salts or rennet, and the coagulum formed being fine and loose, and dissolving readily in an excess of acid. On the other hand, it has been claimed that this depends upon the diverse percentages present in the two forms of milk and the relative difference in the amounts of lactalbumin and of salts. Bosworth and Giblin[1] found the casein of cow's milk and of goat's milk practically identical. Whereas in cow's milk it is present as a calcium caseinate, in human milk it probably acts as a potassium caseinate. (See also pp. 109; 112.) Inasmuch as the coagulation of casein is favored by soluble calcium salts, and retarded by the salts of the alkalies, it may well be that the small amount of calcium and of casein and the larger amounts of sodium and potassium in human milk is the cause of the production in it of small and finely divided curds by the action of rennin (Bosworth).[2] Whatever the chemical differences, if any, may be, the work of Bordet,[3] Wassermann[4] and others, has shown from a biological standpoint that dissimilarities do exist between the caseins of different mammalian milks. The experiments have demonstrated, that whereas the blood-serum of animals sensitized to one mammalian milk will react with the protein of the milk of this species, it will not do so with other species.

The lactalbumin is in solution in the milk. It is allied to or identical with serum-albumin.

Non-Protein Nitrogen.—In addition to the nitrogenous bodies described as protein, there are others classified as "non-protein nitrogen," or "residual nitrogen." They constitute approximately from 15 to 20 per cent. of the total nitrogen of the milk. They consist in large part of urea, but other substances have also been described (Rietschel).[5]

Mineral Matter.—The salts of milk are a somewhat complex substance. A study of those of human milk by Harrington and Kinnicutt[6] gave the following results:

TABLE 37.—COMPOSITION OF THE MINERAL MATTER OF HUMAN MILK
(Harrington and Kinnicutt)

Calcium phosphate	23.87
Calcium silicate	1.27
Calcium sulphate	2.25
Calcium carbonate	2.85
Magnesium carbonate	3.77
Potassium carbonate	23.47
Potassium sulphate	8.33
Potassium chloride	12.05
Sodium chloride	21.77
Iron oxide and alumina	0.37
	100.00

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[1] Jour. Biol. Chem., 1918, XXXV, 115.
[2] Amer. Jour. Dis. Child., 1921, XXII, 193.
[3] Ann. de l'instit. Pasteur, 1899, XIII, 225.
[4] Verhandl. XVIII, Cong. inn. Med., 1900, 501.
[5] Jahrb. f. Kinderheilk., 1906, LXIV, 125.
[6] Rotch, Pediatrics, 1901, 130.

Another reliable analysis by Söldner[1] is as follows:

TABLE 38.—PERCENTAGES OF MINERAL MATTER IN HUMAN MILK
(Söldner)

Potassium oxide	0.0884
Sodium oxide	0.0357
Calcium oxide	0.0378
Magnesium oxide	0.0053
Ferric oxide	0.0002
Phosphoric oxide	0.0031
Sulphuric oxide	0.0090
Chlorine	0.0591

A series of analyses by Holt, Courtney and Fales[2] of milk of the middle portion of lactation gave the following results:

TABLE 39.—PERCENTAGES OF MINERAL MATTER IN HUMAN MILK
(Holt, Courtney and Fales)

Calcium oxide	0.0458
Magnesium oxide	0.0074
Phosphoric oxide	0.0345
Sodium oxide	0.0132
Potassium oxide	0.0609
Chlorine	0.0358
Total ash	0.2069

Probably the amounts of the ingredients vary much in different milks, and certainly in the fore-milk and the last milk of a single nursing. The total quantity of mineral matter in human milk is likewise subject to decided variation within normal limits, from 0.15 to 0.25 per cent. being a not unusual range. Human milk as compared with that of the cow is poorer in calcium and in phosphorus, but possesses a larger amount of iron. In neither milk is there sufficient iron for the infant's needs, but this is a matter of no consequence, at least in the earlier months of life, since the iron stored in the liver supplies the deficiency. The phosphorus in human milk is to a great extent in a different state from that contained in cow's milk. It exists largely in organic combination, and is believed to be chiefly in the form of lecithin and nucleone, both of which are in small amount in cow's milk. About 77 per cent. of the phosphorus in human milk is in organic combination, against about 27 per cent. in cow's milk. Most of the mineral matter of the milk is in solution except the calcium phosphate. Burhans and Smith[3] found the salts of the milk varying somewhat with the race, the calcium and organic phosphorus being decreased in Negroes and the chlorides increased. Other mineral salts may perhaps occur in milk other than those mentioned in the usual analyses. Thus Hess, Supplee and Bellis[4] have noted that copper is a regular constituent of human milk. Changes in the proportions of the mineral constituents may be produced by the long-continued use by the mother of a defective diet (Courtney).[5] Sisson and Denis[6] state also that decided variations in certain substances, such as chlorine, are frequent in mentally unstable women.

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[1] Zeitschr. f. Biol., 1903, XLIV, 61.
[2] Amer. Jour. Dis. Child., 1915, X, 229.
[3] Amer. Jour. Dis. Child., 1923, XXVI, 303.
[4] Jour. Biol. Chem., 1923, LVII, 725.
[5] Amer. Jour. Dis. Child., 1923, XXVI, 534.
[6] Amer. Jour. Dis. Child., 1921, XXI, 389.

Citric Acid.—This is present in an average amount of 0.05 per cent. (Scheibe).[1]

Ferments.—It has seemed probable from numerous investigations that various ferments and allied bodies play a rôle of some importance in rendering human milk better suited than cow's milk for the needs of the infant's economy. This is, however, very uncertain, since cow's milk, at least, appears to be as digestible after boiling, which destroys the ferments, as before. Moreover the difficulty in keeping the milk from the action of bacteria renders the study very perplexing, inasmuch as these may produce the same effects as do the ferments (Morse and Talbot).[2] A diastatic ferment, amylase, reported by Béchamp[3] and others, is claimed not to be present in cow's milk. A fat-splitting ferment, lipase, is stated by Marfan and Gillet[4] to be active in human milk, and but slightly so in cow's milk. One which decomposes salol is described by Nobécourt and Merklen[5] as present in human milk, but absent from cow's milk. The existence of this ferment is questionable. Moro and Hamburger[6] found one which coagulates fibrin, likewise usually absent from cow's milk. A proteolytic ferment is described by Babcock and Russell[7] and others, and a glycolytic ferment by Spolverini[8] as present in both kinds of milk. Superoxidase, peroxidase and reductase occur in both human and cow's milk.

Protective and Other Bodies.—Moro[9] showed that although no bactericidal substances were to be found in human milk, yet that the blood-serum of breast-fed children exhibited a bactericidal power much greater than that of those artificially fed, and that the former do not contract pyogenic diseases so easily. The protective power would appear to be drawn from the mother's milk. Diphtheria-antitoxin is found in the milk of immunized animals, and, indeed, the well-known comparative immunity of early infancy as regards many of the infectious diseases may depend in part on the presence in the breast-milk of immunizing substances, and the passage of these into the serum of the child. Infants born of mothers with typhoid fever may give the Widal reaction; and the Wassermann reaction may be positive in the breast-milk of syphilitic women (Klauder and Kolmer).[10] Although this is certainly true of the young of some animals, as the goat and the cow, the investigations of Kuttner and Ratner[11] with diphtheria antitoxin make it seem probable that in the human infant the transmission of protective bodies is chiefly through the placenta. Specific agglutinins are also probably transmitted to the child through the mother's milk. It is still, however, a debated question as to what degree the antibodies, agglutinins, and the like, are transmitted by the human placenta before birth, and to what degree through the milk. *Vitamins* are present in human milk in probably sufficient amount to satisfy all the needs of the infant, provided, however, that the mother has not been fed over long periods of time on a diet deficient in these elements (see p. 195).

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[1] Ref. Morse and Talbot, Diseases of Nutrition and Infant Feeding, 1920, 122.

[2] *Loc. cit.*, 129.

[3] Compt. rend. de l'acad. des sci., 1883, XCVI, 1508.

[4] Marfan, Traité de l'allaitement, 1903, 31.

[5] Rev. mens. des malad. de l'enf., 1901, XIX, 138.

[6] Wien. klin. Wochenschr., 1902, No. 5, 121.

[7] Rep. Wisconsin Agric. Station, 1898. Ref. Raudnitz, Ergeb. d. Physiol., 1903 II, 1 Abt., 285.

[8] Arch. de méd. des enf., 1901, IV, 705.

[9] Jahrb. f. Kinderheilk., 1902, LV, 396.

[10] Jour. Amer. Med. Assoc., 1921, LXXVI, 1635.

[11] Amer. Jour. Dis. Child., 1923, XXV, 413.

Caloric Value.—According to the estimations of Schlossmann[1] the average value of a liter of human milk in large calories is 782 (740 calories per quart), while that of average cow's milk may be assumed as about 670 calories (654 per quart). Heubner[2] gives 700 calories (663 per quart) for human milk and 690 (653 per quart) for cow's milk. (See Caloric Value of Cow's Milk, p. 110.) The caloric value of the individual milk-elements is considered elsewhere (pp. 46 and 123).

Action of the Different Constituents of Human Milk.—(See also pp. 40 and 128.)

Water.—The large percentage of water in the milk indicates the need of the infant especially in that particular. Concentrated food is not as easily assimilated as a rule by healthy infants, even though the actual amount of solid matter ingested is not altered, and the work thrown upon the excretory organs is also increased if sufficient water is not ingested. The amount of water required by an infant is proportionately many times greater than in adult life. There is little need, however, of water given by itself when the diet is entirely liquid, since enough is obtained in the food; but for other reasons it is well to accustom an infant to it. Including that contained in the milk, it is claimed (Widmer)[3] that in the first 5 weeks there is needed 127 c.c. for each kilogram (2.6 fl.oz. per pound) of body-weight, and at the end of the 1st half year 115 c.c. per kilogram (1.7 fl.oz. per pound). This constitutes respectively about 1/6 and 1/9 of the body-weight.

Fat.—One of the functions of the fat of the milk is that of increasing the weight of the body by the deposition of it in the tissues. It is also needed for the proper formation of certain tissues, notably of the nervous and osseous systems. A still more important function is the sustaining of the body-temperature. As already stated (p. 41) this object is gained by metabolic processes much more easily from the consumption of fat than from that of protein. This leaves the latter for the object for which it was primarily intended; *i.e.*, the formation of nitrogenous tissue. A deficiency in the amount of fat in the breast-milk requires that a very much larger amount of protein or of carbohydrate must be ingested than the needs of the child in other respects demand, or perhaps than its digestive powers will tolerate.

Another result of a proper amount of fat in the milk is a normal condition of the stools, which always contain a considerable quantity of it. Too little fat is liable to result in constipation. On the other hand, too high a percentage of fat is prone to produce disturbance of the gastric digestion and diarrhea, or sometimes constipation with soap-stools. The fact that so large a percentage of fat is contained in human milk points to its great importance in the food. In artificial feeding we are often compelled for various reasons to replace this to a certain extent; but this is not as Nature planned, and the fat should not be reduced unless conditions demand it.

Sugar.—Like the fat the function of sugar is the production of heat needed by the child. To a considerable extent it may replace the fat, both for this purpose and for the formation of new fatty tissue. Yet an infant who must rely for any length of time upon sugar to make up for a deficiency of fat in the milk is liable to suffer eventually; for, even though gaining in weight, tissue-formation is prone to be defective, and

[1] Arch. f. Kinderheilk., 1900, XXX, 324.
[2] Lehrb. d. Kinderheilk., 1911, 51.
[3] Jahrb. f. Kinderheilk., 1916, LXXXII, 177.

the strength is below normal. What is said here of the sugar natural to human milk applies equally well in a large degree to other varieties, as well as to the starchy food used later in infancy, since this latter must be converted into sugar before it can be absorbed. The effects of other sugars and of starch upon digestion are considered more fully elsewhere. (See pp. 41 and 130.)

Protein.—The protein is the essential tissue-producing substance of the milk, since it is the only one containing nitrogen. Too low a percentage of protein is certain to be followed eventually by malnutrition in some form, even though the infant may show no wasting. Protein produces heat in its consumption. Consequently an infant could sustain life and weight for a time on a diet solely protein in nature. To accomplish this, however, so large an amount is required that the general condition of the child would certainly be harmfully affected in some way; and for the production of heat the other elements of the food should be relied upon. (See p. 42.)

Mineral Matter.—Although the entire purposes of the salts are not understood, yet they certainly play an important and essential part in nutrition. The salts of calcium are required for the formation of osseous tissue. In addition calcium in the milk is a requisite for the proper action of rennet and a deficiency may produce nervous symptoms. (See Spasmophilia Vol. II, p. 526.) Phosphorus, too, is needed for the osseous and the nervous structures. The deficiency in the iron of the milk is made up by the withdrawal by the system of the iron stored in the liver.

Bacteria in Milk.—Healthy human milk generally contains a few bacteria. This has been shown by Honigmann,[1] Cohn and Neumann,[2] Ringel,[3] Köstlin,[4] and others. The germs make their way in through the nipple. Consequently the first milk drawn by the child when nursing is that in which they are contained. The remaining milk is generally sterile. The principal germs found are the staphylococcus albus and, less commonly, aureus (Köstlin). For practical purposes human milk may be considered germ-free. When the mammary gland is suppurating the number of germs in the milk is necessarily much increased. This may also sometimes be true where the woman is the subject of sepsis. Certain pathogenic germs, as those of tuberculosis and typhoid fever, may very exceptionally occur in the milk of women suffering from these diseases. Wang and Coonley[5] in 450 examinations of the milk of 28 tuberculous women found tubercle bacilli in no instance with 1 possible exception. Uhlenhuth and Mulzer,[6] inoculating rabbits with milk from syphilitic women, were able to produce in them the lesions of syphilis.

Blood in Milk.—Hemorrhage from the nipple may occur when there is fissuring of the nipple, in acute and chronic mastitis, in tumor or trauma of the breast, and as a form of vicarious menstruation. Occasionally it seems to be physiological, due to congestion following delivery. The subject is discussed by Abt.[7]

Characteristics and Effects of Poor Milk.—Owing either to the individual peculiarity of the baby, or to faults in the quality of the milk, some infants fail to digest the maternal secretion but thrive on that of a wet-nurse; or the milk which disagrees with one infant suits another perfectly. An analysis of the milk will often show in what the fault in composition consists. In other instances the milk is found to be chemically normal, yet does not agree or the infant fails to thrive. Much more important than the analysis is the character of the symptoms produced, and especially a failure of the normal gain in weight; and one can afford to temporize with moderate diarrhea, vomiting, or colic, rather than wean the baby if the weight is satisfactory. Of course this does not apply if the symptoms are severe. One of the faults of the milk is that of being too rich in both fat and protein; the percentage of sugar not varying materially in the milk of different women. This over-rich milk is oftenest observed in the case of women who are taking too little exercise, eating too freely, and digesting well. The infant exhibits the various symptoms of indigestion and often loses weight. Submitting to a proper dietetic and hygienic regimen will usually correct the difficulty. Occasionally it is possible to relieve the infant's condition by allowing it to nurse for a few minutes only and then giving it a bottle of water, thus diluting the milk in the stomach. Less often the fat is increased and the protein normal in amount. Sour vomiting and curds in the stools are then liable to appear.

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[1] Zeitschr. f. Hygiene u. Infectionskrankh., 1893, XIV, 207.
[2] Virchow's Archiv., 1891, CXXVI, 391.
[3] Münch. med. Wochenschr., 1893, XL, 513.
[4] Arch. f. Gynäk., 1897, LIII, 201.
[5] Jour. Amer. Med. Assoc., 1917, LXIX, 531.
[6] Deutsch. med. Wochenschr., 1913, XXXIX, 879.
[7] Arch. of Pediat., 1923, XL, 719.

Much more frequently, however, a poor milk is supplied containing either a low, or oftener a high, percentage of protein and a very low percentage of fat. Poor milk of this sort is frequently scanty in quantity, and is seen oftenest in neurotic, debilitated, anemic, over-worked, or over-anxious women. The infants do not thrive and may be constipated and exhibit no symptoms of indigestion whatever, or may sometimes suffer (if the protein is in large amount) from intestinal indigestion with colic and loose stools, often brownish and offensive. Vomiting is not a frequent symptom. Efforts should, of course, be made to improve the quality of the milk (p. 106) by attending to the condition of the mother; but when the trouble continues after the early weeks of the child's life it is not likely that these efforts will be successful.

Examination of the Breast-milk.—There are several indications that milk is being secreted in sufficient *quantity*. If the breasts fill up well and become hard and round between the nursings; if the infant nurses from but one breast and is satisfied by that; and if it does not require more than 20 minutes to complete its nursing, the probability is that the milk secreted is abundant. On the other hand, if the breast remains soft and flabby; if the child nurses much too long and seems dissatisfied, or will nurse but a few minutes and then refuse to make further efforts; if it cries much, yet seems to have no colic or other disease; and if it does not gain in weight, yet has no illness to account for this, the milk is probably insufficient in quantity or perhaps poor in quality.

A direct method is to pump out or press out the breast-milk and measure it. Yet this is often unsatisfactory owing to the frequently experienced difficulty in emptying the breast in this way. A rough estimation of the character and quantity of the milk may be made by seeing with what degree of ease and in what quantity it can be expressed from the breast by the hand. The "thumb-nail test" also is serviceable to a similar extent as a test of quality, a large drop—not, however, the first which comes from the breast—being drawn upon the thumb-nail, and its color, transparency and coherence indicating the richness of the milk. Normal milk should be white and opaque, and should preserve the drop-form even when the nail is held vertically. All this, however, is but approximate. The only satisfactory method of determining what amount of milk the child really obtains is by weighing it or the mother immediately before and immediately after the nursing, using scales which record ounces.

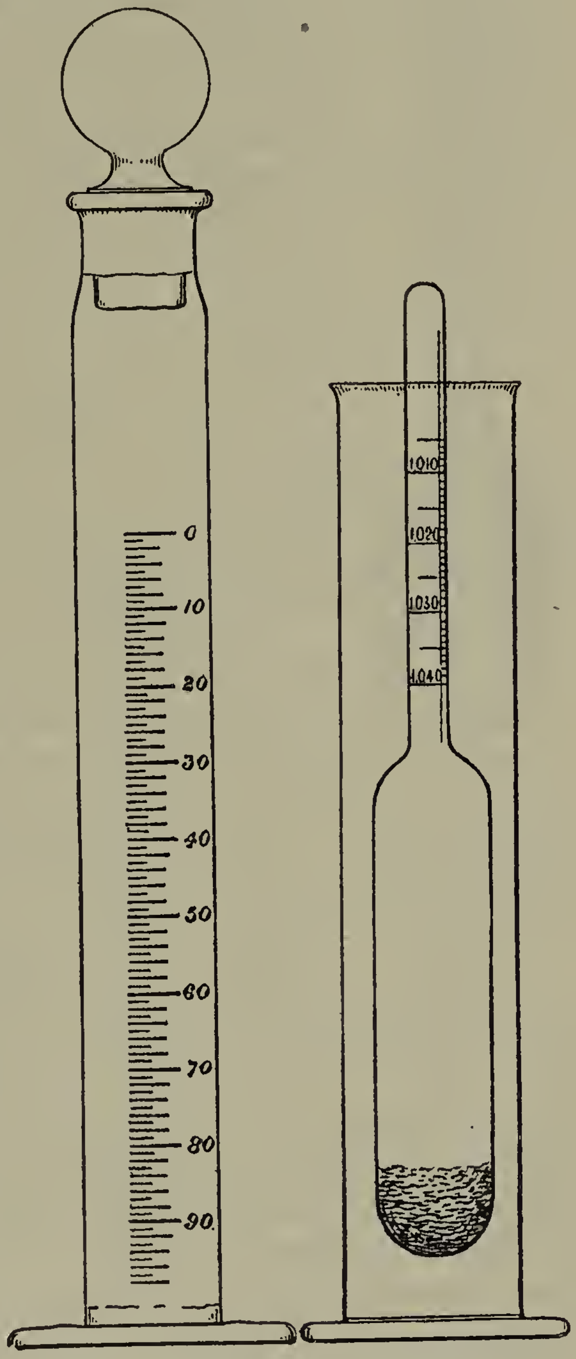

Fig. 17.—Cream Gauge and Lactometer.

For approximate analysis of human milk. (*Holt, Arch. of Pediat.*, 1893, *X*, 193; 202.)

As regards the examination of the milk in other respects, the *reaction* is tested by litmus paper, or still more accurately by a solution of phenolphthalein. The *specific gravity* is determined by any small specific gravity glass which does not require much milk. A small urinometer (Fig. 17) answers every purpose. The accurate scientific estimation of the *percentages* of the milk-elements, with the exception of the fat, can be made only by one having a considerable knowledge of chemistry. Various methods may be employed, however, which give satisfactory approximate results. The milk to be examined should preferably be all that can be pumped or expressed from the breast. Inasmuch as the percentages of sugar and of mineral matter are so nearly constant, it is only the variation in the fat and protein which we need determine for practical purposes. The percentage of fat is estimated by a small creamometer. This is a cylindrical vessel (Fig. 17) holding 10 c.c. (0.34 fl.oz.) and graduated to show the percentage of cream present. It is filled with milk to the zero mark, corked well and allowed to stand at the room-temperature of about 70°F. (21.1°C.) for 24 hours. The percentage of cream is then read off and that of fat calculated on the basis that this will be to that of the cream as 3 is to 5; *i.e.*, 5 per cent. of cream equals 3 per cent. of fat. The average amount of cream should be 7 per cent.

This method is only an approximate one, and a better procedure is the employment of the Babcock centrifuge, which, however, requires 17.6 c.c. (0.598 fl.oz.) of milk (Fig. 18). A similar apparatus needing but 5 c.c. (0.169 fl.oz.) (Fig. 19) is the milk-testing glass devised for use with any small centrifugal machine, such as is employed in urinary examination. The principle consists in the oxidation of the sugar and protein by sulphuric acid, with the addition of fusel oil and hydrochloric acid. The steps in the examination are as follows: (1) Shake up thoroughly the sample of milk to be tested, and with a pipette fill the glass up to the 5 c.c. mark. (2) With another pipette add concentrated sulphuric acid, 1 c.c. at a time, shaking and rotating the glass after each addition. Continue to add the acid until the lower part of the neck is reached. (3) Fill the neck of the glass almost to the top with a mixture of equal parts of amyl-alcohol and concentrated hydrochloric acid. (4) Centrifuge for 3 minutes, and then read the thickness of the fat-layer by means of the calibrations on the neck of the glass; reading always between the bottom of the upper meniscus and the bottom of the lower meniscus. The figures obtained represent percentages of fat.

The percentage of protein may be determined directly with reasonable accuracy by the method described by Boggs.[1] This consists in precipitating it with phosphotungstic and hydrochloric acids in an Esbach tube and reading off the percentage after 24 hours. The reagent consists of Solution A, containing 25 grams of phosphotungstic acid dissolved in 125 c.c. of distilled water, and Solution B, 25 c.c. of concentrated hydrochloric acid added to 100 c.c. of distilled water. The technique is as follows: (1) 2 c.c. of the milk is diluted up to 20 c.c. with water. (2) With this 1 to 10 dilution the Esbach tube is filled up to the mark "U." (3) A mixture of equal parts of Solution A and Solution B is added up to the mark "R." (4) The tube is now gently inverted several times to mix the solutions and then set aside in a cool place for 24 hrs. At the end of that time the protein in the milk has been deposited in the bottom of the tube and may be read off directly in percentages.

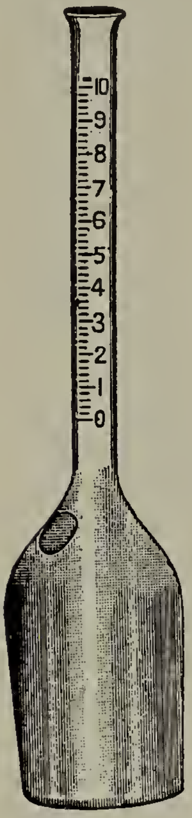

Fig. 18.—Babcock's Milk-test Bottle. (*D. H. Burrel and Co.*)

Conditions Altering the Character of the Milk.—Some of the abnormalities in composition already described (p. 99) depend upon various known causes, among which may be enumerated the following:

(*a*) **Period of Lactation.**—This exercises some influence upon the composition of the milk; yet, as a rule, not to any very considerable extent. The statistics of different investigators are not entirely in accord. The colostrum of the first few days, as we have seen, is rich in protein and in salts (p. 91). This is modified rapidly after the 3d day, and by the end of 2 weeks an average milk is obtained which varies but little, although individual milks may change considerably and irregularly from time to time. According to investigations made by Camerer and Söldner[2] (57 analyses), V. and J. S. Adriance[3] (120 analyses), and Schlossmann[4] (218 analyses), there occurs a slight but steady diminution in the percentage of protein beginning 2 to 4 weeks after child-birth, the loss by the 7th month of lactation being about 0.5 per cent. Toward the end of lactation there is a decided diminution in the amount of protein, the percentage of sugar slightly increases, while the mineral matter diminishes. The percentage of fat shows no regular change as lactation advances, unless it may be a slight decrease (Camerer and Söldner). Sharples and Darling[5] find no noteworthy alteration in any of the constituents which can be called characteristic of different periods of lactation, and much the same result was obtained by Holt, Courtney and

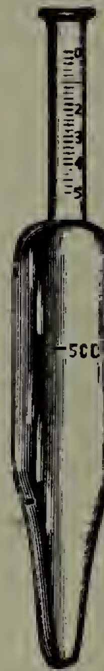

Fig. 19.—Flask for Bausch and Lomb Centrifuge.

For determining the fat-percentage of milk.

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[1] Johns Hopk. Hosp. Bull., 1906, XVII, 342

[2] Zeitschr. f. Biol., 1898, XXXVI, 277.

[3] Arch. of Pediat., 1897, XIV, 22.

[4] Arch. f. Kinderheilk., 1900, XXX, 288.

[5] Bost. Med. and Surg. Jour., 1903, CXLVIII, 416.

Fales.[1] This lack of material change is well illustrated in a case reported by Birk[2] in a wet-nurse, who at the end of 3 years less 1 month, had supplied 2167 liters (2288.35 quarts) of milk, with very little variation from the normal composition at the end of this period.

(*b*) **Intervals of Nursing and Time at the Breast.**—Peligot[3] had shown by experiments with asses' milk that the longer the fluid is in the udder; *i.e.*, the longer the interval between nursings; the more watery it becomes. The same is true of human milk. It is apparently especially the fat which is increased in amount by shortening the nursing intervals. There is a difference, too, in the milk from the breast at the beginning, in the middle, and at the end of nursing, again the change being chiefly in the fat. The analyses made by Mendes de Leon[4] and by Adriance[5] give figures illustrating the truth of this. Harrington's[6] analysis of cow's milk tends to confirm this difference for human milk since similar conditions would naturally exist.

Table 40.—Fat-content of Human Milk

	Mendes de Leon Fat, per cent.	Adriance Fat, per cent.
Full breast	2.62	2.27
Middle milk	4.06	2.79
Nearly empty breast	6.02	3.65

Table 41.—Fat-content of Cow's Milk (Harrington)

	Fat, per cent.	Total solids, per cent.	Water, per cent.	Ash, per cent.
Fore milk	3.88	13.34	86.66	0.85
Middle milk	6.74	15.40	84.60	0.31
Strippings	8.12	17.13	82.87	0.82

(*c*) **Number of Pregnancies and Age of Mother.**—According to Adriance[7] the milk of women between 20 and 30 years of age is slightly richer in fat and protein and slightly poorer in sugar than that of older women. The same is true of primiparæ as compared with multiparæ. The differences, however, are neither very material nor very constant, and may be disregarded provided only that the health of the mother is good.

(*d*) **Pregnancy.**—This generally diminishes the quantity of the milk secreted and affects its quality, the amount of fat especially being reduced, and the milk sometimes approaching the character of colostrum-milk. In other cases the milk remains abundant and of good quality, and the infant could safely be kept upon the breast; but this is not the rule, and on the mother's account the child should be weaned.

(*e*) **Menstruation.**—No certain alteration of the milk follows if menstruation reappears. In some cases the percentage of fat is diminished and that of the protein and of the sugar increased, or the general health of the infant may exceptionally be acutely disturbed. In most instances the character of the milk is entirely unaffected, or, perhaps, at most only during the days when the menses are present. Often the menses appear but once, and do not return until lactation is normally over. The greatest caution, therefore, should be exercised lest weaning be hasty and unnecessary. If acute symptoms develop the child could be fed artificially for the few days while menstruation lasts. An exhaustive study by Bamberg[1] led to the conclusion that there is no reason to believe that menstruation makes the milk harmful to the infant. (See also Causes making Nursing Inadvisable, p. 83.)

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[1] Amer. Jour. Dis. Child., 1915, X, 233.

[2] Monatsschr. f. Kinderheilk., 1923, XXV, 30.

[3] Hermann, Handb. d. Physiol., V, Th. 1, 404. Ref. Rotch, Keating's Cyclop. Dis. Childr., 1889, I, 289.

[4] Zeitschr. f. Biol., 1881, XVII, 501.

[5] *Loc. cit.*, 22.

[6] Rotch, Pediatrics, 1901, 133.

[7] *Loc. cit.*, 22.

(*f*) **Diet.**—Diet often exercises great influence upon the character of the milk secreted. This is, however, chiefly upon the protein and the fat, the sugar and salts being little affected. Insufficient nourishment of the mother produces milk deficient in total solids, especially the fat. The protein is either diminished or increased in quantity. It is also likely that a diet deficient in the necessary vitamins will produce a milk faulty in this respect also (see p. 195). An excessive amount of liquid taken often diminishes the total solids in all particulars, although in other cases the amount of milk is much increased without any diminution in the percentage of the solid ingredients. An abundant and rich diet, if well digested, given to women previously under-fed, increases both the fat and the protein and the quantity of milk. On the other hand, an over-abundant diet sometimes tends either merely to increase the adipose of the mother, or to injure the quality of the milk secreted and produce indigestion. It has been thought that the percentages of fat and of protein are increased by a highly nitrogenous diet, but diminished by a vegetable one, and that a diet rich in digestible fat also increases the fat in the milk. It is questionable, however, whether any such alteration in diet affects any of the milk-elements except in the case of women who have been previously long under-fed. It is possible that certain vegetables which possess a strong odor and taste may communicate these to the mother's milk, which may thus become unpleasant to or disagree with the child. It is likely, too, that some women exhibit idiosyncrasies toward certain articles of food, and that after they have partaken of them their milk may disagree with the baby. These are, however, all exceptions, and as a rule a healthy nursing woman may eat any digestible food without fear of any special influence of it upon the child.

(*g*) **Exercise.**—This, too, influences the character of the milk decidedly. Deficient exercise combined with a healthy appetite is liable to increase the percentages of protein and of fat; a proper amount of exercise to decrease an excess, if the woman has been too inactive; excessive exercise, with consequent fatigue, to increase the protein.

(*h*) **Mental and Nervous Influences.**—The effect of these is greater than perhaps from any other cause. It not infrequently happens that fright, anger, sorrow, or other great emotion will quickly so affect the milk that the infant is made ill by it. What the changes are is not certainly known. In some instances there is a sudden great diminution in the amount of the total solids, especially the fat, and increase in that of water. In others the protein is much increased in amount. In some cases there seem to be actual toxic changes produced in the milk. It sometimes even happens that the secretion of milk is quickly and permanently arrested by some emotional cause, or may have its quality affected and its amount much diminished by more prolonged nervous influences such as worry, repeated fatigue, and the like. The nervous mother is the one particularly liable to have a milk poor in fat and too rich in protein.

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[1] Zeitschr. f. Kinderheilk., Orig., 1913, VI, 424.

(*i*) **Illnesses.**—A good state of the general health is often a prerequisite to the secretion of good and abundant milk. In other cases the milk continues unaffected in spite of the mother being delicate and frail. This is often at the expense of the mother. Acute temporary ailments have but little effect upon the milk. In more severe febrile diseases the amount of milk may be much diminished and the percentage of fat decreased and that of the protein increased.

(*j*) **Drugs.**—These may be divided into those claimed to influence the secretion of milk and those which are excreted by the mammary glands with the milk. The former will be referred to again in considering the modification of breast-milk (see below). Of the latter it may be said that, contrary to the opinion among the laity, few drugs pass into the milk to any considerable extent, and the occurrence is so irregular and uncertain that efforts at medication of the child by way of the breast-milk would be unsatisfactory. The statements of investigators are largely contradictory. The following conclusions are based chiefly upon the publications of Fehling,[1] Houselot,[2] Engel,[3] and others.

Alcohol imbibed in very large amounts may appear only in traces in the milk. It may, however, taken in this way by the mother or wet-nurse produce serious illness in the infant. *Atropine, hyoscine* and *colchicine* pass directly into cow's milk in small amounts, and probably do so as well into human milk. *Opium* taken by the mother very rarely has any effect on the child. Yet, as cases are on record where dangerous action has occurred, it is best to administer it cautiously and in doses smaller than usual. *Iodine*, given to the mother in the form of iodides or applied as iodoform in a dressing, passes readily into the milk. *Bromine* does so to some extent (Rosenhaupt)[4] and occasionally in a way to produce a marked bromide eruption. Knowles[5] collected 5 such instances from medical literature, and others have been reported, as by Van der Bogert[6] and Boone.[7] *Mercury* has practically no effect. It passes to the child only in small amount and after prolonged administration to the mother. *Chloral* has, as a rule, no influence. Any effect appears only in weakly children who are nursed within an hour after the ingestion of the drug by the mother. *Arsenic, antimony, hexamethylenamine, antipyrine,* and *phenacetin* pass into the milk to a limited extent. In the form of arsphenamine arsenic appears to have a decided effect on the child in some instances. *Quinine* has little if any influence on the infant as a rule, and only when given to the mother on an empty stomach, and when her child is still in its 1st half-year. *Lead* and *iron* enter the milk to some extent. *Salicylic acid* in its different combinations affects it very decidedly. The *mineral and vegetable acids* have no effect upon it. The *saline purgatives* occasionally pass into it to some degree, while the *vege-*

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[1] Arch. f. Gynäk., 1886, XXVII, 331.

[2] Rev. prat. d'obstet. et de paediat., 1901, Nos. 1 and 2.

[3] Sommerfeld's Handb. d. Milchkunde, 1909, 810. Ref. Morse and Talbot, Diseases of Nutrition and Infant Feeding, 1915, 114.

[4] Arch. f. Kinderheilk., 1904, XL, 131.

[5] New York Med. Jour., 1909, LXXXIX, 586.

[6] Amer. Jour. Dis. Child., 1921, XXI, 167.

[7] Canad. Med. Assoc. Jour., 1922, XII, 570.

table purgatives, as senna, aloes, rhubarb, and cascara, as a rule do not (Gow).[1] *Chloroform* and *ether* appear to be without influence, with rare exceptions.

Modification of Breast-milk.—From what has been stated in previous sections it is evident that whereas the mother's milk may often be found insufficient or insuitable for the infant, it may sometimes be possible in various ways to modify the secretion in quantity or quality and thus to avoid the abandoning of nursing.

Quantity.—A threatened drying up of the secretion may often be prevented by seeing that the breast is thoroughly emptied at each nursing, giving both breasts if the milk is scanty. There is no better stimulant to increase secretion than this. When for any reason the infant does not nurse satisfactorily the milk should be expressed, lest the amount produced diminish. (See p. 79.) The taking of an abundant amount of liquid in some form is often an aid, with due caution that this does not merely increase the quantity while diminishing the total solids in a breast-milk already poor in quality. Milk and cocoa, however, have no advantage over weak tea or water, so far as quantity is concerned; and in the case of a woman already well-nourished, may merely result in making her unduly fat. Malt liquors undoubtedly have a decided influence in increasing the quantity in some cases and may be used for this purpose, with caution that no addiction to alcohol is established. (See also p. 81.) In all under-nourished women a generous diet should be given and over-work avoided. In women of a nervous temperament the life should be as quiet as possible, all exciting influences avoided, and rest at night be undisturbed, the care of the infant at this time being delegated to the nurse, and, if it seems best, the night-feedings being of artificial food or of milk previously expressed from the breast and kept on ice. With all nursing-women care should be taken that they spend a considerable part of each day quietly in the open air; driving, sitting, or taking easy walks. Among other methods which are recommended to increase the secretion are gentle daily massage of the breasts, faradization, and the administration of galactagogues. Regarding the last there is no certain evidence that any of these are of value. Experiments upon animals by some investigators appear to show such power in the administration of corpus luteum, placenta, the posterior lobe of the pituitary body, and the suprarenal glands, or in the auto-injection of human milk, but the results claimed have not been definitely established. Any benefit obtained would appear to be from a temporary stimulation, liable to be followed by a decrease. Pilocarpine, cotton-seed, anise, and other drugs have been tried, but there is little evidence in their favor.

Quality.—In cases where the milk is in general too rich, the condition can often be remedied by insisting upon a more active life with abundant exercise, and by reducing the amount of food taken. Lengthening the interval between the nursings and diminishing the total solids ingested may be of avail. The excess of *protein* so common in defective breast-milk in neurotic women can sometimes be remedied by increasing the amount of exercise up to a healthful feeling of fatigue, and by taking measures to relieve the nervous condition. If, however, the exercise goes beyond this point, the percentage of protein may be increased. This is the condition of the milk existing in the over-worked and under-fed women of the poorer classes. Here an increase of diet and of rest may bring the milk closer to the normal. Influencing the protein-percentage by the diet is, however, usually unsuccessful except in those women who are over-fed and idle; and in the starvation cases in which, as sometimes happens, all the elements of the milk are deficient in quantity, instead of the protein being high as is usually the case. An excessive percentage of *fat* can be lessened, with the protein, in the over-fed and idle women referred to, by reducing the total amount of food ingested and increasing the exercise. It is also reduced by lengthening the nursing intervals. When the amount of fat is deficient usually little can be accomplished except in the starvation cases, where an abundant diet and a diminution of work may increase the fat-percentage with that of the protein and the total secretion. Alteration of the diet has little effect on the secretion of fat in women already well-nourished and exercising sufficiently. (See also p. 104.) The amount of *sugar* in the milk cannot be influenced with any certainty. It is less variable than either the fat or the protein. In some cases the administration of sugar has increased the percentage (Lust),[1] in others not at all.

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[1] Practitioner, 1893, L, 168.

CHAPTER IV

ARTIFICIAL FEEDING IN THE FIRST YEAR

The nourishment of an infant with anything other than the secretion of the human breast is properly termed "artificial feeding," or "substitute feeding." The problem is often one of the most difficult which the physician encounters. The fact that we are dealing with a diet other than the natural one renders an absolute solution of it impossible, since, as far as our present knowledge extends, we are unable in any way to construct a food which is exactly like human milk. Moreover, we have to do always with the child as an individual, to whom general rules can apply only in a general way. Our inability to produce a food for infants which can be looked upon in any way as a standard must make us cautious in forming an arbitrary or hasty decision regarding the propriety or impropriety of a certain method of feeding in a given case, providing the infant is actually, and not only apparently, thriving upon its food. Nothing of this militates against the effort at an establishment of a scientific basis for the feeding of infants in general.

COW'S MILK

Of all foods the milk of some mammal is to be preferred as the basis, since it is in this class that the natural food of the infant belongs. That of various animals has at times been employed in different countries and for various theoretical reasons, the ass, goat, sheep, buffalo, sow, and mare having all been tried. These mammalian milks differ from human milk in various particulars, as is shown by the table on p. 108

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[1] Monatsschr. f. Kinderheilk., Orig., 1912, XI, 236.

TABLE 42.—COMPOSITION OF MAMMALIAN MILK
(Richmond)[1]

	Water, per cent.	Fat, per cent.	Sugar, per cent.	Casein, per cent.	Albumin, per cent.	Ash, per cent.
Cow	87.10	3.90	4.75	3.00	0.40	0.75
Goat	86.04	4.63	4.22	3.49	0.86	0.76
Ewe	79.46	8.63	4.28	5.23	1.45	0.97
Buffalo	82.63	7.61	4.72	3.54	0.60	0.90
Woman	88.2	3.3	6.8	1.0	0.5	0.2
Mare	89.80	1.17	6.89	1.84		0.30
Ass	90.12	1.26	6.50	1.32	0.34	0.46
Mule	91.50	1.59	4.80	1.64		0.38
Bitch	75.44	9.57	3.09	6.10	5.05	0.73
Cat	81.63	3.33	4.91	3.12	5.96	0.58
Rabbit	69.50	10.45	1.95	15.54		2.56
Llama	86.55	3.15	5.60	3.00	0.90	0.80
Camel	86.57	3.07	5.59	4.00		0.77
Elephant	67.85	19.57	8.84	3.09		0.65
Sow	84.04	4.55	3.13	7.23		1.05
Porpoise	41.11	48.50	1.33	11.19		0.57
Whale	48.67	43.67	7.11			0.46

For practical purposes the milk of the cow is the only one which can generally be made use of in most civilized countries, and any greater nearness in composition of other milks to human milk is of no special advantage, since all require *modification,* as by dilution or the addition of sugar, cream, water, etc., or in other ways, before they should be used.

GOAT'S MILK.—The milk of the goat may sometimes be advantageously employed when that of the cow is not obtainable. The analyses of Bosworth and Van Slyke[2] give a percentage-composition of fat, 3.80, sugar, 4.50 and protein, 3.10. It is therefore sufficiently similar to cow's milk to permit of it being modified in the same way. It is supposed to be superior in that the goat is comparatively free from tuberculosis. It has been claimed that the casein coagulates very firmly, but the studies of Bosworth and Giblin[3] showed no chemical difference between it and the casein of cow's milk. The milk acquires the unpleasant odor of the animal unless great precautions are taken. It is claimed also that it contains a greater percentage of iron than is present in cow's milk, and to be more like human milk in this respect (McLean),[4] and in the smallness of the fat-globules. It offers, however, no special advantage over the milk of the cow, except in the rare cases where there is an allergic susceptibility to the latter. It should be pointed out in this connection that severe anemia has repeatedly been reported as developing in infants fed upon goat's milk, especially in the first half-year of life. The cause appears to be unknown. Clearly it does not consist in a deficient amount of iron. (See extensive review by Brouwer;[1] also by Kirsch, Hoffer and Kirsch.)[2] With the frequency with which goat's milk has been employed in many countries, such an occurrence must be exceptional and avoidable (Blühdorn).[3] A very excellent résumé of our knowledge of the characteristics of goat's milk has been given by Calvin.[4] It is often difficult to obtain fresh goat's milk, and especially in a clean condition.

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[1] Dairy Chemistry, 1899, 323.
[2] Jour. Biol. Chem., 1916, XXIV, 187.
[3] Jour. Biol. Chem., 1918, XXXV, 115.
[4] Zeitschr. f. Kinderheilk., Orig., 1912, IV, 168.

Buffalo's Milk.—In some eastern and southern countries milk from cows cannot be obtained, and it has been proposed to substitute the milk of the water-buffalo. Analyses were made by Levine and Cadbury[5] and a study made of the method of employing the milk for infants. The analyses gave percentages of fat, 12.60, sugar, 3.70 and protein, 6.04. Noteworthy is the high percentage of protein and still higher of fat. These percentages can be reduced to those suitable for infants by diluting with 3 or more parts of water, and adding the required amount of sugar. Cadbury[6] reports good results with the milk in feeding infants in Southern China.

Characteristics of Cow's Milk.—The color of cow's milk is more yellowish-white than that of human milk. When absolutely fresh and obtained with proper care it possesses an indefinite, slightly sweetish taste and is practically without odor. The specific gravity ranges within normal limits from 1028 to 1033, with an average of 1032. The reaction as ordinarily obtained is faintly acid or amphoteric to litmus. With phenolphthalein it is always acid, and to a greater degree than human milk. The fat-globules vary greatly in size, the larger ones decidedly predominating. They are less numerous than in human milk and the emulsion consequently not so fine. Coagulation occurs readily by acid and by rennin, the resulting curd being firm and tough. When a certain degree of acidity is reached the milk will coagulate when heated. The nature of the process of coagulation by rennin has been much discussed. According to Bosworth and Van Slyke[7] the "casein" exists in milk in the form of a calcium caseinate. In the presence of rennin the molecule of this salt is broken up into two molecules of calcium paracaseinate. This being less soluble than the other, especially in the presence of a soluble calcium salt, it is precipitated and, with the fat, forms the curd. The addition of lime-water in sufficient quantity to milk forms a basic calcium caseinate which is not acted upon by rennin, the milk being alkaline and the coagulation not taking place by this ferment in an alkaline medium. When citrate of soda is added to milk coagulation is likewise delayed, or finally inhibited if sufficient is used, on account of the formation of a soluble calcium-sodium caseinate. Rennin transforms this into calcium-sodium paracaseinate which is soluble. The presence of acid favors the acid of rennin, perhaps by the formation of soluble calcium-salts from the insoluble calcium phosphate present in the milk. Bosworth[8] maintains that although the casein of cow's milk is essentially the same as that of human milk, in the latter it probably exists in the form of potassium-caseinate, which is not well coagulated as compared with the calcium caseinate of cow's milk. The smaller amount of casein present in human milk is also a factor.

[1] Jahrb. f. Kinderheilk., 1923, CII, 257 and CIII, 51.
[2] Wien. klin. Wochenschr., 1924, XXXVII, 568.
[3] Münch. med. Wochenschr., 1922, LXIX, 1220.
[4] Arch. of Pediat., 1921, XXXVIII, 584.
[5] China Med. Jour., 1918, XXXII, 536.
[6] Amer. Jour. Dis. Child., 1920, XIX, 38.
[7] Amer. Jour. Dis. Child., 1914, VII, 298; Journ. of Biol. Chem., 1913, XIV, 203.
[8] Amer. Jour. Dis. Child., 1921, XXII, 193; 613.

Effect of Heat on Milk.—Raising the milk to a temperature of 50°C. (122°F.) produces a slight skin upon the surface, which becomes greater if the milk be boiled. This is due largely to a partial coagulation and drying of the casein, while fat as well as calcium salts (Terry)[1] are contained in it in considerable amount. A temperature of 75°C. (167°F.) partially precipitates the lactalbumin, and prolonged boiling does this entirely. Heating up to or near the boiling point gives rise to the characteristic odor of boiled milk, partly by the production of a sulphur-compound from the lactalbumin, and a brownish tint is developed by the boiling if prolonged, dependent upon the caramelization of the sugar. The cream, too, rises imperfectly or not at all in milk which has been heated for half an hour to a temperature of 65°C. (149°F.) (Rosenau),[2] owing to the effect upon the emulsion of the fat globules; and the action of rennin is interfered with, the curd which forms being softer and more flocculent. The process of coagulation by rennin is even more disturbed if the milk has been boiled. Brennemann[3] has shown that boiled milk forms softer curds, even in the human stomach as well as in the test tube. The rising of the cream from milk is prevented to some extent even at a temperature of 145°F. (63°C.). Among other changes produced by heat are the driving off of the carbonic dioxide, oxygen, and nitrogen; a precipitation of the phosphates of the alkaline earths and of part of the citric acid;[1] a diminution in the organic phosphorus, and a destruction to some degree of the ferments, the alexins and the bactericidal properties of the milk. Few of these changes, however, occur except by a raising of the temperature to over 65°C. (149°F.) (Hippius);[4] and even the lactalbumin is not greatly affected at not over this temperature (Rupp).[5] A continued elevation to less than this degree consequently leaves the milk practically unchanged in most respects, with the exception of the interference with the rising of the cream, which is, as stated, unsatisfactory and uncertain. A large number of bacteria of different sorts are destroyed by a temperature of 65°C. or even 60°C. (149°F. or 140°F.), although the peptonizing bacteria of the spore-bearing class and many acid-producing germs are more resistant. (See Bacteria of Cow's Milk, p. 113.) The heating of milk for a brief time to the temperature of boiling or even higher has little if any effect on the fat-soluble vitamin A. The water-soluble vitamins B and C are more easily inhibited or destroyed by heat. (See Vitamins, p. 195.)

Effect of Freezing.—Many varying statements have been made as regards the effect of freezing upon milk. It would appear to produce a separation of the oil-globules, but other action is uncertain. Long-continued freezing has been claimed to have a very decided influence upon the chemical composition (Pennington, *et al.*).[6] Such freezing as may ordinarily be encountered in the household employment of milk does not usually have any harmful effect upon infants receiving it, although sometimes constipation, vomiting or diarrhea may be produced. (See Bacteria of Cow's Milk, p. 113.)

Caloric Value.—This naturally varies greatly with the differences in the composition of cow's milk. The range of figures is from 614 to

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[1] Arch. of Pediat., 1921, XXXVIII, 465.

[2] Hygienic Laboratory Bull., 1912, No. 56, 648.

[3] Arch. of Pediat., 1917, XXXIV, 81.

[4] Jahrb. f. Kinderheilk., 1905, LXI, 365.

[5] U. S. Dept. Agricult. Bull., 166. Ref., Grimmer, Monatsschr. f. Kinderheilk., Referat.,1912, XII, 662.

[6] Jour. Biol. Chem., 1908, IV, 353; 1913, XVI, 331.

724 calories per litre (581 to 685 cal. per qt.). Heubner[1] uses 690 to express the caloric value of a litre of milk (653 cal. per qt.).

Examination of Cow's Milk.—There is so little variation in the amount of protein, sugar and salts in mixed herd-milk that for practical purposes these may be taken as fixed amounts, and only tests made for the specific gravity, the reaction and the variation of the fat, as well as the presence of bacteria and adulterants. Variations in the specific gravity may indicate that the milk has been tampered with. The removal of cream from the milk increases the specific gravity and the addition of water lowers it. The reaction is tested by litmus paper in the ordinary way. Strongly alkaline milk has probably been adulterated.

The amount of fat may be roughly estimated by the use of the cream-gauge, but only if the test is commenced before cooling after milking has taken place. A larger cylindrical gauge should be used than for human milk, and the calculation is made on the basis that the percentage of cream which has risen after 6 to 8 hours divided by 3 will give the percentage of fat present in the milk (Richmond).[2] Cream rises more or less irregularly, and the best method for determining the fat is the use of the Babcock centrifuge, or the smaller Leffman-Beam glass as described under Human Milk (p. 101). In the testing of cream with this it is necessary to dilute 5 or more times.

If it is desired to make an approximate estimation of the protein present, the method of Boggs may be employed. (See p. 102.) When used with cow's milk the dilution of milk should be 1:20 instead of 1:10 as for breast-milk, and the reading in the tube must be multiplied by the figure 2.

In addition it is sometimes necessary to examine milk for the presence of preservatives, especially boric acid, salicylic acid and formaldehyde, and to see whether it has been heated. The methods are not difficult, but are better described in special contributions to this subject. (See Richmond,[3] Freeman,[4] Douglas,[5] Whitefield,[6] Winslow,[7] and others.) A microscopical examination is also required in many instances for the detection of pus.

Composition of Cow's Milk.—The milk of healthy cows may vary widely, depending upon peculiarities of the individual and of the breed, the character of the food, the period of lactation, the amount of milk which has been drawn from the udder, the time of day, and other factors. Even the mixed milk of a herd is subject to changes, and different breeds vary one from the other. The variation is particularly evident in the fat, that of the other ingredients, especially the sugar and mineral matter being but slight. As a result of the conditions mentioned the published analyses of the chemical composition of cow's milk vary considerably. The following table gives an approximate average analysis:

TABLE 43.—AVERAGE COMPOSITION OF COW'S MILK

Water	86.0–88.0 per cent.
Total Solids	12.0–14.0 per cent.
Fat	3.5– 4.0 per cent.
Sugar	4.5 per cent.
Protein	3.5– 4.0 per cent.
Salts	0.7 per cent.

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[1] Lehrb. d. Kinderheilk., 1911, I, 51.

[2] *Loc. cit.*, 103.

[3] *Loc. cit.*

[4] Med. Rec., 1899, Jan. 21.

[5] Lancet, 1903, July 4.

[6] Pediatrics, 1900, Jan. 1.

[7] The Production and Handling of Clean Milk, 1909.

Nature and Normal Variation of the Constituents of Cow's Milk. (See also Human Milk, p. 93, for fuller description of the differences.)

Fat.—The fat, as stated, is by far the most variable element. Percentages run in round numbers all the way from 2 to over 6 per cent. in different cows and from 2.5 to over 5 per cent. in different herds (König).[1] The herd-milk of Jersey and Guernsey cattle is from 1 to 1.5 per cent. richer in fat than is average cow's milk; while the surgar and protein are also somewhat in excess. Holsteins and Ayershires produce a milk slightly poorer than the average in fat (Lythgoe).[2] The amount of fat depends not only upon the breed, but upon the food given and the general treatment and health of the animal. It much resembles in composition the fat of human milk, olein and palmatin predominating; but the volatile fatty acids are relatively more abundant and the amount of oleic acid less than in human milk. The envelope of the fat-globules is similar to that of human milk.

Sugar.—The sugar may range in individual cows from 3.5 to 5.5 per cent. in round numbers, but in mixed herd-milk the variation is inconsiderable. The nature of the food has some influence upon it. The sugar is lactose and generally believed to be identical with or closely allied to that of human milk.

Protein.—The protein may range from 2.5 to 4.5 per cent. (König) in different healthy cows, but in mixed herd-milk is fairly uniform, the variation being not over 0.5 per cent. The percentage of 4, formerly commonly accepted, is generally not equalled. That of 3.50 more fairly represents the standard. The protein consists of casein, lactalbumin, and small amounts of lactoglobulin, as in human milk but in different proportions. The results obtained by investigators vary, Lehmann[3] giving the ratio of the casein to the lactalbumin (with lactoglobulin) as 10:1 (casein 3 per cent., albumin 0.3 per cent.); Richmond[4] as 7.5:1 (casein 3 per cent., albumin 0.4 per cent.); Schlossmann[5] as 6:1 (casein 3.19 per cent., albumin 0.53 per cent.); König[6] as 5.5:1 (casein 2.88 per cent., albumin 0.51 per cent.); Van Slyke[7] as about 4:1 (casein 2.48 per cent., albumin 0.66 per cent.) and White and Ladd[8] as 3:1 (casein 2.94 per cent., albumin 0.9 per cent.). The casein coagulates more readily by acids and by rennin than does that of human milk, very possibly differs chemically from it, and certainly does so biologically. (See pp. 95, 109 and 133.) One of the most important differences between human and cow's milk is in the amino-acid composition. It is probable that the amino-acids in human milk are in better relative amounts and proportions for the growth of the human infant than in cow's milk. Hijikata[9] found the amino-acids lysin, arginin, guanin, adenin, cholin and histidin in cow's milk.

Mineral Matter.—The mineral matter of cow's milk varies according to different analyses, and is influenced to some extent by the food. The analysis of Söldner is compared in the following table with that of Bunge for human milk, showing the contrasts of the relative percentages. The figures are those quoted by Hammarsten.[1]

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[1] Chemie der menschlichen Nahrungs-u. Genusmittel, 1903, I, 119; 130.
[2] Jour. Indust. Eng. Chem., 1914, Vl, 899.
[3] Arch. f. d. gesammte Physiol., 1894, LVI, 577.
[4] Dairy Chemistry, 1899, 120.
[5] Zeitschr. f. phys. Chem., 1896, XXII, 211.
[6] *Loc. cit.*, 153.
[7] Jour. Amer. Chem. Soc., 1893, 605.
[8] Phila. Med. Jour., 1901, Feb. 2.
[9] Jour. Biol. Chem., 1922, LI, 165.

Table 44.—Percentages of Mineral Matter of Cow's Milk and of Human Milk

	Cow's milk, Söldner	Human milk, Bunge
Potassium oxide	0.172	0.0703
Sodium oxide	0.051	0.0257
Calcium oxide	0.198	0.0343
Magnesium oxide	0.02	0.0065
Ferric oxide	0.00035[2]	0.0006
Phosphoric oxide	0.182	0.0469
Chlorine	0.098	0.0445

The mineral matter of cow's milk differs from that of human milk chiefly in the much larger total amount present, in the greater proportion of phosphorus, calcium, potassium, and magnesium, and the smaller quantity of iron. Part of the calcium is combined with the casein; the rest is united with phosphoric acid to form phosphates, which hold the casein in suspension in the milk. Copper is regularly present in cow's milk (Hess, Supplee and Bellis).[3]

It may well be that the difference in the salt-content of human milk and cow's milk, as shown in the table, may account in great part for the difficulty in the digestion of the latter. Phosphates and calcium caseinate act as buffers; *i.e.*, as substances which are capable of uniting with acid or alkali without undergoing a change in chemical reaction. As Marriott and Davidson[4] maintain, cow's milk by reason of its higher calcium and phosphorus content and its consequent higher buffer-value can neutralize a considerable portion of the acid of the gastric juice. This acid has important functions to perform, and any decided reduction in the amount of it may be associated with digestive disturbance.

Ferments, Extractive Matter, Etc.—Cow's milk differs further from human milk in the presence in it of a much larger percentage of citric acid (cow's milk 0.2 per cent.; human milk 0.05) and a much smaller amount of lecithin and of nucleone. The citric acid content of the milk is variable and is affected by the ration (Supplee and Bellis).[5] A number of ferments are to be found. Other characteristics of human milk are, however, absent from that of the cow. (See p. 97.) Raw cow's milk under certain conditions has a bactericidal action which causes a decrease in bacterial content on standing (Chambers).[6]

Vitamins.—Good cow's milk contains all the vitamins an infant requires. It is necessary, however, that the cow shall have been properly fed. This is particularly true of the water-soluble vitamin C, or antiscorbutic vitamin, which is destroyed to some extent by drying. Consequently cattle feeding on summer-pasture produce milk much richer in this vitamin than do those on the dry fodder of winter. (See p. 195.)

Bacteria in Cow's Milk.—All cow' milk, as ordinarily supplied, contains numerous bacteria. Even when first drawn from the udder this is the case, although it is true that by far the greatest number of bacteria are found in the first part of the milk, the last portion being nearly free. In addition to the germs within the udder, direct contamination of the milk occurs through the dust of the stable, dust from the cow's udder and belly, contaminating cow-manure, the hands of the milker, and, most especially, unclean utensils (Harding, Breed, Stocking and Hastings;[1] Prutcha, Weeter and Chambers[2]); and, inasmuch as milk forms one of the best of culture media, the multiplication of these is extremely rapid and the number of varieties great. Park and Holt[3] isolated and studied 239 species. The average market-milk contains from 2,000,000 to even 10,000,000 or more bacteria to the c.c. (Bergey);[4] a number often greater than is found in the sewage of our large cities (Rosenau).[5] The milk produced in dairies managed with sufficient care need never contain more than 10,000 to the c.c., while an average of from 2000 to 2500 is perfectly possible. The varieties which may be present are very numerous, depending upon the surrounding sources of contamination. According to Bergey those derived from the udder are forms of staphylococcus and streptococcus and the pseudodiphtheria bacillus. Those entering in other ways may be classified into *non-pathogenic* and *pathogenic.*

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[1] Phys. Chem., 1904, 455; 460.
[2] Bunge. Zeitschr. f. Biol., 1874, X, 309.
[3] Jour. Biol. Chem., 1923, LVII, 725.
[4] Amer. Jour. Dis. Child., 1923, XXVI, 542.
[5] Jour. Biol. Chem., 1921, XLVIII, 453.
[6] Jour. Bacteriol., 1920, V, 527.

Of the *non-pathogenic* the most frequent are the lactic acid-producing bacteria, developing lactic acid by acting upon the sugar. Prominent among these are the streptococcus lacticus, the bacillus lactis acidi and, less frequently, the bacillus lactis aërogenes. These germs enter the milk during milking or afterward, at the same time with others, some of which are often displaced by the production of an acidity which is unsuited to their growth. A second class is the butyric acid group, breaking up the sugar and fat and producing butyric acid. A third group is composed of the proteolytic bacteria which, usually after coagulating the milk, may cause a breaking up of the protein. Many of these are also capable of producing lactic acid, as for instance the bacillus coli, which is the one of this group most commonly present. The bacillus proteus, bacillus alkaligenes and certain other germs belong to the proteolytic class. Numerous other organisms may be present, among them pyogenic germs derived from a diseased udder, bacteria giving rise to various discolorations of the milk, or others which give it a bitter or other unpleasant taste. The germs of the lactic acid group do not themselves appear to be harmful, since the acid produced serves to check the growth within the intestine of the proteolytic bacteria. The aim, however, should be to have present as few germs of any kind as is possible.

A large variety of *pathogenic* germs has been found at times in milk, and epidemics have repeatedly been traced to this source. Kober[6] collected 330 outbreaks of infectious diseases traceable to the milk-supply, and the number has much increased since his publication. Infection through milk is true especially of typhoid fever, scarlet fever and diphtheria. Epidemics of septic sore throat have been produced in the same way. Tubercle bacilli of the bovine type may also be found in cow's milk if the udder is tuberculous, although they occasionally occur even when it is healthy, in this case the germs entering the milk after milking. The

[1] Univ. of Illinois Agric. Exper. Stat., 1917, Circular No. 205.
[2] Univ. of Illinois Agric. Exper. Stat., 1918, Bull. No. 204.
[3] Med. News, 1903, LXXXIII, 1066.
[4] Univ. of Penna. Med. Bull., 1904, July–August.
[5] Hygienic Lab. Bull., 1912, No. 56, 429.
Amer. Jour. Med. Sci., 1901, May, p. 552.

human type of tubercle bacillus is probably the more frequent in cow's milk, entering it from the dust of the air, the hands of a tuberculous milker, or in other ways. Among other pathogenic germs occasionally occurring in milk, and in some instances producing disease in infants and children, are those of cholera, dysentery, and anthrax.

The number of bacteria in cream is greater than in milk, even when this is obtained by centrifugating and with all precautions against contamination. Nevertheless the experience of the Milk-Commission of the Philadelphia Pediatric Society[1] has demonstrated that a standard of 25,000 germs to the c.c. of centrifugated cream is not too rigorous. When cream is obtained by the gravity process the number of bacteria is decidedly increased. In comparing the different layers of top milk, the greatest number of bacteria are found near the top.

By the heating of the milk many of the germs can be destroyed. Many of the forms of lactic-acid-producing bacteria are killed at a temperature of 60°C. (140°F.) as are most of the non-spore-bearing pathogenic germs. The tubercle bacillus is resistant, but will yield to a temperature of 155°F. (68.3°C.) continued for 30 minutes. Rosenau[2] found it no longer infectious when heated to 60°C. (140°F.) for 2 minutes. The butyric-acid bacilli and the spore-bearing proteolytic germs require a higher temperature, and even that of boiling does not destroy the spores. Consequently it has been believed that the temperatures usually employed for pasteurizing milk, which destroy the lactic acid bacteria, give a fuller opportunity for the subsequent growth of the dangerous proteolytic germs, because there was no later development of acidity to check their growth, and no warning of danger by the milk turning sour; and that especial care was necessary to prevent such a growth in milk which had been heated in this way. It has been shown, however, by Ayers and Johnson[3] that not all of the germs producing lactic acid are killed at a temperature less than 168°F. (75.5°C.), and that consequently there is no special opportunity given for the development of the proteolytic bacteria. In fact, the majority of both the acid-producing and the proteolytic germs are destroyed at this temperature, and the subsequent development of both sorts occurs in heated milk with the same readiness and with the same relationship as in raw milk. (See also pp. 119; 126.) It has been recommended by Budde[4] to add small amounts of hydrogen peroxide to the milk, and then to heat this to from 50 to 52°C. (122 to 125.6°F.), the object being to destroy the bacteria more completely. It is not certain that the process is as entirely harmless as claimed.

Freezing does not destroy the harmful bacteria. The lactic acid organisms do not grow at low temperatures whereas proteolytic bacteria do. Putrefaction may, therefore, take place even though curdling does not occur (Rosenau).[5]

Pus in Milk.—All cow's milk shows under the microscope a certain number of leucocytes. As a result of the study of a large number of cows, Bergey[6] and Stokes[7] concluded that the presence of more than 10 leucocytes per field of a 1/12 immersion lens, obtained by centrifuging 10 c.c. (0.338 fl.oz.) of milk, constitutes pus, especially when the cells are grouped in small masses and accompanied by chains of streptococci. There is, however, a very great range in the number of cells found in milk from healthy cattle, and the determination of the presence of pus by numbers alone is unsatisfactory (Lewis).[1]

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[1] Arch. of Pediat., 1904, XXI, April.

[2] Hyg. Lab. Bull., 1912, No. 56, 684.

[3] Bull. 126, Dept. of Agriculture, Bureau of Animal Industry.

[4] Milchzeitung, 1903, No. 44, 690. Ref. Kastle and Roberts, Hygienic Lab. Bull., 1912, No. 56, 385.

[5] The Milk Question, 1912, 293.

[6] Dept. of Agric. of Penna., Bulletin No. 125.

[7] Ann. Rep. Health Dept. of Baltimore, 1898. Ref. Bergey.

Cream.—Cream is to all intents merely a super-fatted milk, and, strictly speaking, any milk containing more than 4 per cent. of fat should be called cream. It is true that as the percentages of fat increase those of sugar and of mineral matter diminish slightly. Regarding the protein Adriance[2] and Richmond[3] claim that the percentage decreases as that of fat increases, and this is the generally accepted view. There is considerable discrepancy in the statements made regarding the degree of diminution in the percentages of protein and sugar with increasing strength of the cream in fat. The following table represents approximate averages based upon the figures given by a number of investigators:

TABLE 45.—PERCENTAGES OF FAT, SUGAR AND PROTEIN IN CREAMS OF DIFFERENT STRENGTHS

Per cent. cream	Fat	Carbohydrate	Protein
32	32	3.40	2.5
20	20	3.90	2.9
16	16	4.20	3.05
12	12	4.30	3.20
10	10	4.40	3.30
7	7	4.45	3.40

In the proportions to which the cream ordinarily employed must be diluted to obtain a sufficiently small amount of fat, the variation of the percentages of the other solids is immaterial and may be disregarded. Cream is obtained either by skimming or dipping, in which case it is called "gravity cream," or by the use of the "separator" and then called "centrifugated" or "separator" cream. Practically all the cream sold on the market is separator cream. By this process it may be made of almost any strength desired up to 30 per cent. or even 40 per cent. or more of fat by regulating the speed of the machine. The centrifugated cream has the great advantage that it can be prepared almost as soon as the milk is obtained from the cow and is consequently much freer from bacterial growth. A disadvantage claimed is that the centrifugal force breaks up the emulsion and is harmful to the infant. I have never witnessed any injury from this source, and the careful experiments of White and Ladd[4] indicate that no difference exists between mixtures made from gravity cream and those from centrifugated cream, and that the separation of visible globules of fat sometimes seen in bottles prepared for infant-feeding may occur with either and is due to other causes, especially the combined influence of heat and the shaking during transportation.

Top=milk.—This is the term applied to any number of the upper ounces of the milk in a milk-jar after having stood a number of hours. Strictly speaking top-milk is cream, of a strength varying with the number of ounces of milk which is removed from the jar. As, however, the laity persist in regarding as cream only the part which has separated with a distinct line of demarcation from the milk below, and as not infrequently it is desirable to remove more than this, the employment of the title "top-milk" is preferable. It designates the amount of milk removed, whether only 1 oz. or 31 oz., from the quart-jar of milk. Even with the precaution in the use of terms, and in spite of directions which seem explicit, mothers frequently persist in removing from the jar only the number of ounces which they require instead of the number ordered by the physician. It is manifest that the strength of the top-milk in fat is in inverse proportion to the number of ounces removed, and that a very decided difference in the milk-mixture results if, for instance, the top 2 oz. are taken instead of 2 oz. from the top 8 or more of the quart after removing all of this.

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[1] Amer. Jour. Dis. Child., 1913, VI, 225.
[2] Arch. of Pediat., 1900, May; 1904, Jan.
[3] Dairy Chemistry, 1899, 215.
[4] Phila. Med. Jour., 1901, VII, 218.

To obtain top-milk, a quart of milk, as soon after milking as possible, is strained into one of the ordinary quart milk-bottles, closed, cooled, and then kept on ice or in ice-water for 6 hours or longer. In dairy-milk delivered in jars the cream has already risen, and there is no need for the bottles to stand longer before removing the top-milk, if the cream-layer is sharply defined. To obtain the top-milk one may employ syphoning, pouring, or dipping. With the former a glass tube bent into the form of a syphon is filled with sterile water, the long end of this is kept closed with the finger, and the short end then inserted carefully into the jar until it reaches the bottom. The finger is now removed, the water allowed to flow out, and the milk which follows received in a graduated vessel, the "top milk" meanwhile sinking slowly toward the bottom of the jar. When the desired number of ounces remain in the jar, as determined by observing how many have escaped, the syphon is removed. Great care must always be taken that the jar, syphon, and other vessels are scrupulously clean. Sucking the water through the syphon by the mouth in order to start the flow is, of course, out of the question. A much better and easier method is the employment of the dipper devised by Chapin[1] and holding 1 oz. of milk[2] (Fig. 20). In employing it enough of the top-milk is first skimmed off with a teaspoon and placed in the dipper to allow using the instrument without causing the milk to overflow. As many ounces are then dipped off as are required. By letting down the dipper carefully and slowly into the milk the top layer is disturbed practically not at all. A modification of the dipper has a displaceable bottom which allows of the instrument being pushed into the jar without danger of causing an overflow. The original device is, however, preferable in many respects.

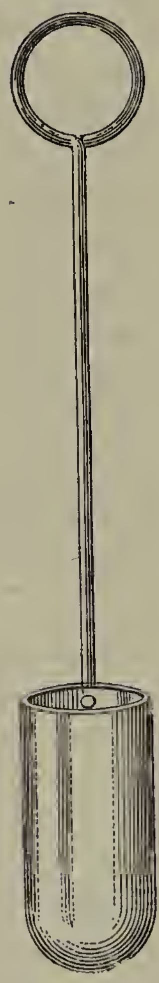
FIG. 20.—CHAPIN DIPPER.

Still another method consists in pouring off carefully the number of ounces of top-milk desired. This has been shown by Townsend[3] to give a top-milk with a percentage somewhat less than that obtained by the other methods. As it involves a different calculation it is not to be recommended.

The great advantage of top-milk is its economy and its convenience when separator-cream of known strength in fat is not obtained. Its disadvantages are that it contains a greater number of bacteria, and that the fat-percentages in it are subject to decided variation, investigators differing somewhat in opinion regarding this. The strength undoubtedly varies with different milks, those rich in fat, such as Jersey, giving a higher fat-percentage in a definite number of ounces of top-milk. Due allowance must be made for this. Thus, if a milk containing 5 per cent. of fat is employed, the removal of the upper 2 oz. of the quart after standing reduces the fat to 4 per cent. Further removal by dipping may now be done, taking the number of ounces which would have been used had the milk originally been of 4 per cent. fat-strength. That is to say, the "upper 8 oz.," for instance, would mean the upper 8 of the 30 oz. remaining in the jar after the top 2 oz. had been removed. The results of a series of unpublished examinations of the fat-strength of top-milk made by Dr. Chas. A. Fife, combined with a number of estimations published by others have led me to the adoption of the top-milk strengths which will be referred to later under Calculation of Milk Formulæ. (See p. 141.) These give results sufficiently accurate for practical purposes.

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[1] New York Med. Jour., 1899, LXX, 657.

[2] This dipper can be purchased through druggists, or from the Cereo Co., Tappan, N. Y.

[3] Bost. Med. and Surg. Jour., 1903, CXLVIII, 414.

Skimmed Milk, Bottom Milk, Fat-free Milk.—The milk remaining after any of the cream has been removed by skimming or by dipping off the cream-layer is denominated skimmed milk or bottom milk. Its percentages of sugar and of protein are slightly higher than, but for practical purposes the same as, those of whole milk. If 2 oz. of the top is removed, the fat-strength equals 3 per cent.; if all the milk below the cream-layer is used the fat-strength is about 1 per cent. If all but the lower quarter of the jar is taken away the percentage is less, frequently then being from 0.2 to 0.5 per cent., and the milk may be considered as fat-free. (See p. 141.) Actually fat-free milk, however, can be obtained only by the use of the separator. A comparison of the relative strengths of the different elements in whole milk, skimmed milk and fat-free milk, as given by Morse and Talbot,[1] may be seen in the following table:

TABLE 46.—COMPARISON OF WHOLE MILK, SKIMMED MILK AND FAT-FREE MILK

	Fat	Sugar	Protein
Whole milk	4.0	4.5	3.50
Skimmed milk	1.0	5.0	3.55
Fat-free milk	0.25	5.0	3.65

Requirements in Good Milk.—There are certain requisites which must be insisted upon in order to make cow's milk a safe and satisfactory food. First of all it must be clean. The centrifugating process applied to ordinary cow's milk obtained without sufficient care shows that this is far from being true of it. Cleanliness with regard to contamination by bacteria is important. There should be an absence of pathogenic germs and but a small number of those of other sorts. The milk should be tampered with in no way, as by the addition of such preservatives as boric acid and formaldehyde, the removal of any of the cream, or the addition of water. Much of that furnished is very decidedly over 24 hours old before it is used. If it has been pasteurized it should be distinctly stated on the bottle that this has been done. An unusually low bacterial count in milk not known to have been produced with especial care suggests the employment either of chemical preservatives or of heat.

[1] Diseases of Nutrition and Infant Feeding, 1920, 230.

The milk should be kept constantly at a temperature not over 45°F. (7.2°C.) from the time it is bottled until it is prepared for use, and the time of transportation should be as brief as possible. Uniformity in strength is necessary, with conformity to the analytical standards of normal milk. If the cow's milk varies materially it is manifestly impossible to modify it in a way which will agree with the child to whom it is fed. If it is from cows which are ill its quality becomes impaired, or it may become decidedly harmful to the infant. Consequently mixed herd-milk is to be preferred to that from a single cow, as less liable to exhibit sudden alterations in strength.

Dairy-Methods.—The best methods of dairy-management must be followed to ensure the fulfilling of these requirements for good milk. The cows should receive a carefully adjusted diet and be exercised daily. They should not be allowed to pasture at large, eating what they may find. They should not be fed immediately before milking on account of the dust produced thereby. The milk should be from a mixed herd, preferably of "grade" cattle. Jersey cattle produce a milk too rich in fat; Holstein and Ayreshire one rather poor (see p. 112). The stable must be well-ventilated, commodious, dry, screened, and kept strictly clean, and the cows so stalled that they cannot soil themselves or reach their udders with their heads. They should be cleaned and brushed daily, but not just before milking, and the hair upon the bellies, udders, inside of the thighs, and tails should be clipped short. The udders and teats should be washed with sterilized water before milking, and the milk-men should dress in washable sterilized clothing and caps, and wash the hands before the milking of each cow. Various mechanical milkers have been devised, but do not appear to have come into general use in the best dairies. Vessels of any kind into which milk enters must be kept bacteriologically clean. The milk-pails should be of special design intended to prevent the ready entrance of dust from the cow's belly or from the air of the room. Immediately after milking the milk should be rapidly cooled, strained, put in sterilized milk-jars and sealed. The cows should be in good health, and, to insure this, frequent veterinary examinations must be made, both of the animals and of their surroundings, and all those affected by any disease, especially by a suppurating affection of the udder or by tuberculosis, must be withdrawn promptly. Jersey cattle are claimed to be particularly prone to tuberculosis in this country, and all highly bred cows are, as a rule, of less vigorous health than "grade" cattle. Strict precautions, too, must be taken against the existence of any infectious disease among the employees of the dairy or their families, lest such become communicated to infants through the milk.

Certified Milk.—The evident lack in ordinary dairies of the fulfillment of any such regulations as those just mentioned, led to the establishment in various cities of Milk-Commissions, which in 1907 associated themselves as the American Association of Medical Milk-Commissions. The requirements detailed above constitute in most respects a partial abstract of the regulations adopted by the Association. The purpose of a milk-commission is to prescribe rules and regulations for dairies, with which a contract is to be made, and to see by inspection that the prescribed requirements and agreements are fulfilled. Personal systematic inspection of the dairy is made from time to time by members of the Commission, and repeated examinations of the buildings and the cows conducted by the Commission's veterinarian, and of the milk by its chemists and bacteriologists. To dairies the product of which fulfills the requirements a certificate is given to that effect, and such milk is properly called "certified milk." It is hardly necessary to say that much milk is advertised and sold by dealers as "certified" which in no way belongs in this category, the certifying being either a fiction or done by those employed by the dairy for this purpose.

Properly certified milk usually and rightly sells at an increased price, since the cost of its production is necessarily increased. With sufficient ordinary care, however, a great improvement in the quality of ordinary market-milk can be made without any material increase in the expense.

GENERAL PRINCIPLES OF SUBSTITUTE FEEDING

Comparison of Human and Bovine Milk.—Before proceeding to the study of the preparation of milk-mixtures, we may conveniently place before us in tabular form the contrasts between human and bovine milk which have already been considered. The figures are approximate ones:

Table 47.—Principal Contrasts of Average Bovine and Human Milk

	Cow's milk	Human milk
Specific gravity	1032	1030–31
Color	White.	Bluish-white.
Bacteria	Always present.	Practically none.
Coagulation by rennet and acid	Readily and produces firm curds.	Less easy and produces soft, flocculent precipitate.
Reaction to litmus	Generally acid.	Alkaline or amphoteric.
Fat	4 per cent.; containing more volatile fatty acids.	3.5–4 per cent.; containing less volatile and more oleic acid.
Sugar	4.5 per cent.	7 per cent.
Total Protein	3.5–4 per cent.	1–1.50 per cent.
Total Lactalbumin	0.3 per cent.[1]	0.5 per cent.[1]
Total Casein	3.0 per cent.[1]	1.2 per cent.[1]
Mineral matter	0.7 per cent.	0.2 per cent.
Total Solids	12–14 per cent.	12–13 per cent.
Water	86–88 per cent.	87–88 per cent.

The figures for lactalbumin and casein are those of Lehmann.[1] Reference to pp. 94 and 112 shows the variation among investigators as to the relative proportions of these two elements. All indicate, however, the relative increase in the proportion of lactalbumin in human milk as compared with cow's milk.

Modification of Milk.—The object in adapting cow's milk to infant-feeding is to change or *modify* it in some way, since without this change it is not tolerated by the majority of infants in the 1st year of life. A widespread misconception exists among the laity regarding this. By them "modified milk" is often spoken of as though it were some specific sort of food. As a matter of fact any element taken from or added to milk modifies it, even the mere addition of water. Modification is, therefore, a wide term indicating any change produced artificially in the milk. It becomes obvious from the study of the tabular composition that cow's milk in its natural state is far from suited to the healthy infant during the 1st year of life. Simple dilutions alter the proportions of all the solids uniformly, and are sufficient in some instances, while in others other methods are required. In brief the objects of modification are chiefly to prevent the development of bacteria or to reduce the number already present; to prevent the formation of the firm, large rennin curd; to alter the reaction; to reduce the protein, and sometimes to diminish especially the casein by the employment of whey; to reduce the salts; and to maintain or lessen the percentage of fat, some reduction being usually necessary on account of the large proportion of volatile fatty acids in cow's milk. Theoretically the accomplishment of such changes seems easy. Practically it is impossible, no matter what we do, to produce a perfect imitation of human milk. This should be recognized at the outset. The differences in the character of the fat cannot be satisfactorily altered by any modification; the biological characters of the casein are not the same; the ferments are different. So, too, although it would appear at first sight that the nearer the percentage of the food-elements corresponded with those of human milk, the better suited the food would be for the infant, experience has taught that although often desirable, it is far from being universally applicable; owing in part to the different ultimate constitution of the various elements, and in part to the individuality of the infant. No one formula can be expected to suit all healthy children; and the problem of adapting the requirements to the infant with digestive disturbance is many times greater.

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[1] Lehmann, Arch. f. d. gesammte Physiol., 1894, LVI, 577.

Percentage=feeding.—With the growth of a fuller knowledge of the composition of cow's milk came the expansion of the principles of what has been called "Percentage-feeding." The percentage-method was not in itself new. Biedert[1] emphasized its importance in a general way; and still earlier, in 1858, Cumming[2] gave a clear exposition of it. There had been, however, no special stress laid upon the importance of modifying as required the percentages of one or another ingredient of the milk-mixture, and no method devised for a ready accomplishing of these alterations, until the writings of Rotch[3] brought the matter into prominence. Percentage-feeding marked a great advance in the scientific feeding of infants. Physicians had for years prescribed modified cow's milk; *i.e.*, they had ordered a varying number of ounces of milk diluted with water, adding, it might be, cream and sugar, and changing these modifications to suit the case. The results, of course, were percentage-modifications of the cow's milk ingredients, yet what these were the prescriber usually did not know. The method was inexact because the *thought* was in terms of such composite articles as "milk" and "cream;" and to obtain accuracy in composition and in results was impossible in this way. In the percentage-method one thinks in definite percentages of "fat," "sugar," "protein," etc., determining how much of each the individual infant requires. The percentages thus chosen are then transformed into terms of the number of ounces of milk, water, cream, etc., required to be equivalent to these. (See p. 139 *et seq.*) The great advantages are increased accuracy of thought and the consequent better results to be expected. The problem, for instance, of how to increase the protein of a milk-mixture without increasing the fat can be solved only by thinking in percentages.

The percentage-method has suffered from much lack of comprehension and from misstatement. It has been looked upon as an innovation, considered complicated, and called by European writers the "American method." There is, in fact, nothing complicated about it, and any schoolboy who has studied the rules of proportion, possesses all the mathematical knowledge required. As to its newness, it is, as stated, not new. The only thing really new in it was the *method of thinking*, by which the food-mixture is built up synthetically from its elements to attain the desired chemical composition. It has nothing whatever to do with the choice of the food-elements, and is in no sense a *method of feeding*. It merely records with approximate accuracy the percentage amount of these elements employed, and enables a change readily to be made in them according to the requirements of the case. Much, too, has been written in recent years regarding the superiority of the so-called "simple dilution" of milk over percentage-feeding. This shows a total lack of comprehension of the subject. There can be no possible contrast or contradiction between them. Any physician is entitled to use whole milk diluted with water, and with the addition of sugar, in any amount the case requires, and in fact such dilutions are all that are needed in many instances. The important matter for scientific feeding is to realize the percentages of fat, sugar and protein which are obtained in this way; and such recognition makes the method a percentage one; and in any event the proper amount of sugar to be added has to be determined by a percentage-calculation. The fact is not to be forgotten, however, that the normal healthy baby needs and can digest fat; and that the invariable employment of diluted whole milk reduces this element below the infant's capacity and requirements, and forces the use of other elements in excess to make up the caloric deficiency. Without the knowledge of percentage-composition no scientific infant-feeding is possible. (See also pp. 40 and 128.)

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[1] Untersuch. ü. d. chem. Unterschiede d. Menschen- u. Kuhmilch, Inaug. Dissert., Giessen, 1869.

[2] Amer. Jour. Med. Sci., 1858, XXXVI, 25. Food for Babies, 1859.

[3] Brit. Med. Jour., 1902, Sept. 6, p. 653.

It is to be understood that the percentage-method is not supposed to give absolutely accurate statements of the amount of the various food-elements present. This is not, however, a matter of practical importance. It is not so much minute knowledge of the *initial composition* which is desired, as the ability to increase or decrease with comparative accuracy one or more of the ingredients at will, according to the necessities of the case and guided by the knowledge of the mutual relationships which the elements bear to each other. It is evident, too, that the employment of the "caloric method," to be described in the next section, is, in the nature of things, an impossibility without the knowledge of the percentage-composition of the milk-mixture.

The "Caloric Method" (See also p. 45).—This in its origin was simple. Heubner[1] as a result of careful experimental work upon a few healthy breast-fed infants, came to the conclusion that 100 calories per kilogram of body-weight of the breast-fed child should be rendered daily by the food in the first part of the 1st year, or 120 calories in the case of artificially fed children, with a gradual progressive diminution in number during the year. This figure expressive of the division of the total caloric need by the weight of the infant he called the "Energy-quotient." In avoirdupois it is expressed by 45 calories per pound of body-weight. Heubner then adopted a series of different dilutions, using ⅓, ½, or ⅔ of whole milk in water, and merely added a sufficient percentage of sugar and starchy material to make up the calories to the required normal amount. Various later investigators have confirmed his views, although with considerable difference of opinion as to the figures which most accurately express the energy-quotient; and various other calculations have been made permitting the use of other than these simple dilutions, yet based upon a calculation of the calories contained. The energy-quotient at present very commonly adopted for breast-fed and artificially fed infants is 100 calories per kilogram of body-weight for the first quarter of the 1st year, 90 for the second, 80 for the third, and 70 for the fourth. In avoirdupois weights these would be approximately equivalent to 45, 40, 35 and 30 calories, respectively, per pound of body-weight.

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[1] Zeitschr. f. diät. u. physikal. Therapie, 1901, V, 13.

The method is of great advantage in that it enables one promptly to determine whether or not a normal infant may be considered to be under-fed or over-fed as far as calories go. It has, however, distinct disadvantages, if followed too implicitly. Not only is there, as stated, a discrepancy in the definition of what actually constitutes the energy-quotient for normal infants, but for premature and emaciated infants the figure given by Heubner may be distinctly too low, inasmuch as it is based upon the *weight* of the child and not upon its *body-surface* or *body-bulk* as it should be. On the other hand, the activity of the child is to be considered; one exhibiting free movements of the body, or crying much, having a greatly increased heat-dissipation, and requiring, consequently, more calories; while a marantic infant whose movements are sluggish, feeble and infrequent, and whose life-forces are at a low ebb may be sufficiently nourished by food containing distinctly fewer calories than would have been expected. The difference, too, between the sleeping and the waking state is decided. We have no means of knowing, therefore, what the energy-quotient really is for infants other than those in health, and it is probable that the requirements vary with the child (Howland.)[1] The greatest disadvantage of the method, if followed strictly and not as its originator intended, is the tendency for the physician to take too little account of the relative assimilability of the different food-elements, but to regard it as a matter of indifference which is employed, so long as the food contains sufficient calories. Of course in actual practice no experienced pediatrist would act according to this; but the less experienced physician may readily be led into the mistake. It is evident that a food may contain the requisite number of calories and yet be constructed upon an entirely wrong basis. Protein, for instance, might have been left out completely, and the caloric requirements still be met; but on such a food life would be impossible; while it is becoming increasingly evident to most pediatrists that fat plays a role which cannot be successfully taken by carbohydrates. The method is, therefore, like that of percentages, not a *method of feeding*. It is only one of calculation, serviceable in estimating whether or not the food answers the infant's caloric needs. It aids in no way in determining how the food shall be composed in order that these needs shall be met.

If perfectly understood there can be no possible conflict between the two so-called "methods." The percentages of the elements of the food should first be selected according to the digestive capacity of the infant, and the caloric value of these then calculated to determine whether the energy-requirements are fulfilled.

With the accepted data that 1 gram of fat furnishes in the economy 9.3 calories; and 1 gram of protein and of carbohydrate each 4.1 calories, it is readily possible to estimate the total number of calories in a litre of any milk-mixture, if the percentage-composition is known. Thus supposing an infant is receiving a litre of a food-mixture containing fat, 3 per cent.; sugar, 6 per cent. and protein, 1.5 per cent: If we multiply 1000 grams by each of these figures we obtain as present in the food:

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[1] Amer. Jour. Dis. Child., 1914, V, 393.

Fat = 1000 × 0.03 = 30 grams
Sugar = 1000 × 0.06 = 60 grams
Protein = 1000 × 0.015 = 15 grams

If now we multiply each of these amounts by the number of calories which 1 gram produces, we obtain:

Fat = 30 × 9.3 = 279.0 calories
Sugar = 60 × 4.1 = 246.0 calories
Protein = 15 × 4.1 = 61.5 calories
Total in 1 litre of food = 586.5 calories

Since the infant requires in its food 100 calories per kilogram of its body-weight, we find that 586.5 calories divided by 100 = 5.865; *i.e.* the weight in kilograms of the infant which should thrive upon this quantity of food, so far as the caloric value is concerned.

When the infant is fed and weighed in terms of ounces, as is customary in English-speaking countries, a fourth calculation is required reducing the metric to avoirdupois system. As this is inconvenient, it is better to make the calculations from the beginning in English measures. A very simple formula has been devised by Fraley[1] which gives results quite sufficiently accurate for practical purposes. This formula in brief is

$$(2F + S + P) \times 1\tfrac{1}{4}Q = C$$

or, in detail, twice the fat-percentage, plus the sugar-percentage, plus the protein-percentage, multiplied by one and a quarter times the total quantity of food-mixture given in the day, equals the total number of calories furnished by the day's food. Supposing, for example, a milk-mixture contains fat, 3 per cent.; sugar, 6 per cent.; protein, 2 per cent.; 6 bottles of 6 oz. each administered; *i.e.*, 36 oz. in the 24 hours. Substituting these figures the formula will read $(2 \times 3 + 6 + 2) \times (1\tfrac{1}{4} \times 36) = 14 \times 45 = 630$ calories. Dividing this by 45, the number of calories required per pound of body-weight, we have resulting 15 pounds. That is to say, the formula should be sufficient for a child weighing 15 pounds. Further experience has shown that this formula gives a total caloric value which is a trifle low, and a more accurate result can be obtained by using the modification suggested by Holt and Howland.[2] In the form of an equation this reads: $(2F + S + P) \times 1.3$ = No. of calories in 1 fl. oz. of the food. The result thus obtained multiplied by the total number of ounces of food used during the day gives the caloric value of the day's food.

A knowledge of the caloric value of milk and milk-derivatives, as well as of various other articles of diet suitable for infants and children, is often of great value. A list of such equivalents will be found later (pp. 191 and 193).

Details for Milk-modification.—With the differences between cow's milk and human milk in view, as shown in the table already given (p. 120), we may take up the method to be employed to eliminate these as far as is necessary.

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[1] Arch. of Pediat., 1912, XXIX, 123.
[2] Diseases of Children, 1916, 181.

Altering the Reaction.—The attempt to accomplish this has ordinarily been made by the addition of lime-water. Just the amount of lime-water required is uncertain, but it is probable that from 25 to 50 per cent. of it must be added to accomplish any result. The action of lime-water or other alkali after its ingestion is not thoroughly understood, and it is questionable whether in the present state of our knowledge any alkaline addition is necessary or advisable. It is true that lime-water, if in this large amount, neutralizes the acidity of cow's milk, as also later of the hydrochloric acid secretion in the stomach, and delays or prevents the formation of large, tough protein curds (see p. 109); but, on the other hand, it may act as a buffer-substance (see p. 113) and be harmful by preventing the normal activity of the gastric secretion. The action of alkalies upon the time of emptying of the stomach is also still a disputed matter (see p. 35). It is evident, therefore, that our knowledge of the action of lime-water or other alkali is far from complete. It is certainly a mistaken idea that lime-water should be added in order to supply mineral matter for the proper development of the osseous tissues. This is entirely unnecessary.

Preventing the Formation of Firm Casein-coagula.—This is sometimes necessary, and is to be accomplished in various ways. The use of large amounts of alkali, as stated, aids in the matter. Citrate of soda has been much employed to prevent the coagulation, giving generally about 1 grain for every ounce of milk. Boiling the milk is another efficacious method (p. 110); or the total amount of casein in the mixture may be reduced, and the deficiency of protein made good by the giving of lactalbumin in the form of whey (see p. 151). The addition of a cereal diluent instead of water mechanically prevents the formation of tough coagula, acting, it is believed, as a protective colloid. From 0.60 to 1.20 per cent. of starch should be employed. (See p. 131.) Peptonizing the milk may be used in some cases (p. 153), or buttermilk given (p. 153), since the casein in this is not affected by rennet. In casein-milk also (p. 157) the casein is in a form in which large, tough curds cannot develop.

Altering the Proportions of the Milk-elements.—Should it be the intention to alter the composition of cow's milk to make it more closely resemble human milk—a procedure not always to be desired—the reduction of the percentage of the proteins is a simple matter if we treat these bodies as a unit. It is accomplished by merely adding to the milk some attenuant, generally water, in the proportion of 2 or 3 times its volume. If, however, we have regard to the composite nature of the protein-element, and wish to approach the composition of human milk more closely than simple dilution accomplishes, it is necessary to reduce the percentage of casein while leaving the lactalbumin unaffected. This can be done by the use of whey-mixtures, since the casein has been removed from them by the action of rennin. The employment of whey for this purpose will be described later (p. 151).

Disregarding the difference in the proteins, and with the intention of simulating human milk in other respects—a procedure, as stated, not always to be desired—it will be noticed in consulting the table on p. 120 that in diluting cow's milk with 2 or 3 parts of water the protein-percentage sought for is reached, and that the salts likewise are reduced sufficiently for all practical purposes. Simultaneously, however, the amount of fat will be made decidedly below the percentage present in human milk, and that of sugar still more so. The obvious remedy is to add sugar and fat to the mixture. The percentage of sugar is increased merely by adding enough to bring the figure up to the required amount. The percentage of fat may be maintained either by diluting a top-milk of such an initial strength that when the protein is sufficiently reduced the fat will still be of a higher percentage than it; or by replacing a certain amount of the whole milk used in the mixture by an equal quantity of a strong cream of known strength. The simple calculation necessary to reduce the percentage of protein, maintain that of the fat and increase that of the sugar, will be more fully discussed in considering Home-Modification. (See p. 136.)

Removal of Bacteria. Sterilizing. Pasteurizing.—Much better than the removing of bacteria is the prevention of their entrance. In the best milk, obtained by the method previously outlined (p. 119), often no treatment of any sort is necessary, the germs being so few in number that the infant can tolerate them well. With ordinary milk, however, and even with the best milk in the hottest summer weather, some procedure is needed. It must be remembered that no degree of heat can do more than partially destroy the harmful toxins which may have been formed already, and which are the cause of many illnesses. Milk which contains these cannot be made fit to use.

Sterilization.—The term *sterilization* as commonly applied to milk consists in the subjection of it for a short time, ½ to 1 hour, to a temperature of 212°F. (100°C.). This is not strictly a sterilization, since some varieties of bacteria remain, and spores of others are not destroyed at this temperature. The first practical "sterilizer" for household use in the feeding of infants was that devised by Soxhlet.[1] Since then many have been invented, that of Arnold being very well known, all acting on the same principle; viz. the subjecting of the milk in closed bottles to the action of steam, thus raising the food to a temperature of nearly or quite 212°F. (100°C.). The bottles, previously boiled, are filled with as much of the milk-mixture as is needed for each feeding; the necks carefully wiped and then stopped with sterilized, non-absorbent cotton, and the filled bottles stood in the rack intended to hold them and this then placed in the receptacle and subjected to the action of steam for an hour. After this the bottles should be cooled as rapidly as possible, and when cool enough not to break put upon ice and kept there.

The effect of heat upon milk has already been described (p. 110). Sterilizing at the temperature of 212°F. (100°C.) causes a certain degree of decomposition of the sugar; precipitates the lactalbumin; produces some alteration of the casein, and interferes with its coagulation by rennin; probably alters to some extent the mineral matter; destroys some of the ferments, and decidedly lessens the bactericidal power, which would have tended to prevent the growth of bacteria for a time. The use of sterilized milk may sometimes give rise to the development of infantile scurvy. Yet in spite of this list of apparently important alterations, the danger from sterilization is certainly over-rated. There is no positive proof that sterilized milk is, or is not, less well-digested than is raw milk, in spite of the large amount of evidence offered upon each side of the question. It is certainly better to raise the milk to the temperature of boiling than to employ it raw if not of the highest quality.

Pasteurization.—This term had its origin in the employment by Pasteur of a temperature less than 100°C. (212°F.) for the preservation of wine. As applied to infant-feeding it consists in the subjection of the bottles of milk-mixture, prepared as for sterilizing, to a temperature of less than boiling, one of from 140 to 165°F. (60° to 73.9°C.) being ordinarily recommended, and the heat maintained for from 30 to 60 minutes. This is sufficient to kill the pathogenic bacteria and the larger number of the non-pathogenic organisms. (See p. 115.) It will not, however, destroy the spores of the proteolytic bacteria. The effect upon the chemical composition of the milk is certainly less than is the heating to the boiling point, and at 140° or even at 150°F. (60° to 65.5°C.) is practically absent; but mechanically a difference is produced, shown especially in the imperfect rising of the cream (p. 110). Particular care must be taken to cool the milk rapidly afterward and to keep it cold, otherwise the protein is decomposed by the bacteria which may develop, and the milk, although it may not sour, is rendered especially dangerous (Bergey).[1] Pasteurization is a delicate process, since a temperature higher than 150°F. (65.5°C.) possesses some of the disadvantages of sterilizing, while one lower than 140°F. (60°C.) does not act sufficiently upon the germs. To be efficient it must be done accurately. All such makeshifts as putting the bottles into a dishpan of hot water, heating in a sterilizer for a certain number of minutes, and the like, are to be condemned, unless a careful observation is made of the actual degree of temperature attained, and the duration of this. A sterilizer may, it is true, be made use of, taking care not to raise the temperature to that of boiling. In this case, however, a thermometer must be employed and the temperature of the water constantly watched. This is a troublesome and time-consuming method and requires more knowledge than can be expected of an average nurse-maid and of many mothers. Various pasteurizers have been constructed. A very convenient and accurate one, easy to use, has been devised by Freeman[2] (Fig. 21). This consists of a metal pail with a groove about its circumference indicating the amount of water to be put into it, and a rack to hold the bottles, each bottle fitting into its own cylindrical metal cup, which, when water is poured around each bottle, prevents these from cracking when exposed to the heat. In the employment of the pasteurizer the pail is filled with water up to the grooved line, placed upon the stove or other source of heat, and the water raised to the boiling point, replacing any water which may have boiled away. Meanwhile the bottles are prepared as for sterilization and placed in the rack, and enough water poured into each cup to surround them. The milk in the bottles should be cold, and any bottles not needed for food should be filled with cold water and put in the rack. This is necessary to maintain the proper balance. The pail is now removed from the fire, set upon a wooden or other non-conducting table out of draughts, the rack put in, the lid put on, and the apparatus allowed to stand for 1 hour. In 10 minutes the temperature of the milk has risen to 55°C. (131°F.) and in 20 minutes from the beginning to slightly over 60°C. (140°F.), and this temperature is maintained for the remaining 40 minutes. The lid is now removed and cold water

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[1] Münch. med. Wochenschr., 1886, XXXIII, 276.

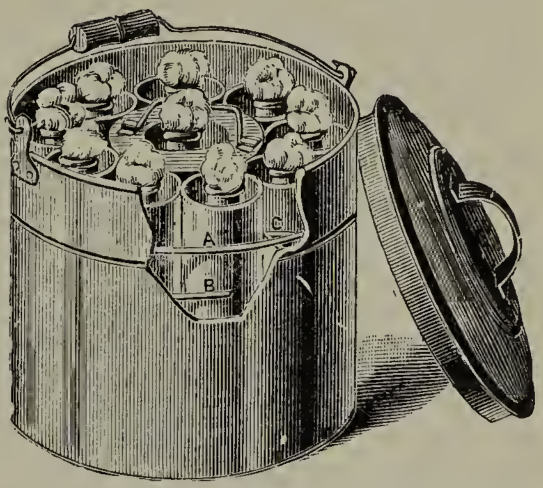

Fig. 21.—Freeman Pasteurizer.

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[1] Proc. Path. Soc. Phila., 1905, 102.

[2] Med. Rec., 1892, XLII, 8; Arch. of Pediat., 1896, 595; Jour. Amer. Med. Assoc., 1907, XLIX, 1740.

from the faucet is allowed to flow freely into the pail, overflowing into the sink. This produces the rapid cooling of the bottles which is necessary. When sufficiently cool they are at once put upon ice and kept there. The Freeman pasteurizer has the great recommendation that its employment is perfectly simple, and that it requires no watching whatever. Owing to the almost complete harmlessness of pasteurization, it should be employed with all milk not produced under the most careful supervision, and even with this it is advisable in hot weather. Any possible danger of the development of scurvy is readily prevented by the administration of orange-juice.

What has been written above applies entirely to *home-pasteurization,* which is the method always greatly to be preferred. In recent years *commercial pasteurization* has become very extensively used. Several procedures have been employed. The "flash" method passes the milk over hot pipes, heating it for a few seconds to the temperature of about 170°F. (76.7°C.). It is entirely untrustworthy. The "holding" method maintains the milk at 140 to 155°F. (60° to 68.3°C.) for 20 minutes or longer. This method is satisfactory if properly done; but in its commercial application it may easily fail of attaining the desired temperature. As shown by Schorer and Rosenau,[1] the safest commercial method is to pasteurize in sealed bottles, but at least 30 minutes should be allowed to raise the milk to 145°F. (62.8°C.), and this temperature maintained for 30 minutes more. Certainly any pasteurizing done commercially without strict supervision is liable to be dangerous. The dealer is tempted to employ the cheapest method which will retard the growth of the lactic-acid-producing organisms and prevent souring; while for the physician souring is the least of the evils which may occur, and may be a valuable index of the care which has been taken of the milk. The proteolytic germs are far more dangerous to the infant. Further, the purchasing of pasteurized milk by the mother, independently of medical advice, gives her a false sense of security and engenders lack of sufficient care of the milk after it has been delivered. As a matter of fact, all pasteurized milk requires the same care in keeping it cold that applies to the handling of raw milk. Perhaps the greatest disadvantage of commercially pasteurized milk delivered in the bottle as whole milk, is the unsatisfactory manner, already referred to, in which the cream rises. This makes the employment of either top-milk or skimmed milk subject to great uncertainty. The fat in the former is less than it should be, and that in the latter often so great that in cases of disturbed digestion a sufficiently low fat-percentage cannot be obtained.

The Action of the Different Food-elements.—Although a cow's milk-mixture which contains percentages similar to those of human milk, and which possesses the requisite number of calories, theoretically should agree with and properly nourish the human infant, yet, as already pointed out (p. 121) the insurmountable differences between the two varieties of milk make this not at all the necessary result. As a matter of fact, many variations from the standard are often required. It is important to understand the action of the different milk-elements in health and in the production of symptoms of indigestion, and, with this knowledge, to change one or another until the suitable food is obtained. This action has already been studied to some extent in discussing the absorption and metabolism of breast-milk (pp. 40 and 45), and the action of the different elements of breast-milk (p. 98). It is rare, in my experience, that a healthy infant cannot be fed with very fair success upon cow's milk, if it has been properly started. The problem arises in the case of those who have already been improperly fed. Occasionally, it is true, an idiosyncrasy to cow's milk is encountered, and such infants cannot take it at all. This condition is generally an acquired one, dependent probably upon an allergy to cow's milk-protein developed through faulty efforts to feed this during a period of indigestion in very early infancy. The action of the ferments may be ignored, as we know comparatively little of the actual part played by them in the diet. The mutual influence which the different elements of the food exert upon each other is, however, a matter of importance and is receiving increasing attention.

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[1] Jour. Med. Research, 1912, XXVI, 127; Amer. Jour. Dis. Child., 1912, III, 226.

Water.—The importance of sufficient water in the food has already been referred to in discussing human milk (p. 98). It seems entirely reasonable that the effort should be made to preserve a large percentage of water in the food of artificially fed infants. Under certain circumstances in disease it is better that the amount be reduced; and, as a matter of fact, it has been claimed by Helmreich and Schick[1] and others that even healthy infants do better on foods in which the percentage of water is diminished to one-half the usual amount, and that the ordinary quantities of water given are a tax upon the skin and the kidneys. The question evidently requires further study, but the advantage of concentrated food in under-weight infants, who are taking nourishment poorly, seems well borne out by the experience of Lust[2] and others. (See also Concentrated Feeding, p. 149.)

Fat.—The fat of the milk-mixture is the element most easily causing digestive and nutritional disturbances, and many infants show a great and persistent intolerance for it. While in theory 4 per cent. should be well-borne, this being the amount existing in human milk, it is usually necessary, in my experience, to use decidedly less than this in making a milk-mixture, owing to the excess of volatile fatty acids present in cow's milk. This is particularly the case in very young healthy infants, and in others older whose digestion has been impaired by illness. For all such an initial percentage of 0.5 or 1.0 per cent. is to be selected, and the increase above this made very carefully. Even with those who have shown a power to digest cow's milk-fat, 3 per cent. or at most 3.5 per cent. is generally as high as it is safe to go. (See Lactic-acid Milk, p. 155.) This is especially true in home-modifications if portions of top-milk and bottom-milk have been combined, since in the latter there is a certain amount of fat present which may increase the percentage slightly above that indicated by the calculation. Fat is indeed the commonest cause of frequent sour vomiting with the rancid odor of butyric acid, although an excess of sugar may likewise produce sour, watery regurgitation. So, also, if the fat is in too large an amount or not well digested it combines with the heavier alkalies, as calcium and magnesium, and produces soap-stools; or, uniting with potassium or sodium, causes diarrhea with white soft curds, and in this way an undue amount of fat may be lost. An increase of the carbohydrates will sometimes cause the fat to be absorbed. Where there is an unusual intolerance for the fat of cow's milk, this element must be withdrawn or reduced in amount. For this purpose butter-milk (p. 153) is suitable. Efforts have been made to use other kinds of fat, especially olive oil (Ladd).[3] It is necessary, however, to have this made into an extremely fine emulsion by a special process. Even lard and cocoanut oil have been tried (Gerstenberger, *et al.*;[1] see p. 171).

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[1] Zeitschr. f. Kinderheilk., 1921, XXX, 121; 147.
[2] Arch. f. Kinderheilk., 1922, LXXII, 72.
[3] Transac. Amer. Pediat. Soc., 1915, XXVII, 117.

A very common practice with many physicians is the giving of a food-mixture rich in cream for the sake of overcoming constipation. This is a dangerous procedure in infants in the 1st year; and there are better ways. On the other hand, it is to be remembered that fat produces more than twice as many calories as do the other milk-elements, and that without a sufficient amount of it the quantity of food necessary to supply these may be unduly great; further that without the fat there may be a diminished absorption of calcium. Infants who are not able to take a proper amount of fat are liable to be insufficiently nourished.

Carbohydrate.—The sugar of the milk is generally digested well. It is seldom necessary to begin with less than 5 per cent., and under ordinary circumstances it is not required to increase beyond 6 or 7 per cent. Much discussion has arisen as to the variety of sugar which should be employed. Usually it would seem best to select lactose for this purpose. This is the natural sugar of the food, animal in its origin, and by its slower absorption tends to remain longer in the intestine, favoring the growth of the normal intestinal flora. It also undergoes butyric acid fermentation less readily than does maltose, and is better tolerated than is this or cane-sugar when there is disordered gastric digestion with vomiting. On the other hand, under the influence of abnormal bacterial growth lactose may become more harmful than other sugars. Where a rapid absorption and increase of weight is desired, saccharose and, especially, maltose-dextrin preparations are to be preferred and can often be taken in large amounts (see p. 42) by healthy infants. They are serviceable in this way when, on account of difficulty in the digestion of fat, it is necessary to supply the requisite calories by giving a high carbohydrate diet.

The carbohydrate in the food tends to favor the digestion of the protein, and when administered in high percentage will often change completely the character of stools showing protein-indigestion. It often favors, too, the absorption of the fat; perhaps by increasing the acidity of the intestinal tract and thus preventing the union of the fatty acids with the alkalies to form soap-stools. If, however, given in excess, any form of sugar may cause diarrhea, and a consequent diminished absorption of the food-elements. Not more than 7 per cent. of lactose should be administered to the healthy infant (Morse and Talbot),[2] and when larger percentages of carbohydrate are required, maltose-dextrin preparations, saccharose, or starch is better.

The term "maltose" as used in the feeding of infants is generally employed to designate one of the combinations of dextrin and maltose on the market. There are a number well known, all consisting principally of mixtures of maltose and dextrin, produced in the dextrinizing of starch. (See pp. 131; 165.) The percentage of maltose ranges roughly from 40 to 85 per cent. and of dextrin from 10 to 60 per cent. The preparations containing the more maltose are more rapidly absorbed, but, on the other hand, are more liable to produce diarrhoea, and those with considerable dextrin are to be preferred. The following table of analyses of some of them is derived from the advertisements of the manufacturers.

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[1] Transac. Amer. Pediat. Soc., 1915, XXVII, 94.

[2] Diseases of Nutrition and Infant Feeding, 1915, 264.

Table 48.—Preparations of Maltose and Dextrin

	Water, per cent.	Fat, per cent.	Protein per cent.	Maltose, per cent.	Dextrin, per cent.	Starch, per cent.	Ash, per cent.	Mineral matter added, per cent.
Borcherdt's malt sugar	1.95	...	4.4	87.4	4.35	.....	1.9	
Loeflund's food maltose	.....	...	.....	40.0	59.7	.....	0.3	
Maltzymose	22.1	...	1.61	67.2	6.95	.....	2.0	
Mead's dextri-maltose No. 1	5.0	...	.....	51.0	42.0	.....	.....	2.0 sodium chloride
Mead's dextri-maltose No. 2	5.0	...	.....	52.0	42.0	.....	.....	
Mead's dextri-maltose No. 3	5.0	...	.....	51.0	42.0	.....	.....	3.0 potassium bicarb.
Borcherdt's malt soup extract	21.79	...	6.4	57.57	11.7	.....	2.54	1.1 potassium carbon.
Borcherdt's dri-malt soup extract	3.8	...	8.66	71.1	13.5	.....	2.94	1.1 potassium carbon.
Borcherdt's dri-malt soup extract with wheat flour	2.3	...	8.2	46.8	9.4	31.1	2.2	
Loeflund's malt soup stock	22.3	...	3.73	54.26	17.6	.....	2.11	
Maltine Company's malt soup extract	29.9	...	3.42	62.3	3.0	.....	1.36	1.1 potassium carb.
Mead's dry malt soup stock with wheat flour	5.0	...	.....	47.0		47.0	.....	1.0 potassium bicarb.

To this table might well be added Karo corn-syrup. The red-label variety is said to contain 23.5 per cent. water, 33.2 per cent. dextrose, 5.0 per cent. sucrose, 37.75 per cent. dextrin, and 0.55 per cent. mineral matter.

Many disordered conditions of the digestion have been attributed to the influence of the sugar of the food, and without question with more or less reason. Yet a careful review of the subject by Porter and Dunn,[1] combined with their own experiments, led to the conclusion that the danger of severe symptoms from the giving of the sugars in considerable amount has been greatly exaggerated, and recent knowledge bears this out.

Starch.—It very frequently is of great advantage to employ the addition of starch in the food-mixture, and even in the form of a thick gruel it has been advantageously employed in some forms of vomiting in early infancy. Its caloric value is practically the same as that of sugar. Although the pancreatic secretion possesses amylolytic power even at birth, this is only slight before the age of 2 or 3 months, and when starch is given before this age it is for purposes other than absorption; such, for instance, as the prevention of the formation of curds. In the form of barley-water or other amylaceous decoctions it is often of great service. There is no reason, however, to give starch to every infant as a routine measure. In fact it should be used only for a definite purpose and in definite percentage-strength. Generally 1 per cent. at most is sufficient for simple dilutions. A table of the strength of various cereal decoctions will be given later (p. 164).

Starch is an extremely useful carbohydrate in those cases where a high percentage of this latter is required, being often better tolerated when combined with a dextrin-maltose preparation than is the latter when given alone. It is the existence of this combination which makes the malt-soup preparations so serviceable in many instances (p. 166). This is probably in part due to the mechanical colloidal action of the starch upon the protein, and in part dependent upon the slower breaking up of the starch-molecule as compared with sugar and dextrin. If, however, starch is given continuously in excessive amounts, and especially if without the addition of milk, or with only small amounts of it, severe symptoms may arise. The infant becomes pale, flabby and perhaps too plump; indigestion with diarrhea develops; there is undue fermentation in the alimentary tract, with loss of power of digestion and the development of a wasted condition, or sometimes of increase of weight dependent in part upon an extensive edema. (See Injury from Starch, p. 727.)

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[1] Amer. Jour. Dis. Child., 1915, X, 77.

Protein.—Other things being equal, the protein of milk is tolerated in relatively high amount, and seldom produces disturbances of digestion. Yet it is better to begin with not over 1 per cent. in the mixture, and then gradually to increase this as the needs demand. The quantity required by the infant is not very large, although greater relatively than in the adult. The average minimum amount is 1.5 grams per kilogram of body-weight (10.5 grains per pound), and many infants need slightly more than this (p. 43). The great difference between the protein and the other elements is that whereas these may replace each other to some extent this amount of protein is absolutely required, and can be substituted by nothing else. Infants fed upon a diet insufficient in protein cannot really thrive. Although they may often be quite fat, perhaps from the large amount of carbohydrate which has been administered, they are anemic, flabby and possess little actual strength or muscular development. As regards the different proteins of the milk, it has been claimed in many quarters that the casein is always tolerated in high percentage provided that of the whey is reduced; the benefit from the reduction depending probably upon the diminution in the amounts of sugar and of salts rather than of the lactalbumin. This requires further confirmation, and there is decided evidence opposed to it. It would seem probable, for instance, that the improvement in symptoms undoubtedly sometimes seen after the employment of peptonizing, once so much in vogue, would indicate that the percentage of casein was not the matter of indifference which has been maintained. There is, further, no question from the experience of many physicians that many ill infants who have done badly upon food containing considerable casein will thrive on whey-mixtures. This would seem reasonably to be expected when one considers the comparatively large amount of whey-protein in human milk as compared with the casein. (See Table 47, p. 120.) For many years whey-cream mixtures were popular with physicians, and deservedly so in selected cases. In fact, it has been pointed out (Holt)[1] that sometimes the feeding with high casein-percentages may not be tolerated unless whey, with its comparatively high carbohydrate content is present also; and that, further, the whey is needed on account of the large percentage of certain amino-acids present in it. Sometimes the whey in the mixture is well borne if the fat-percentage is low, as is the case when buttermilk is employed.

Sometimes the protein matter of milk is found in the stools as hard, yellowish-white curds (see p. 43), but this appears to be of little moment unless the curds are very numerous, and seems to be prevented if the milk has been heated to a high temperature, or by other measures already discussed (p. 125). Failure to digest protein also often results in colic or in the development of offensive, green, diarrheal stools, or of soap-stools with an offensive odor.

Much has been written concerning the digestibility and absorption of foreign proteins and the deleterious effects which may or may not occur. The subject has already been alluded to. (See p. 43 and the section on Allergy, p. 750.) There seems to be no question that such results can follow. In the cases described by Benjamin[1] there was pallor, great atony, free perspiration and sometimes eczema, and Glanzmann[2] and Moll and Stransky[3] have emphasized the damage which may be produced by protein, caused by a long-continued use of too large amounts of casein-milk. In an instance reported by Hoobler[4] a decidedly stuporous condition developed, whereas in cases recorded by Holt and Levene[5] there was continued fever. Tanaka[6] found that although with superalimentation with casein the protein was absorbed, yet there soon develops an increased lack of retention of nitrogen. The influence of foreign proteins in the food even when in small amount may sometimes show itself in other ways. Thus, for instance, in the early weeks of life, and especially in infants suffering from any gastro-intestinal or nutritional disorder, it is possible for the foreign protein to pass unaltered or partly digested through the intestinal wall and to produce modified evidences of allergy. In some instances this amounts to an actual intolerance, and certain foreign proteins, even in very small quantity, cause decided and at times alarming symptoms, even in apparently healthy infants and in older children. This may occur with the casein of cow's milk, but an actual allergic condition of this nature produced by it is certainly uncommon. Even a protein which cannot be classed as foreign may rarely produce similar symptoms, as, for instance, in the cases where an intolerance for human milk has been observed. Oftener protein-intolerance is seen in older children, and is especially noticed with that of egg or of certain of the cereals, varying with the case. Very similar symptoms can sometimes be occasioned by other food-stuffs, such as certain fruits. The symptoms consist not only in severe gastro-intestinal manifestations, but in nervous disturbances including prostration, cutaneous eruptions, dyspnea, sneezing, wheezing respiration, and other symptoms suggestive of an asthmatic attack. Cutaneous tests upon children have been made with the various proteins by Schloss,[7] Talbot,[8] and others, which confirm the clinical experience that it is the protein which is the active cause of the disturbance.

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[1] Arch. of Pediat., 1916, XXXIII, 13.

Mineral-matter.—The salts are certainly necessary for the life of the infant, and are absorbed and retained in proportion to the retention of nitrogen. With the exception of iron they are present in cow's milk to an excess which renders them sufficient for the infant given any ordinary dilution. It has been claimed that they are not always well-tolerated, and that the reduction of the salts and of the sugar, as results from the removal or reduction of the whey, is of service in some cases of difficult digestion. The whole question of the part played by the salts in digestion is, however, far from being understood. (See also p. 44.)

The Selection of Percentages.—What follows may be regarded in the light of a summary of the substance of the preceding sections. At the outset it must be clearly stated that it is impossible to formulate any fixed rules for the percentage-strength of the milk-ingredients which even the normal child will require. The tables often published showing the strengths to be used at different ages are liable to be misleading and harmful, in that they tend to make the inexperienced practitioner attempt

[1] Zeitschr. f. Kinderheilk., Orig., 1914, X, 185.
[2] Jahrb. f. Kinderheilk., 1915, LXXII, 251.
[3] Jahrb. f. Kinderheilk., 1922, C, 3.
[4] Amer. Jour. Dis. Child., 1915, X, 153.
[5] Amer. Jour. Dis. Child., 1912, IV, 266.
[6] Jahrb. f. Kinderheilk., 1918, LXXXVIII, 161.
[7] Amer. Jour. Dis. Child., 1912, III, 341.
[8] Bost. Med. and Surg. Jour., 1918, CLXXIX, 285.

to fit the baby to the table, rather than the food to the baby. The fact has already been pointed out that after the early weeks the proportions of the ingredients of human milk do not alter materially. The infant simply takes a larger quantity as it grows older. In artificial feeding, on the other hand, owing to the difficulty in the digestion of the fat and the differences in composition between the proteins of cow's milk and of human milk, the percentage of these latter in the mixture usually must be raised, as otherwise the bulk of the food required to supply the protein needed might be far too great if properly diluted. The only dependable guide is the state of the general health of the infant, the condition of its digestion, and the rate of growth in weight. To make the age by months the guide is, I believe, a serious error. Should the health and weight be all that can be desired, increase in the strength of the food in any respect is to be made cautiously if at all. If the condition indicates an insufficient food-supply, the quantity is to be increased or the percentages raised. If digestive disturbances arise, the food must be further modified. There are naturally certain percentages which experience has taught that the average child can be expected to take at certain ages; but the feeding of each individual infant is a matter of trial, since no one can know in advance what the tolerance for cow's milk will prove to be.

With these limitations the following directions may be given as a general guide: The healthy infant in the first few weeks of life may be started with such a formula as fat 1 per cent., milk-sugar 5 per cent., and protein 1 per cent. Very probably there will be no gain in weight, or even a slight loss with this weak mixture, and it is likely that a stronger initial one could have been used. It is safer, however, to begin with the low formula in order to test the effects upon digestion. There is no hurry about the gain in weight. To accustom the infant to its new food and not to disturb the digestion are the important matters. If the food agrees well it may be changed in a few days to fat 1.5 per cent., and then again to fat 2 per cent., sugar 6 per cent., and protein 1.5 per cent. These changes, of course, go hand in hand with an increase in the quantity, which will be considered in the next section. With such a formula as the last given, the infant may be quite contented and thrive well for a considerable time. Should, however, the digestion be in perfect order but the infant hungry and the weight at a standstill, the strength must be increased to fat 2.5 or 3 per cent., sugar 6 or 7 per cent., and protein 1.5 or 2 per cent. It is manifest that hunger may be satisfied and gain in weight accomplished by giving an unduly large quantity of a weak formula, but the policy is not a good one. After the age of 6 months it is generally necessary to make a further increase in the strength of the food, giving perhaps fat 3 or 3.5 per cent., sugar 5 or 6 per cent., and protein 2 or 3 per cent. The frequent difficulty in digesting fat should still be borne in mind, and in most cases an amount over 3 per cent. should be tried with great caution. Many infants even at a year of age cannot take entirely undiluted cow's milk. What changes shall be made in the food, what elements increased or decreased in amount, is to be determined by a study of the effects, as outlined in the preceding section (p. 128). The method of preparing the food-mixture containing the percentages desired will be considered in the sections upon Laboratory-modification (p. 135) and Home-modification (p. 136) of the milk.

The Quantity of Food and the Frequency of Feeding.—The quantity of the milk-mixture to be given varies, of course, with the age, and approximates the table for the amount of breast-milk as given on p. 90. The frequency of feeding, too; the hours for this; and the relation of feeding to sleep, are governed by the principles which control breast-feeding (p. 81), and the following table, an elaborated form of that upon p. 82, gives an approximation of the frequency and the amounts for each feeding at different ages, the quantity being gradually increased as the age advances:

TABLE 49.—INTERVALS AND AMOUNTS FOR ARTIFICIAL FEEDING

Age	Intervals of feeding, hours	Number of feedings in 24 hours	Number of feedings at night after 10 P.M. and before 6 A.M.	Amount at each feeding, ounces	Total amount in 24 hours, ounces
1 to 4 weeks.......	2–2½	8–10	2	1–3	10–30
4 weeks to 3 months	2½–3	7–8	1	2½–5	20–35
3 to 4 or 5 months..	3–3½	6–7	1(?)	4–6	28–36
4 or 5 months to 1 year.	3–4	5–6	0	6–10	36–50

The length of the intervals and the number of feedings are much the same as for breast-feeding, and the divergence of opinions regarding the matter has been referred to in that section. There is, of course, much latitude to be allowed in using the table, dependent upon the digestion and the demands of the child. Regularity is important, but the amount taken at a feeding and the number of feedings in the twenty-four hours vary greatly with the case. An infant always hungry may need to exceed the amount for its age or to be fed at shorter intervals; another with large appetite may need longer intervals. It is important, too, to be guided considerably by the weight. An unusually large child may require more, while a small marantic infant may be able to take only much less than the age calls for. There can be no fixed rule. In general, a healthy infant may have all the food it will take at the feeding-time, provided it can retain and digest it; but care should be observed that an unusually large appetite is not dependent upon the giving of too weak a formula.

Laboratory-modification of Milk.—Through the efforts of Dr. T. W. Rotch,[1] ably seconded by Mr. G. E. Gordon the principle of the "Milk Laboratory" was evolved, and one such established in Boston in 1891 by the Walker-Gordon Laboratory Company. Similar laboratories have since been organized in many of the larger cities of the United States and in London. These institutions have been of the greatest benefit in giving an impetus to the scientific feeding of infants. Their object is to prepare and furnish any food-combination ordered by the physician, including those in which "differential protein" feeding is employed; *i.e.* in which the lactalbumin and casein are prescribed separately; and those in which cereal decoctions of known strength, buttermilk, and the like take a part. The physician merely writes a prescription calling for the various percentages he needs, the total number of bottles, the amount of food for each, the nature of the diluent, and any other requirement desired. If the food is to be pasteurized or sterilized, a starchy addition to be dextrinized, fermentation by lactic acid bacilli to be

[1] Arch. of Pediat., 1893, X, 97.

produced, or any other change made, this is specified in the prescription. It is manifest that a milk-laboratory is not only a great convenience to the physician to whom it is accessible, but that the resulting mixtures will be compounded more accurately than when made at home; since the employees are certainly better trained than is the mother or the nurse, and a careful control can be kept over the percentage-strength of the milk and cream used. Unfortunately the price of laboratory modified milk is necessarily considerably higher than persons of restricted means can afford, and for this reason, as well as on account of the usual inaccessibility of a laboratory for the majority of infants, home-modification is that which must oftenest be adopted.

Home-modification of Milk.—The calculation by the physician of the desired formulæ is in reality a very simple process. The preparation of the food can be carried out by any ordinarily intelligent and properly directed mother or nurse. The chief advantage of home-modification consists in its economy as compared with laboratory feeding. Although less accurate than this latter, it approaches it closely if a cream of definite known strength can be procured; and even for home-prepared whole-milk or top-milk mixtures it is quite sufficiently accurate for ordinary purposes. This is especially true since the possibility has extended so widely of procuring milk certified by a recognized Milk Commission. Milk of this sort should be obtained whenever feasible; and, when not, the requirements described (p. 118) should be fulfilled as far as possible. It is particularly important that it be of a fairly uniform strength in fat, and not over 4 per cent. When it is necessary to employ a richer milk, a slight change in the procedure is required. (See pp. 118 and 140.)

Many unwarranted statements have been made and published regarding the difficulty supposed to attend the preparation of milk-mixtures in the home, and the complications involved in the prescribing of these by physicians, the simple dilution of whole milk being urged without any percentage calculations. Percentage-feeding and home-modification may be made unnecessarily difficult, it is true, but do not in any way need to be so. The calculation of the desired percentages (p. 139) may, as already indicated, be made by any physician with the simplest school-boy knowledge of arithmetic. It is not so much lack of knowledge as unwillingness to take trouble which interferes on the part of the physician; but to prescribe the best obtainable food for the baby is so important that there can be no possible excuse for any physician who will not undertake to do this in the best way. The objection that home-modification of milk is too complicated for the mother is so futile that it hardly merits consideration. The mother has nothing whatever to do with percentages; and as she is obliged to modify the milk in some way, it can certainly cause no more trouble to dilute, say, the upper half of a quart of milk with water, than to dilute the whole quart. Other things being equal, the simplest way is the best way; and if the desired results can be obtained by the dilution of whole milk with water, this may well be done. But in the very numerous instances where a mixture is required in which the percentage of the fat is greater than that of the protein, it is only by the employing of a top-milk that the results can be obtained. (See also pp. 139; 140.) With the aid of clear, concise, written directions there is little chance of error; and mothers to whom the first preparation of any milk-mixture seems difficult rapidly find the processes becoming easy. But if the physician is hazy in his own mind regarding the matter, or neglects to give explicit instructions, the mother naturally grows confused and discouraged, whatever sort of food is ordered and whatever method employed.

Articles Required in Preparing the Food.—*Bottles.*—These should be of well-annealed glass to prevent breaking when exposed to heat. They may be of any shape desired, but are best narrow and cylindrical, since they occupy less space when of this form, and fit better in the pasteurizer. The mouths of the bottles should be rather wide, rendering cleansing easier. The bottles must be perfectly smooth within, without angles or depressions which can collect milk. A series of markings pressed into the glass indicate the number of ounces of contained fluid. There should be enough bottles provided to permit of all the food for twenty-four hours being prepared at one time.

Nipples.—These are preferably of black rubber, and of conical or slightly bulbous shape. The openings should be large enough to allow the milk to drop easily when the filled bottle is inverted, but not to run from it in a stream. If the holes are not of sufficient size they may be enlarged with a red-hot needle. As the nipple gets worn by use it collapses too easily and the holes grow too large. Special forms of nipples to "ventilate" the bottle are not to be recommended, as it is difficult to keep them clean. Too long a nipple is liable to press upon the palate and produce ulceration. The nipple attached to a rubber tube cannot be sufficiently condemned, as it is almost impossible to keep the tube clean. It is fortunately largely out of vogue.

Cream-dipper.—For the purpose of obtaining top-milk the most satisfactory method is the employment of the Chapin dipper. This has already been referred to (p. 117).

Sugar-measure.—The most convenient method of measuring the lactose is to employ the Chapin dipper. While holding 1 oz. of milk, it gives somewhat more than ½ oz. (Av.) of milk-sugar. This fact must be impressed upon the mother. Another way is to employ a tablespoon, but this is less satisfactory, as the variation in size is so great. Approximately 3¼ level tablespoonfuls, 2 rounded tablespoonfuls or 1½ dipperfuls of milk-sugar are equivalent to 1 oz. Av. Still another method is to place 1 oz. of sugar, as determined by weighing it, in a small paste-board box, level it, and then cut off the box at the line of the sugar. If tablespoonfuls are to be employed, the level one is better, inasmuch as the rounded tablespoonful gives more variation. Whatever method is used, the sugar should be poured into the measure until it is over-filled; the holder tapped sharply once or twice on the table, not more, and the excess of sugar removed with a case-knife. Too long tapping settles the sugar to too great an extent. Cane-sugar and the maltose-dextrin preparations vary somewhat in weight from that of lactose, the latter being a trifle lighter and the former decidedly heavier. Should it be desired to substitute one of them for lactose, it may be roughly estimated that 1 oz. of lactose equals 3¼ level tablespoonfuls or 1½ dipperfuls; 1 oz. of a maltose-dextrin, 4 level tablespoonfuls or 2 dipperfuls; and 1 oz. of saccharose, 2¼ level tablespoonfuls or 1 dipperful. (For weight of tablespoonfuls and dipperfuls of sugar and cereal-flours see also p. 162.)

Glass Graduate; etc.—A graduate holding 8 fl.oz., with the first ounce divided into drachms, is a great convenience in measuring the amounts of fluid required. One of the graduated nursing bottles may, it is true, be used instead, but these are not so accurately marked.

In addition are required a glass funnel, a bottle-brush, some sterilized unabsorbent cotton to make the stoppers for the bottles, pulverized boric acid, a bowl or pitcher in which to mix the food, and a Freeman pasteurizer described on p. 127.

Regarding the ingredients required, the best milk possible is to be obtained, running close to 4 per cent. butter-fat. (See Certified Milk, p. 119.) If purchased cream is used it should be separator cream, freshly obtained, of a guaranteed percentage-strength in fat, and with a low bacterial content. Milk-sugar should be purchased at least a pound at a time, as it is cheaper in this way. The fact that it is frequently impure is not to be forgotten. Should the physician desire to employ lime-water, this may be purchased or made at home. In the latter case a piece of unslaked lime the size of an egg is put into a gallon of water, which is then stirred vigorously and allowed to settle. The first water is poured off and fresh added. It should be kept covered from dust (see p. 125).

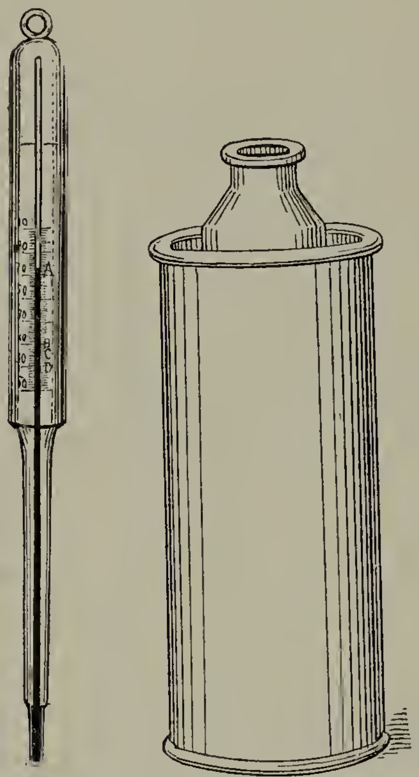

Fig. 22.—Milk-bottle, Heating Tin, and Thermometer.

Preparing and Giving the Food.—First is determined the number of bottles which the infant shall have in twenty-four hours and the number of ounces at each feeding, the multiplication of one number by the other giving, of course, the total amount required for the day. All of this is to be prepared at the one time, as soon as possible after the milk arrives. Should delay be unavoidable the milk must be kept on ice until needed. The required amount of sugar is then measured in the manner described, added to the amount of water or other diluent needed for the mixture, this raised to the boiling point for a moment, and then cooled. As the addition of the sugar increases the volume of the water slightly, greater accuracy may be obtained by boiling the sugar in a *portion* of the water only, and then adding enough additional water to bring the volume up to that of the total amount of solution to be added. The difference, however, is not very considerable. The boiling of the sugar-solution is necessary only if the mixture is to be used raw, on account of the bacterial content of the sugar often existing.

The amount of top-milk, cream, whole-milk, skimmed milk, and water—as the case may be—is now measured and added to the sugar-solution and all the ingredients mixed well, the volume of the whole equalling the amount of food desired for twenty-four hours. Should it be intended to sterilize the mixture at 212°F. (100°C.) and the use of lime-water be desired, this should not be added until the process is over. With pasteurizing at 140° to 150°F. (60° to 65.5°C.) there is no such necessity, inasmuch as the lime-water is not affected by this temperature. Into each bottle is now poured the amount for each feeding, and the neck of the bottle wiped clean and dry and then stoppered with a cotton-plug. The bottles are now put into the pasteurizer and the food pasteurized, if this is desired, cooled, and kept on ice. When a bottle of food is needed, and then only, it is removed from the ice and placed in a tin of cool water; and this is then warmed until the milk is at 98 to 100°F. (36.7° to 37.8°C.), but not more. This is determined by a thermometer made for the purpose and placed in the milk-bottle, not in the water

(Fig. 22). If the bottle is plunged directly into hot water the glass is liable to crack. The nipple is now put on and the child fed. In heating the food a tin sufficiently tall should be employed to allow the water to cover the bottle to the neck, but narrow enough to reduce to a minimum the actual amount of water required (Fig. 22). Placed upon a small alcohol-heater or gas-stove, the water and bottle of milk will thus be raised quite rapidly to the required temperature. This rapidity is an important matter when a hungry child is crying impatiently for food.

As with breast-feeding, regularity in the hours for food must be followed. The child should empty its bottle in from 10 to 20 minutes, and should not be allowed to suck at it indefinitely or to go to sleep until it has finished its meal. Any milk left over must be thrown away at once; never warmed up and given again later. The bottle should be held by the hand so that its neck is always full of milk. It should be drawn from the mouth from time to time to allow air to enter through the nipple in order to keep this from collapsing. The child should be held in the lap or in the arms while being fed. It is a bad plan to prop the bottle up with pillows to enable the infant to suck while it is in bed. After being used the bottle should be at once washed with a strong solution of washing soda, scrubbed with a bottle-brush, and rinsed out well with pure water. Shortly before they are filled all the bottles should be boiled at one time. The nipples should be scrubbed thoroughly without and, after turning inside out, within, and afterward be kept submerged in water or in a boric acid solution until needed. Before using it is well to plunge them for a moment in boiling water.

The Calculation of the Milk-formulæ.—In employing home-modification, milk-mixtures may be made either (1) from a combination of whole-milk and cream of a definite known strength, or (2) from dilutions of whole milk, top-milk or skimmed milk. The pages which follow are for the guidance of those readers prepared to take for themselves an active interest in the calculation of the amounts of the different elements required in the food. For those who would prefer to avoid the trouble, the milk-card shown on p. 144 may be found a useful guide. Various methods of computing the percentages have been proposed; none of them giving other than approximately accurate results, but near enough for practical purposes.

1. Whole-milk-and-cream Mixtures.—This was almost the only method employed for home-modification some years ago, but it has since given place almost entirely to top-milk mixtures. It is explained here because still in use by some physicians or in some hospitals; and because it has distinct advantages in occasional instances. It is applicable only when a cream of definitely known strength can be obtained. The simplest method of determining the amount of whole-milk and of cream required is that proposed by Baner.[1] We need not go into the derivation of his equations, but the calculation is as follows:

Let Q = the total quantity of food needed for 24 hours
F = the percentage of fat desired in the mixture.
P = the percentage of protein desired in the mixture
S = the percentage of sugar desired in the mixture.
C = the amount of cream to be used in the mixture.
CF = the percentage-strength of the cream in fat.
L = the amount of sugar (lactose) to be added to the mixture.
W = the amount of water to be added to the mixture.

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[1] New York Med. Jour., 1898, Mar. 12, 345.

Then
$$\text{(I)}\quad C. = \frac{Q}{CF - 4} \times (F - P).$$
$$\text{(II)}\quad M. = \frac{Q \times P}{4} - C.$$
$$\text{(III)}\quad W. = Q - (M + C).$$
$$\text{(IV)}\quad L. = \frac{Q \times (S - P)}{100}.$$

The denominator of equation (I) is always the percentage-strength of the cream in fat, minus 4.

As an example of the use of the formulæ; suppose that it is desired to give an infant 8 bottles of 4 oz. each, making a daily quantity of 32 oz.; that this shall contain fat 3 per cent., sugar 6 per cent. and protein 1.5 per cent., and that the cream is of 20 per cent. fat-strength.

If we substitute these figures in the equations these read:

$$\text{(I)}\quad C. = \frac{32}{20 - 4} \times (3 - 1.50) = \frac{32 \times 1.5}{16} = 3;\ \textit{i.e.}\ C = 3\ \text{oz. 20 per cent. cream.}$$

$$\text{(II)}\quad M. = \frac{32 \times 1.50}{4} - 3 = 9;\ \textit{i.e.}\ M. = 9\ \text{oz. whole milk.}$$

$$\text{(III)}\quad W. = 32 - (9 + 3) = 20\ \text{oz. water.}$$

$$\text{(IV)}\quad L. = \frac{32 \times (6 - 1.50)}{100} = 1.44;\ \textit{i.e.}\ \text{practically 1.5 oz. sugar.}$$

Should Equation II result in O, it would mean that no milk, but only cream of the strength indicated, is to be used in making the mixture. Resulting in a minus quantity it denotes that the formula desired cannot be constructed with the strength of cream employed, and that one with a higher percentage of fat must be selected. When it is necessary to employ a milk containing 5 per cent. of fat, the figure may be reduced to 4 per cent. by dipping off and discarding the top 2 ounces of the quart after the cream has separated, and then shaking up the bottle well before the remaining milk is used as "whole milk."

The criticism may properly be made that Baner's formulæ are inaccurate, since they assume that milk and cream each contain 4 per cent. of protein, whereas milk more often has 3.5 per cent., and cream has somewhat less sugar and protein than has whole milk. A comparison of the real and the calculated protein-percentages obtained shows, however, differences which are entirely negligible. It is only when employing milk-mixtures containing as much as 3 per cent. of protein that the difference between the calculated and the actual protein amounts present reaches as much as somewhat less than 0.5 per cent.

2. **Top-milk, Skimmed-milk and Whole-milk mixtures.**—In recent years the dilution of top-milk, whole-milk or skimmed-milk has become the popular method of preparing the food. It offers every advantage in convenience and in reasonably approximate accuracy in the percentages present. Many figures have been given by different investigators of the strengths of varying numbers of ounces of the milk in a quart milk-jar. They are close enough to show that this method of feeding can be employed satisfactorily. With the exception of the fat, the average infant can, as a rule, tolerate decided variations in the milk-elements; and the important matter after all, as has been stated previously, is not so much the initial accuracy of the percentages as it is the comparative accuracy of the *changes* in the food which it may be desired to make from time to time.

With this in mind, and aware especially of the importance of rendering the calculating as simple and as comprehensible as possible, I have for a number of years followed and taught the following plan. The percentages are based upon the published figures of different observers and upon extensive unpublished investigations made at my suggestion by Dr. Charles A. Fife. The percentages of protein and of sugar have intentionally been slightly distorted for reasons presently to be given. The title "skimmed-milk" designates any milk from which all or a portion of the cream has been removed. (See p. 118.)

Table 50.—Percentage-strengths of Top-milk and Skimmed-milk Layers

Milk obtained from a quart of 4 per cent. milk by dipping, as previously described (p. 117), gives:

	Fat, per cent.	Protein and sugar, per cent.	Approximate ratio
Upper 2 oz	24.0	4	6 to 1
Upper 4 oz	20.0	4	5 to 1
Upper 6 oz	16.0	4	4 to 1
Upper 8 oz	12.0	4	3 to 1
Upper 10 oz	10.0	4	2.5 to 1
Upper 16 oz	8.0	4	2 to 1
Upper 20 oz	6.0	4	1.5 to 1
Upper 24 oz	5.0	4	1.25 to 1
Upper 32 oz. (whole-milk)	4.0	4	1 to 1
Lower 30 oz	3.0	4	0.75 to 1
Lower 28 oz	2.0	4	0.50 to 1
Lower 16 oz	1.0	4	0.25 to 1
Lower 8 oz	0.5	4	0.0 to 1[1]

This table applies to milk containing approximately 4 per cent. of fat. With rich milk, showing 5 per cent. of fat, all that is necessary is to remove and discard the top 2 oz. and then to proceed with the dipping as though this had not been done. That is to say, by the "top 8 oz.," for example, is indicated the upper 8 oz. remaining after the top 2 oz. have been discarded. The figures in the table are not absolutely accurate, and are not intended to be. They are sufficiently so for practical purposes, and the discrepancy is slight as compared with the great convenience in application. As previously shown, the protein of whole milk is nearer 3.5 than 4 per cent., and is even slightly less than this in the top-milk usually employed; but the difference after the required dilution has taken place is so slight that it is negligible. This statement applies equally well to the difference between the adopted 4 per cent. of sugar and the actual 4.5 per cent., and to the fact that the upper 16 oz. is more properly stated as containing from 7 to 7.5 per cent. fat. The great convenience of the approximate ratios which the table shows quite justifies the slight distortion of the figures. It is only when high protein-percentages are used that the discrepancy in the percentages is to be considered—a calculated protein-strength of 3 per cent. in the mixture showing a deficiency of not quite 0.5 per cent. If one bears in mind the protein-needs of the infant (pp. 43 and 132) it is easy to determine whether the deficiency is one which must be considered. This will not be necessary unless the minimum requirement is being closely approached, and this, of course, will not happen when high protein mixtures are being employed.

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[1] The amount of fat in the lower 8 oz. is so small that it may be ignored in the "approximate ratio."

In employing the table we can conveniently follow the plan of making a 20-oz. mixture our basis. Should we need, for instance, 36 oz., twice the 20 oz. may be prepared, and the extra 4 ounces discarded; if 30 oz. is required, 1½ times the 20 oz. is made. It is to be noted first of all that the percentages of protein and of sugar are the same. Whatever the percentage we deduce for the former will be equalled by the latter. Our first step is to determine what percentages of fat, sugar and protein are wanted in the milk-mixture. Suppose, for instance, that we desire one of fat 3 per cent., sugar 6 per cent. and protein 1 per cent. The ratio of fat to protein is that of 3 to 1. In consulting the table it will be seen that the layer consisting of the upper 8 oz. and containing 12 per cent. of fat possesses this ratio, and is the proper one to use. Now it is evident that if 20 oz. of milk contain 12 per cent. of fat, and we wish to reduce this to 3 per cent., *i.e.*, ¼, we merely divide the 20 by 4, and make up the rest with water, *i.e.*, we use 5 oz. of the upper 8 oz., and 15 oz. of water. This may be expressed in an equation, to which reference will be made from time to time as the *basic equation.*

BASIC EQUATION TO OBTAIN THE DESIRED FAT-PERCENTAGE

$$\frac{\text{Total quantity of food} \times \text{Percentage of fat desired}}{\text{Fat-strength of the milk used}} = \begin{array}{l}\text{Quantity of top-milk or}\\ \text{skimmed-milk in the mixture.}\end{array}$$

Applying this to the milk-mixture in question the equation reads:

$$\frac{20 \times 3}{12} = 5 \text{ oz. of upper 8 oz. in a 20 oz. mixture.}$$

The calculation for the dilution to obtain the fat-percentage *is the only one required,* for the desired dilution of the protein follows automatically, and the original 4 per cent. is reduced to 1 per cent. The sugar has been cut down to the same amount, since the percentages of the two are always the same. Wishing, however, to have the sugar in the mixture 6 per cent., it is clear that since 1 per cent. is already present, we need only add 5 per cent.; *i.e.* 20 × 0.05 = 1 ounce of sugar to be added to the 20 oz. mixture. Our milk-formula will then read:

Upper 8 oz., 5 oz.
Milk-sugar, 1 oz.
Water to make 20 oz.

Very frequently a weak cereal-decoction is employed as a diluent. This naturally increases the total carbohydrate-percentage in the mixture; but as a rule this may be ignored, and account taken of the sugar only. Should, however, one wish to allow for the difference, and to add, say, 1 per cent. of starch, reducing the sugar-addition to 4 per cent., we may make a decoction of 1.80 per cent. of starch (see p. 164), and again apply the equation just given, the starch replacing the fat in it. This reads: $\frac{20 \times 1}{1.80}$ = approximately 11 oz., indicating that this amount of the cereal decoction is to be used in place of an equal number of ounces of water to make a 20 oz. mixture. The formula would then read:

Upper 8 oz., 5 oz.
Sugar, 0.8 oz. (*i.e.* 4 per cent. of 20 oz.).
Barley-water, 11 oz.
Water to make 20 oz.

Generally, however, such a refinement of calculation is not necessary.

One more example may be given. Let us suppose that we desire to make the protein-percentage in the mixture given 2 per cent. instead of 1 per cent.; viz. fat 3 per cent., sugar 6 per cent., protein 2 per cent.—a ratio of 3 to 2, with the sugar 6 per cent. as before. Two courses are open to us. First, and most simply, we can select the upper 20 oz. of the quart, containing 6 per cent. of fat and 4 per cent. of sugar—a ratio of 3 to 2, or 1½ to 1, as shown in the table. Using the same basic equation to obtain the percentage of fat, we have $\frac{20 \times 3}{6} = 10$ oz. of the top 20 oz. of the quart required in the 20 oz. mixture. The proportions of protein and of sugar have automatically become 2 per cent., the ratio of 3 to 2 holding good. The other course is as follows:
Using, for instance, the upper 8 oz. as in the first example we derive the mixture:

Upper 8 oz., 5.
Water, 15.,

giving fat 3 per cent., protein 1 per cent. To increase the protein to 2 per cent. without affecting the fat, it is only necessary to add a sufficient amount of milk in which the fat is a negligible quantity, as in the bottom 8 oz. of the quart. As this contains 4 per cent. of protein, it is evident that ¼ of the 20 oz. would contain 1 per cent. of protein. That is to say, by adding 5 oz. of skimmed-milk to the mixture in place of 5 oz. of water we shall have increased the protein-percentage from 1 per cent. to 2 per cent. Our milk-formula would then read:

Upper 8 oz., 5 oz.
Lower 8 oz., 5 oz.
Water, 10 oz.

Having 2 per cent. of protein in the mixture we necessarily have 2 per cent. of sugar, and all that is required to obtain the desired 6 per cent. of sugar is to add the additional 4 per cent.; *i.e.* $20 \times 0.04 = 0.8$ oz.

If a quantity of food is required which calls, for instance, for 10 oz. of the upper 8 oz., it is, of course, necessary to take 8 oz. from each of 2 quarts, or 8 oz. from a quart and 4 oz. from a pint, mix them and use 10 oz. of the mixture.

Where mixtures are desired in which the protein-percentage is in excess of the fat-percentage; *i.e.* skimmed-milk mixtures, the method of procedure is, of course, the same, using only the lower layers in the table in place of the upper. Thus for a mixture of fat 1 per cent. and protein 2 per cent., we can employ the lower 28 oz., containing 2 per cent. of fat, and having the ratio of 0.5 to 1. Our equation for the fat gives us $\frac{20 \times 1}{2} = 10$ oz. of the lower 28 oz. required in a 20 oz. mixture, this naturally reducing the 2 per cent. of fat and 4 per cent. of protein to 1 per cent. of fat and 2 per cent. of protein respectively.

In the cases where it is desired that the percentages of fat and of protein shall be approximately equal, whole-milk may well be employed. If, for instance, a formula of fat 2.5 per cent., sugar 5 per cent., and protein 2.5 per cent. is desired, we substitute in the basic equation the percentage-values of whole-milk as given in the table (p. 141) as follows: $\frac{20 \times 2.5}{4} = 12.5$ ounces whole-milk in the 20 oz. mixture. As this gives us necessarily also 2.5 per cent. of protein and 2.5 per cent. of sugar, the additional 2.5 per cent. of sugar is obtained as before; *i.e.* 20 × 0.025 = 0.5 oz. of sugar to be added.

The whole matter of calculation will be found extremely simple upon trial. It may be summarized in the following three rules:

1. *Select the top, skimmed or whole milk which possesses the ratio of fat to protein desired for the mixture* (Table 50, p. 141).
2. *By using the equation on p.* 142, *calculate the amount of this milk needed in a* 20 *oz. mixture to give the desired fat-percentage. The results will give the desired protein-percentage also.*
3. *The sugar having been, of course, reduced equally with the protein, add as much more to the* 20 *oz. as is needed to raise it to the desired percentage.*

Ready Method for Selecting Amounts to be Employed in Making Various 20-Oz. Milk-Mixtures, and the Caloric Values Resulting

Percentages desired of			Lower 8 oz.	Lower 16 oz.	Lower 28 oz.	Whole milk	Upper 24 oz.	Upper 20 oz.	Upper 16 oz.	Upper 10 oz.	Upper 8 oz.	Water oz.	Sugar oz.	Caloric value of mixture	Calories[1] per oz.
Fat	Sugar	Prot'n													
0.5	5	1	..	..	5	..	..	..	..	..	..	15	0.8	175	9.10
0.5	6	2	..	10	..	..	..	..	..	..	..	10	0.8	225	11.7
1	6	1	..	..	..	5	..	..	..	..	..	15	1	225	11.7
1	6	1.5	2.5	..	..	5	..	..	..	..	..	12.5	0.9	237.5	12.35
1	6	2	..	..	10	..	..	..	..	..	..	10	0.8	250	13.0
1.5	6	1	..	..	..	..	..	5	..	..	..	15	1	250	13.0
1.5	6	1.5	..	..	..	7.5	..	..	..	..	..	12.5	0.9	262.5	13.65
2	6	1	..	..	..	..	..	..	5	..	..	15	1	275	14.3
2	6	1.5	2.5	..	..	..	..	..	5	..	..	12.5	0.9	287.5	14.95
2	6	2	...	..	..	10	..	..	..	..	..	10	0.8	300	15.6
2.5	6	1.5	2.5	..	..	..	..	..	..	5	..	12.5	0.9	312.5	16.25
2.5	6	2	..	..	..	..	10	..	..	..	..	10	0.8	325	16.9
2.5	6	2.5	..	..	..	12.5	..	..	..	..	..	7.5	0.7	337.5	17.55
3	6	1	..	..	..	..	..	..	..	..	5	15	1	325	16.9
3	6	1.5	2.5	..	..	..	..	..	..	..	5	12.5	0.9	337.5	17.55
3	6	2	..	..	..	..	..	10	..	..	..	10	0.8	350	18.2
3	6	3	..	..	..	15	..	..	..	..	..	5	0.4	375	19.5
4	4	4	..	..	..	20	..	..	..	..	..	0	..	400	20.8

FIG. 23.—AUTHOR'S MILK-CARD.

Showing amount of different layers to be used for the preparation of various percentage-mixtures, and the caloric values of the mixtures. (*Griffith, Jour. Amer. Med. Assoc.*, 1918, *LXXI*, 441.)

It sometimes happens that a formula is desired which does not possess a fat-protein ratio like any in the table;—such, for instance, as fat 2.5 per cent., sugar 6 per cent. and protein 1.5 per cent., a ratio of fat to protein equalling 5 to 3. A slight, simple modification of the method then becomes necessary. In the basic equation we first substitute the word "protein" for "fat," making it read:

$$\frac{\text{Total quantity of food} \times \text{percentage of protein desired}}{\text{Protein-strength of milk used}}$$

We now are to find the total amount of milk of any sort,—top-milk, skimmed-milk, or whole-milk,—which will give us 1.5 per cent. of protein. By substituting in the equation, this will be $\frac{20 \times 1.5}{4} = 7.5$

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[1] Using 1.3 as Multiple (See p. 124).

oz. of milk. We next use the original basic equation, and selecting, let us say, the upper 10 ounces of the quart containing 10 per cent. of fat, calculate how much of this must be used to give us the desired 2.5 per cent. of fat. The equation for this will read: $\frac{20 \times 2.5}{10} = 5$ oz. of the upper 10 ounces of the quart. We now have under way a mixture in which the total milk is 7.5 ounces to give us 1.5 per cent. of protein, and in which 5 ounces of this, taken from the upper 10 ounces of the quart, gives us the desired 2.5 per cent. of fat. To make up the lacking 2.5 ounces of milk to furnish the desired 1.5 per cent. of protein we must use the bottom 8 ounces of the quart which will increase the protein but will not materially affect the fat. Our formula will now read:

Upper 10 oz. of a quart, 5 oz.
Lower 8 oz. of a quart, 2.5 ounces.
Water to make 20 oz.

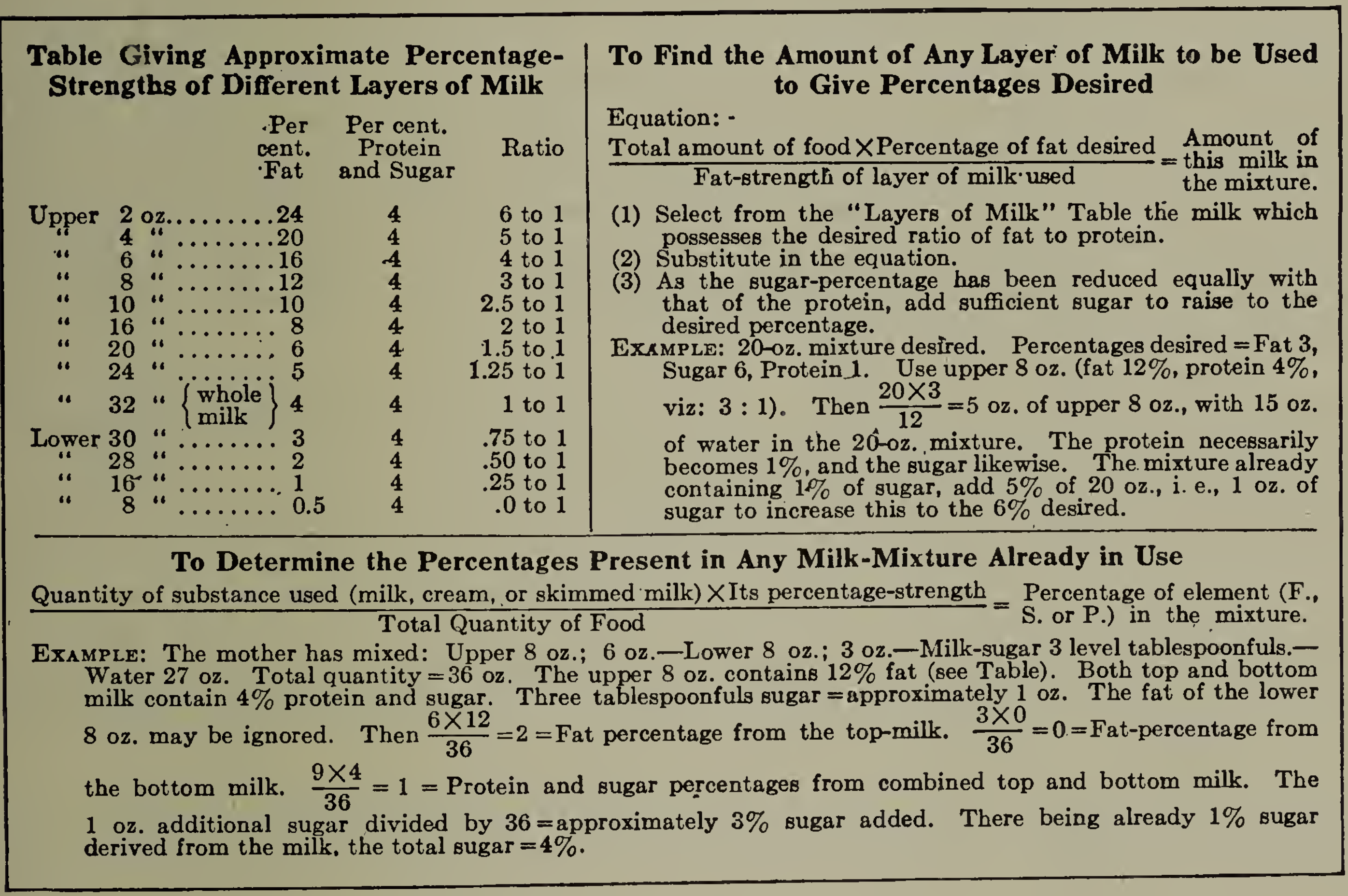

Table Giving Approximate Percentage-Strengths of Different Layers of Milk

	Per cent. Fat	Per cent. Protein and Sugar	Ratio
Upper 2 oz.	24	4	6 to 1
“ 4 “	20	4	5 to 1
“ 6 “	16	4	4 to 1
“ 8 “	12	4	3 to 1
“ 10 “	10	4	2.5 to 1
“ 16 “	8	4	2 to 1
“ 20 “	6	4	1.5 to 1
“ 24 “	5	4	1.25 to 1
“ 32 “ whole milk	4	4	1 to 1
Lower 30 “	3	4	.75 to 1
“ 28 “	2	4	.50 to 1
“ 16 “	1	4	.25 to 1
“ 8 “	0.5	4	.0 to 1

To Find the Amount of Any Layer of Milk to be Used to Give Percentages Desired

Equation: -

$$\frac{\text{Total amount of food} \times \text{Percentage of fat desired}}{\text{Fat-strength of layer of milk used}} = \text{Amount of this milk in the mixture.}$$

(1) Select from the "Layers of Milk" Table the milk which possesses the desired ratio of fat to protein.
(2) Substitute in the equation.
(3) As the sugar-percentage has been reduced equally with that of the protein, add sufficient sugar to raise to the desired percentage.

Example: 20-oz. mixture desired. Percentages desired = Fat 3, Sugar 6, Protein 1. Use upper 8 oz. (fat 12%, protein 4%, viz: 3 : 1). Then $\frac{20 \times 3}{12} = 5$ oz. of upper 8 oz., with 15 oz. of water in the 20-oz. mixture. The protein necessarily becomes 1%, and the sugar likewise. The mixture already containing 1% of sugar, add 5% of 20 oz., i. e., 1 oz. of sugar to increase this to the 6% desired.

To Determine the Percentages Present in Any Milk-Mixture Already in Use

$$\frac{\text{Quantity of substance used (milk, cream, or skimmed milk)} \times \text{Its percentage-strength}}{\text{Total Quantity of Food}} = \text{Percentage of element (F., S. or P.) in the mixture.}$$

Example: The mother has mixed: Upper 8 oz.; 6 oz.—Lower 8 oz.; 3 oz.—Milk-sugar 3 level tablespoonfuls.—Water 27 oz. Total quantity = 36 oz. The upper 8 oz. contains 12% fat (see Table). Both top and bottom milk contain 4% protein and sugar. Three tablespoonfuls sugar = approximately 1 oz. The fat of the lower 8 oz. may be ignored. Then $\frac{6 \times 12}{36} = 2$ = Fat percentage from the top-milk. $\frac{3 \times 0}{36} = 0$ = Fat-percentage from the bottom milk. $\frac{9 \times 4}{36} = 1$ = Protein and sugar percentages from combined top and bottom milk. The 1 oz. additional sugar divided by 36 = approximately 3% sugar added. There being already 1% sugar derived from the milk, the total sugar = 4%.

Fig. 24.—Author's Milk-card.

Reverse side of Fig. 23, showing the percentages in different layers of milk; the calculation of the amount to be used for any formula desired, and the calculation of the percentages in any mixture already in use. (*Griffith, Jour. Amer. Med. Assoc.*, 1918, *LXXI*, 441.)

As the percentages of protein and sugar are the same, this mixture, containing 1.5 per cent. of protein, has also 1.5 per cent. of sugar derived from the milk, and the addition of 4.5 per cent. of sugar is necessary to make the 6 per cent. wanted; *i.e.*, $20 \times 4.5 = 0.9$ oz. of sugar to be added.

The figures in the table (p. 141) run so easily in sequence that they are carried in mind without effort, and a little consideration will reveal many possibilities of producing the same results by different methods. Thus a formula calling for 3 per cent. of fat and 2 per cent. of protein can be made either by diluting 10 oz. of the upper 20 oz. of the quart with 10 oz.

of water as already shown (p. 143), or by mixing 5 oz. of the top 8 oz. with 5 oz. of the bottom and adding 10 oz. of water. One requiring 2 per cent. of fat and 1 per cent. of protein may consist of 5 oz. of the top 16 oz. of the quart and 15 oz. of water; or of 3⅓ oz. of the top 8 oz., 1⅔ oz. of the bottom, and 15 oz. of water. One calling for 2 per cent. of fat and 2 per cent. of protein may be constructed simply by diluting whole-milk with an equal amount of water, or by mixing 4 oz. of the top 10 oz. with 6 oz. of the bottom 8 oz. and 10 oz. of water. In each case the requisite percentage of sugar must be added. The simplest way is usually the best, both for the physician and for the mother or nurse.

For ready reference, those who desire to avoid all calculation as far as possible may employ the milk-card shown in Fig. 23. This is based on the percentage-strengths as given on p. 141. Fraley's formula has been used for the determination of the total caloric values, but the factor 1.3 for the value of a single ounce. (See p. 124.) The milk-mixtures selected are those most likely to be found useful. The employment of fractional percentages smaller than those given offers no practical advantage.

To Ascertain the Percentages Present in Any Milk-mixture.—Inasmuch as the description of the food in use is always given by the mother in terms of milk, cream, top-milk, water, etc., it is important to be able readily to ascertain what percentages of the milk-elements the child is actually receiving, in order to determine whether any ingredient is manifestly present in incorrect proportion and is probably at fault if symptoms of indigestion exist.

The procedure is simple. To calculate the total percentage of any of the milk-elements present in the mixture, multiply the quantity of the substance (milk, cream, etc.) containing it by the percentage of the element present in this, and divide by the total number of ounces of the mixture. In the form of an equation it would read:

$$\frac{\text{Quantity of substance used (milk, cream, etc.)} \times \text{Its percentage-strength}}{\text{Total quantity of food}} = \text{Percentage of ingredient in the mixture.}$$

This is employed to determine both the fat-percentage and that of the protein and sugar. Supposing, for instance, we are told that the infant has been receiving a mixture of:

Upper 8 oz. of a quart, 6 oz.
Lower 8 oz. of a quart, 3 oz.
Milk-sugar, 3 rounded tablespoonfuls.
Barley-water, 27 oz.

making a total food-mixture of 36 oz., containing a total of 9 oz. of milk in all. We are told that the barley-water is made by cooking 2 level tablespoonfuls of barley-flour in 1 quart of water, and replacing the water which has been boiled away. Using now the Table of Percentage Strengths, (p. 141), with the same restrictions as to accuracy as explained regarding it, we have in the upper 8 oz., fat 12 per cent., protein and sugar each 4 per cent.; in the lower 8 oz., fat practically 0 per cent., protein and sugar each 4 per cent. The 3 rounded tablespoonfuls of sugar are equivalent to 1½ oz. (pp. 137, 162). The barley-water gives a percentage of 1.20 of starch (see Table 57, p. 164). Substituting these various values in the equation the figures are as follows:

$\frac{6 \times 12}{36} = 2$ Fat-per cent. obtained from the top-milk.

$\frac{3 \times 0}{36} = 0$ Fat-per cent. obtained from the bottom-milk.

$\frac{9 \times 4}{36} = 1$ Protein-per cent. obtained from the combined top- and bottom-milks $(6 + 3)$.

$\frac{9 \times 4}{36} = 1$ Carbohydrate-per cent. obtained from the combined top- and bottom-milks.

$\frac{27 \times 1.20}{36} = 0.9$ Carbohydrate-per cent. obtained from the barley-water.

Besides this we have added 1.5 oz. of extra sugar to the 36 oz. of mixture; and dividing the 1.5 by 36 this furnishes a percentage-addition of approximately 4 per cent., which, with the sugar already present, and the carbohydrate of the barley-water, gives a total carbohydrate-percentage of about 6 per cent. The food is thus found to contain fat 2 per cent.; carbohydrate 6 per cent.; protein 1 per cent.

For the convenience of my students I had the table for the ready method of the preparation of milk-mixtures printed in pocket-card form (Fig. 23), and upon the reverse side (Fig. 24), the table for layer-milk (p. 141) and the various matters relating to the calculation of formulæ as already described.[1]

Simple-Dilution Feeding.—Many pediatrists advocate the giving of mixtures made by simple dilution of whole milk, on the ground that it is an easier method of infant-feeding. The procedure is open to serious objections, some of them already indicated. (See pp. 122; 136.) It limits very greatly the flexibility in the construction of the formulae, which is so greatly to be desired. The percentage of the protein must necessarily with whole-milk mixtures be always the same as the fat. Should it seem advisable to increase the percentage of fat, that of the protein must likewise be increased beyond that which is actually needed. That this is not a matter of indifference has already been pointed out (see pp. 42, 112, 132), since the energy required to metabolize protein much exceeds that needed for the other elements. It has further been claimed that the fact that fat is often difficult of digestion renders a whole-milk dilution, with its comparatively low fat and with the addition of sugar, all that is required. This is undoubtedly true for many ill infants; but, on the other hand, all recent investigations tend increasingly to show the importance of a proper amount of fat in the food. Our effort should always be, then, to give the desired higher fat-percentage to all infants except those who show an intolerance for it.

An argument also advanced for whole-milk dilution as a routine method of feeding is the greater simplicity in calculation. It seems hardly worth while to discuss this. Our chief aim should not be to seek the easiest way for the physician, but the best method for the child (see p. 136).

Nothing in all this militates at all against the employment of whole-milk dilution in the case of infants who specifically need this more than any other food. Consequently the following explanation is given of the method which may be employed, based upon the practise of those especially in favor of feeding in this way, and combined with procedures which have already been discussed.

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[1] These cards may be obtained from Edward Pennock, 3609 Woodland Ave., Phila., at a cost of 6 cents.

(1) **To obtain the number of ounces to be given at each feeding:**
Add the figure 2 to the figure representing the age in months.

(2) **To obtain the total number of ounces for 24 hours:**
Multiply the number of ounces to be given at each feeding by the number of feedings to be given in 24 hours (v. Table 49, p. 135).

(3) **To obtain the number of ounces of whole milk to be used:**
Multiply the weight in pounds by 1.5 in the first 3 mo. of life.
Multiply the weight in pounds by 1.75 from the 3d to the 5th mo.
Multiply the weight in pounds by 2.0 after the 5th mo.

(4) **To obtain the number of ounces of diluent:**
Subtract the number of ounces of milk to be used from the total number of ounces for 24 hrs.

(5) **To obtain the percentages of fat, carbohydrate and protein in the above mixture:**
Use the method described on p. 146:—

$$\frac{\text{Quantity of substance used} \times \text{Its percentage strength}}{\text{Total quantity of food}}.$$

(6) **To obtain the number of ounces of sugar to be added:**
Subtract the percentage of sugar already present in the mixture (see p. 142) from the percentage of sugar desired (which is usually 6). The resulting figure represents the precentage of the total number of ounces in the mixture to be added as sugar.

Example.—Suppose that a mixture is to be calculated for an infant of 2 months of age whose weight is 9 pounds.

(1) 2 (age in mos.) + 2 = 4 (no. of ozs. at each feeding).

(2) 4 (no. of ozs. each feeding) × 7 (no. of feedings in 24 hrs.) = 28 (total no. of ozs. in 24 hrs.).

(3) 9 (wt. in lbs.) × 1.5 (no. ozs. milk per lb. body wt.) = 13.5 (approximately 14, the no. ozs. milk to be used in the mixture).

(4) 28 (total no of ozs. in mixture) − 14 (no. of ozs. milk) = 14 (no. of ozs. diluent).

(5) $$\frac{14 \text{ (no. ozs. milk)} \times 4 \text{ (per cent. of fat in milk)}}{28 \text{ (total no. ozs. in mixture)}} = 2 \text{ (per cent. fat in mixture).}$$

As the percentage of carbohydrate and protein in whole milk is the same as the percentage of fat it follows without further calculation that 14 ozs. of milk and 14 ozs. of water (or other diluent) = Fat 2 per cent., Carbohydrate 2 per cent. and Protein 2 per cent.

(6) 6 (percentage of carbohydrate desired) − 2 (percentage already present) = 4 (percentage of carbohydrate to be added). 4 per cent of 28 (total no. of ozs. in mixture) = 1.12 (no. of ozs. sugar to be added). Ounces of sugar may readily be reduced to tablespoonfuls (v. p. 137).

This method of calculation, as is the case with any other so-called method, has its limitations. The steps given above are to be used for well infants of average weight. Very young infants, or those with feeble digestion or recovering from acute intestinal disturbances, must receive less than 1.5 ozs. of whole milk per pound of body-weight.

The 5th and 6th steps of the method may be avoided if the sugar to be added to the mixture is calculated in a simpler way. For example, 0.1 oz. of sugar may be added to the mixture for each pound of the infant's body-weight (not more than 1.5 ozs. of sugar ever being added to a mixture). Or 1 oz. of sugar may be added to the day's mixture if the infant is under 10 lbs. in body-weight, and 1.5 ozs. if the weight is over 10 lbs. By these two systems it is possible to overfeed or to underfeed with sugar. If, however, sugar is added (as directed in Step 6) so that the total sugar-percentage is 6, the carbohydrate requirement of the average healthy baby is adequately met and not exceeded.

Concentrated Feeding.—Concentrated foods of different types are often employed in infant-feeding. (See also Water, p. 98.) A number of these are mentioned elsewhere, among them the Czerny-Kleinschmidt butter-flour mixture (p. 169) thick cereal-gruels (p. 199), lactic acid milks (p. 155), the milk-and-pudding food of Moll and Stransky (p. 171) and Keller's malt-soup (p. 166). Finkelstein's casein-milk (p. 157) is also to be considered as a concentrated food, especially when twice the usual amount of curds is added, as well as sometimes 10 per cent. or more of sugar, as is done on occasions. Foods of even higher caloric value have been recommended, and under certain conditions are serviceable. Helmreich and Schick[1] have used a mixture approximately twice the caloric value of milk and which is denominated *Dubo*. This consists of whole milk to which has been added 17 per cent. of sugar, the two being mixed and boiled for 1 minute. Moro[2] advises a formula of butter-flour to which no water is added as a diluent and which consists of 1 liter of whole milk, 30 grams of wheat-flour, 50 grams of butter and 70 grams of sugar; or with the approximate equivalents of milk 1 quart, flour 3¾ level tablespoons, butter 5 level tablespoons and sugar 7 level tablespoons. This is prepared by heating the butter over a gentle fire for 3 to 5 minutes and then adding the flour and boiling the mixture for 4 or 5 minutes until a brownish color develops. The milk, which has been warmed and which contains the dissolved sugar, is then added. This food possesses approximately fat 9 per cent., carbohydrate 14 per cent. and protein 4 per cent., and each ounce of it has a caloric value of 46.8. In another formula Moro increases the flour to 70 grams and decreases the sugar to 50 grams to the liter, or approximately 8¾ level tablespoons of flour and 5 level tablespoons of sugar to the quart of milk. This mixture is prepared as is the other, after which it is boiled until it has the consistency of thick gruel. In a later publication Schick[3] recommends the concentration of breast-milk by the addition of from 8.5 to 17 per cent. of saccharose. He found this especially useful in premature infants, in whom it is difficult to obtain the required calories from the amount of ordinary breast-milk which they can be made to ingest. In the absence of breast-milk, whole cow's milk with sugar may be tried, often with good results.

The *indications* for concentrated feeding are in complementing breast-feedings; in vomiting of a nervous type or due to pyloric stenosis and pylorospasm or to pertussis; in malnutrition without definite digestive disturbance; in anorexia of nervous or other origin or due to infections; or in prematurity or other conditions where, from feebleness or other cause,

[1] Zeitschr. f. Kinderheilk., 1921, XXX, 121; 147; 363.

[2] Monatsschr. f. Kinderheilk., 1920, XVIII, 97.

[3] Arch. of Pediat., 1925, XLII, 397.

there is difficulty in obtaining the ingestion of a sufficient number of calories. This form of feeding is sometimes of value in the exudative diathesis and in eczema of the moist type, and has been recommended in enuresis, serous pleural effusion, and in edema of either cardiac or nephritic origin. The high caloric value of these foods makes it possible to give them in small quantity and at infrequent intervals, and the tolerance of the digestive tract for them is often great. They should be regarded as temporary foods, only to be used when there are distinct indications, and to be discontinued after a few weeks. They are usually contraindicated in hot weather and where there is evidence of indigestion. When they are employed, the fluid intake may be kept up by the administration of water between feedings, unless it is desired to use the diminished amount of water as a therapeutic measure, as in enuresis, pleural effusion or edema.

The Pirquet Method.—So much interest has been aroused in Pirquet's system of nutrition, that a brief synopsis of it seems necessary. It was intended for the feeding of older children as well as for infants. The premises upon which the author bases his system may be stated briefly as follows: It is believed by him that the sitting height bears a very close relationship to the normal body-weight. From this has been formulated a law, according to which the cube-root of 10 times the weight in grams approximately equals the sitting height in centimeters. This may be expressed by the equation:

$$\frac{\sqrt[3]{10\text{ weight}}}{\text{sitting height}} = 1 = \frac{100}{100}.$$

If the weight is greater than the average, the ratio will be more than $^{100}/_{100}$; if it is less than the average the ratio will be less than $^{100}/_{100}$. By this formula Pirquet judges whether the actual weight of an individual corresponds to the weight normal for his sitting height. To make the formula easier to remember the word *Pelidisi* has been coined out of the initials of the constituents: P standing for *pondus*, the body-weight in grams; Pe recalling the fact that it is not simple weight but *pondus decies*, 10 times the weight; Li indicating that the weight must be converted into a linear function by taking the cube-root of it; Di meaning "division" and Si "sitting height."

The calculation of food-values in calories is objected to by him, chiefly because it does not allow for the portion of the food ingested which is unavailable for heat-production, and a physiologic unit is used instead called the *nem* (Nahrungs-Einheit-Milch), which is the nutritive, combustible value of 1 gram of average human milk. This milk-unit is employed as a metric unit, and is combined with the Latin prefixes for metric fractions (a decinem meaning, for instance, one-tenth of a nem, etc.) and with the Greek prefixes for multiples of the unit (a dekanem meaning 10 nems). Tables are available which give the nem-value of food-stuffs. The protein and the fat should be kept each within from 10 to 20 per cent. of the total intake. It was found that when fat was difficult to obtain the difference could be made up with sugar without apparent injury, but this substitution is not advocated except when necessary. Pirquet accepts Henning's work, which is said to show that the length of the intestinal canal is, on an average, 10 times the sitting height, and believes that the square of the sitting height may be taken as a *symbol* of the absorbing surface of the intestinal tract, which stands in true relation to the intake of food. By observing a large number of individuals, data were secured as to the amount of food, calculated in decinems, which is ingested per square centimeter of absorbing intestinal surface as calculated from the sitting height; or, to use another coined word, how many *decinemsiqua* are ingested (*siqua* being the word which represents the square of the sitting height). The maximum amount of food which a normal individual can digest within 24 hours is about 10 decinems per square centimeter of the square of the sitting height (*i.e.*, 10 decinemsiqua). The minimum amount of combustible material to maintain the body-weight in a condition of absolute quiet is 3 decinemsiqua. The *aequum* is the amount of food required to maintain weight under a given condition of activity. The *optimum* amount of food in children is greater than the aequum because at this period additional food must be ingested to insure growth. Usually 1 decinemsiqua is added to the aequum for this purpose.

An example may be cited: A boy of 11 years with a sitting height of 70 cm. has an absorbing surface of 70 by 70 or 4900 sq. cm. The maximum that he could digest would be 10 decinems per square centimeter or 4900 nems. The minimum, if he stays in bed without moving at all, would be 3 decinemsiqua; *i.e.*, 4900 by 3/10 or 1470 nems. Though he sits in school for some hours, he is likely to be very active during the rest of the day. Therefore, 3 decinemsiqua more than the minimum are added to maintain his weight; *i.e.*, 6 decinemsiqua per day. In order to provide for growth and gain we must give 1 decinemsiqua more, or 7 decinemsiqua in all (7/10 of 4900, or 3360), which is his optimum allowance.

This system of calculating nutritional standards and food-values undoubtedly served a most useful purpose in the feeding of large groups of individuals under certain conditions. It is, however, decidedly complicated, and there seems to be little advantage in adopting it. Perhaps the most important criticism of Pirquet's system is that the assumption is quite certainly incorrect, that sitting height and absorbing surface of the intestinal tract and food requirements stand in close relation to each other. The reader who wishes to learn more of the system should consult the publications of Pirquet,[1] Carter[2] and Faber.[3]

Whey.—This consists of milk from which the casein has been removed by the use of rennet although the lactalbumin remains. The amount of fat is diminished very greatly, being entangled in the curd which the rennet produces. Whey is prepared as follows: Into a quart of warm, fresh milk, heated to 100°F. (37.8°C.) are stirred 2 teaspoonfuls of liquid rennet or of essence of pepsin. After coagulation has taken place the milk is placed in the cold for about ½ hour, the curd then broken up with a fork in order to liberate the whey, and the latter strained through cheese-cloth or muslin without pressure. There will be obtained from 1 to 1½ pints of whey. The finer-meshed and thicker the cloth the more fat will be removed by it. When it is desired to produce a whey containing practically no fat, a fat-free, separator milk or the milk from a quart jar after the cream has been removed should be employed instead of whole-milk. Failure to coagulate firmly indicates that not enough rennet was used or that the milk had been boiled previously or was too cold. If whey is to be mixed later with milk or cream, the rennin remaining in it must be destroyed by heating it for some time to a temperature of 140°F. (60°C.) or over (pasteurizing), otherwise the casein of the cream will coagulate. If it is heated to 75°C. (167°F.) coagulation of the lactalbumin begins. Various analyses of whey have been made. The average of many of them, according to the figures given by König,[1] is as follows:

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[1] System der Ernährung, 1917. An outline of the Pirquet System of Nutrition, 1922.

[2] Jour. Amer. Med. Assoc., 1921, LXXVIII, 1541.

[3] Amer. Jour. Dis. Child., 1923, XXV, 339.

TABLE 51.—COMPOSITION OF WHEY (KÖNIG)

Fat	0.32
Sugar	4.83
Whey-protein	0.85
Salts	0.64
Water	93.36

It will be seen from this that the percentage of salts is practically unchanged as compared with milk, and that of sugar slightly increased. Certain later analyses differ from these. Thus White and Ladd[2] found an average of 1.02 per cent. of protein and the Department of Agriculture of the United States[3] one of 1.00 per cent.

Whey-cream Mixtures.—Whey was for many years a very favorite food for cases of weak digestion, either given for a brief period, alone except for the addition of cane-sugar, or combined with small amounts of cream. Falling for a while into disuse, its employment was revived at the time the fear of the injurious effects of a high casein-percentage became prominent; the purpose being to replace the casein by lactalbumin. Later, with increasing confidence in the digestibility of casein, the use of whey has been neglected and even condemned, on the ground that the whey-protein was injurious in many cases of digestive disturbance. This neglect has been harmful, for although the need for whey-combinations is comparatively infrequent, yet there are numerous instances especially in early infancy when they are of undoubted benefit. In fact, the whey is the element which contains the bulk of the necessary amino-acids, the casein being very deficient in these. (See pp. 44 and 132.) Regarding the harmfulness it is doubtful whether, in any case of injury by whey, this is not to be attributed to the sugar or the salts rather than to the lactalbumin.

The production of whey-cream mixtures in which the fat shall be sufficiently abundant and the casein in very small amount can be accomplished only with the employment of the richer creams. This is because with the weaker creams and top-milks of from 8 to 16 per cent. strength, so much needs to be used to obtain sufficient fat that a very considerable percentage of casein is necessarily added also. The whey-cream mixtures are consequently much better prepared by laboratory-modification when possible. Using, however, the top 2 oz. of the quart, containing 24 per cent. of fat, successful home-modifications can be made. Since the desire usually is to have present as much lactalbumin as possible in the food and as little casein, and since the fat-percentage of the whey is so low and so little cream is employed, we shall make no great error in considering the fat as derived entirely from the cream and the protein and sugar from the whey. Suppose, for instance, that 3 per cent. of fat is desired in a 20 oz. mixture, we can construct a food by using the basic equation previously given (p. 142). Substituting the figures we shall have:

$$\frac{20 \times 3}{24} = 2.5 \text{ oz. of 24 per cent. cream.}$$

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[1] Chemie der menschlichen Nahrungs-u. Genussmittel, 1903, I, 389.
[2] Phila. Med. Jour., 1901, VII, 218.
[3] Bulletin No. 28. Ref. White and Ladd, *loc. cit.*

The remaining 17.5 oz. will be whey. The total sugar-percentage as derived from the whey equals nearly 5 per cent. (4.83), and as much more sugar may be added as is desired to bring it up to the required percentage. A closer calculation of the whey-formula is hardly necessary. Those, however, who desire to take into account the amount of casein present in the top-milk and of fat in the whey may follow the methods elaborated by Westcott.[1]

Peptonized Milk.—For infants with feeble digestion the pancreatizing of the milk before it enters the alimentary canal was formerly much used and was sometimes of benefit. This is usually accomplished by the action on the milk of the trypsin derived from the pancreatic juice of the pig, which partially digests the casein before it is ingested and prevents the formation of tough coagula. As the pancreatic extract requires the presence of an alkali in order to act, it is commonly sold combined with bicarbonate of soda, in the form of a powder in individual glass tubes. These are preferable to the "peptogenic" powder on the market, as they permit of greater range in varying the food at will. The pancreatizing is accomplished by dissolving the contents of 1 tube in 2 oz. of cool water and adding to 1 pint of cool milk; and then allowing the vessel containing the mixture to stand in hot water of a temperature of not over 115°F. (46°C.), *i.e.* as warm as the finger can bear without discomfort. It may remain here for 20 or 30 minutes, or a shorter time if the slightest bitter taste is discovered, and is then quickly cooled and kept on ice, or, better, heated quickly to boiling. The temperature of boiling destroys the trypsin and prevents further peptonization, thus avoiding the development of the bitter taste. The peptonizing may be applied similarly to the top-milk, or to the modified milk-mixture after it has been prepared in the ordinary way.

It is not best to continue the use of peptonized milk for too long a time, as it takes away the necessity, and, to an extent, the power of the digestive organs to do their own work. Its employment is serviceable only when there is a disturbance of protein-digestion. It is with reason much less frequently made use of than formerly; but is certainly useful in some cases. In this connection it is interesting to note that Bessau, Rosenbaum and Leichtentritt[2] have shown by repeated experiments that if the peptonizing is done by pepsin instead of pancreatin, the length of time that the milk remains in the stomach of the infant is shortened by 25 per cent.

Buttermilk.—The term "buttermilk" is properly applied to the milk which remains after the butter-fat has been removed by churning. Its employment for the feeding of infants with delicate digestion, for many years widely followed in Holland, appears to have been first urged upon the profession by Ballot[3] in 1865. Renewed attention was directed to it by the writings of de Jager,[4] Salge,[5] Teixeira de Mattos,[6] and, later, by many others. The composition of buttermilk varies according to different published analyses, and depends to some extent upon whether made from whole sweet milk, sweet cream, or sour cream. By all methods the fat-percentage is low, that of the protein rather high, and that of the sugar more or less reduced. The following table gives approximate analyses.

[1] Internat Clinics, 1900, III.
[2] Jahrb. f. Kinderheilk., 1921, XCV, 123.
[3] Nederl. Tijdschr. v. Geneesk., 1865, II, 402.
[4] Nederl. Tijdschr. v. Geneesk., 1895, XXXI, 679.
[5] Jahrb. f. Kinderheilk., 1901, LIV, 681.
[6] Jahrb. f. Kinderheilk., 1902, LV, 1.

Table 52.—Approximate Composition of Buttermilk

Fat	0.5–1.0 per cent.
Sugar	0.3–4.0 per cent
Protein	2.5–4.0 per cent.

The average composition from a large number of published statistics collected by König[1] gives water 90.09, fat 1.02, sugar 4.24, nitrogenous material 3.91, and salts 0.74. Buttermilk is always distinctly acid from the development in it of lactic acid and contains usually about 0.5 per cent. of this. If the acidity grows too great the buttermilk separates into a whey-like and a more solid portion. The casein is always coagulated and is in the form of casein-lactate and it will no longer be acted upon by the rennin. The caloric value is between 300 and 400 calories per litre (284 and 379 per quart) (see also p. 191), being little if any stronger than skimmed milk. Ordinary commercial buttermilk is hardly fit for use in infant-feeding, as it is liable to be contaminated by undesirable germs. It is best made carefully at home in the following manner: A quart or more of either whole milk or skimmed milk, as fresh and clean as possible, is pasteurized and placed in a clean vessel. After the addition of a culture of lactic acid bacilli it is allowed to stand for 18 to 24 hours at a temperature of about 100°F. (37.8°C.) until softly curdled. The Bulgarian bacillus cultures have been most often employed, but other lactic acid germs may be used, including the bacillus acidophilus (see p. 155). Since there are numbers of cultures on the market, many of which have little value for the purpose (Bendick),[2] an active variety should be selected. After standing as directed the milk is then churned energetically for 20 to 30 minutes in a small glass churn, the butter removed, and the buttermilk kept on ice until wanted. Instead of churning buttermilk may be made by using skimmed-milk and beating it thoroughly after souring has taken place. Prepared in either way the living lactic acid germs are still present, and are considered serviceable in some digestive disturbances where it is desired to destroy by their growth the proteolytic bacteria. When only the chemical action of the buttermilk is desired, boiling will destroy the germs. Boiling, however, curdles the casein into large masses, unless vigorous stirring is used to prevent this.

Buttermilk-mixture.—Owing to the low caloric value buttermilk by itself is suitable for infant-feeding only for short periods. To increase its strength the usual custom is to add wheat-flour and cane-sugar to it. The preparation may be made in the following manner somewhat after the proportions recommended by Teixeria de Mattos:[3] 1 level tablespoonful (¼ oz.) of wheat, rice or other flour is rubbed into a paste with 6 to 8 oz. of a quart of buttermilk, and 4½ level tablespoonfuls (2¼ oz.) of granulated sugar are added. This is then mixed with the remainder of the quart and the whole boiled for 25 minutes, with constant stirring. If it is desired to preserve the germs alive, the flour and sugar are boiled in the 8 oz. of buttermilk in a double boiler, and then when cool added to the remaining buttermilk. The buttermilk-mixture prepared in this way gives approximate percentages of fat 1 per cent., carbohydrate 11 per cent., protein 4 per cent., based on König's figures, but varying somewhat with the buttermilk employed. The advantages of the buttermilk-mixture depend upon the relationship of the percentages. It is serviceable in cases where fat is not at all well tolerated and where there is no intolerance for carbohydrate. The caloric value varies between 560 and 660 calories per quart (592 and 697 calories per litre). It is also useful from the fact that the casein has already been coagulated by the acid, and is in a very finely divided state, not capable of being acted upon by rennin.

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[1] Chemie der menschlichen Nahrungs- und Genussmittel, 1903, I, 386.
[2] Jour. Amer. Med. Assoc., 1915, LXIV, 809.
[3] *Loc. cit.*

Lactic-acid Milk.—Lactic-acid milk is a term which might conveniently be limited to whole-milks which have been soured, as in making buttermilk (see p. 153), but which have not had the butter-fat removed by churning or in other ways. It is manifestly a contradiction to speak of "whole-milk buttermilk," since buttermilk contains no butter-fat. Kumiss and Matzoon are of this class. They cannot, of course, be used where a milk-mixture free from fat is desired.

Marriott[1] advocates for athreptic infants the use of undiluted lactic-acid milk to which corn-syrup has been added. The lactic-acid milk is prepared as is buttermilk, but without the churning (p. 154). The corn-syrup used is an ordinary commercial variety. Forty-five volumes of the syrup may be mixed with 55 volumes of water, this mixture containing about 50 per cent. carbohydrate. This thin syrup is added to the lactic-acid milk, and after slight agitation of the mixture by stirring it is put in the refrigerator until it is to be used. It is advisable to begin with a mixture of equal parts of whole lactic-acid milk and fat-free lactic-acid milk (buttermilk) or, in the case of infants convalescing from diarrhea, fat-free lactic-acid milk alone. The proportion of whole lactic-acid milk is then increased until straight lactic-acid milk is fed. The addition of the syrup is then begun; 3 per cent. of carbohydrate being added at first; and if no diarrhea occurs the percentage of carbohydrate is gradually increased up to 10 per cent. or over. The number of ounces of the mixture used should be approximately the same as if breast-milk were being fed.

Another form of lactic-acid milk is that recommended by Shaw and Williams[2] in which the lactic acid is produced by spontaneous souring. Fresh whole milk is poured into a bowl, covered with cheese-cloth and allowed to sour for 2 days in a warm room until it thickens like junket. It is then churned in a small glass butter-churn for several minutes. A heaping tablespoonful of sugar and one of flour for each quart of milk are added and the whole boiled for 5 minutes while stirring. This acid milk is used without dilution. Hamburger[3] had previously stated that milk which had been spontaneously soured by the heat of summer could be utilized by mixing with flour and sugar and then boiling.

Acidophilus Milk.—This has of recent years become a very popular form of lactic-acid milk, both for infants and older subjects. In its preparation fermentation is produced by the bacillus acidophilus in place of the bacillus Bulgaricus. This organism, woich was isolated both by Moro[4] and Finkelstein,[5] is a normal inhabitant of the intestinal tract of man. In health and in the absence of intestinal putrefaction it is found in large numbers in the stools, and may be increased by feeding milk or lactose (Hull and Rettger).[6] If it is desired rapidly to transform the intestinal flora so that the bacillus acidophilus predominates, milk which has been fermented with the organism may be fed. The ingestion of tablets or broth-cultures of the organism is not very effective; certainly not to the same extent as the feeding of acidophilus milk itself, since this contains so many more viable organisms. (See Vol. II, p. 141.) The preparation of acidophilus milk is described by Rettger and Cheplin[1] as follows: Cow's milk is sterilized by heating at 115–120°C. (239–248°F.). A litre (1.056 quarts) of milk should be heated for from about 22 to 24 minutes. After cooling to at least 37°C. (98.6°F.) it is inoculated with pure strains of bacillus acidophilus, and is then kept at a temperature of 35°–37°C.(95°–98.6°F.) for 24 hours. Viable milk-cultures are used as the inoculum, at least 10 c.c. of which are transferred for each litre of milk treated (0.32 fl.oz. per quart).

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[1] Jour. Amer. Med. Assoc., 1919, LXXIII, 1173.
[2] Arch. of Pediat., 1922, XXXIX, 516.
[3] Münch. med. Wochenschr., 1919, XVI, 557.
[4] Wien. klin. Wochenschr., 1900, XIV, 114.
[5] Deutsch. med. Wochenschr., 1900, XXVI, 263.
[6] Jour. Bacteriol., 1917, II, 47.

Acidophilus milk may be used in infancy in the same conditions in which lactic-acid milk, prepared in other ways, would be employed. It is also useful in correcting constipation and in the treatment of colitis (Rettger and Cheplin;[2] Norman and Eggston;[3] Cheplin, Fulmer and Barney;[4] Gompertz and Vorhaus[5]). Feeding acidophilus milk is a valuable adjunct in the treatment of typhoid fever (Torrey[6]) and is indicated in chronic intestinal indigestion. In addition to the favorable action of the organisms in buttermilk, lactic-acid milk, and acidophilus milk, the acid which is present in these milks has an effect in combining with the buffer-substances of the milk (see p. 113).

Lactic=Acid Milk U. S. P.—For the sake of distinguishing clearly, this term may be applied to the food described by Marriott and Davidson,[7] which possesses distinct advantages. Cow's milk is boiled for 5 minutes, thoroughly cooled and the skin removed. U. S. P. lactic acid is now dropped in slowly while stirring, in the proportion of one dram (4 c.c.) to each pint (473.18 c.c.) of milk. This milk keeps well even if not refrigerated. Dilution of the milk is not usually necessary even in new-born or premature infants. Corn-syrup is added to the mixture, using 1 fluid-ounce (30) to the day's mixture for infants up to 2 weeks of age and from 1½ to 2 ounces (52–59) for older infants. If equal amounts of water and the syrup are mixed with the acid before this is added to the milk, the formation of large curds will be avoided.

The reason for the addition of lactic acid as explained by Marriott and Davidson[8] has been discussed on page 113. Sweet cow's milk, by reason of its high buffer-value, is capable of neutralizing a considerable portion of the acid of the gastric juice. This acid has important functions to perform, and any considerable reduction in the amount of it is likely to be associated with digestive disturbances. Milk containing from 0.5 to 0.7 per cent. of lactic acid does not neutralize the acid of the gastric juice to any greater extent than does breast-milk.

Hydrochloric=Acid Milk.—Faber[9] who has studied the pH of the gastric contents also believes in the addition of acid to milk-mixtures. Twenty-five c.c. (0.84 fl.oz.) of tenth-normal hydrochloric acid are added to each 100 c.c. (3.38 fl.oz.) of milk used. The milk is boiled before the addition of the acid. Mixtures with fairly high fat-percentages are necessary to obtain the best results. Marriott and Davidson[1] prefer lactic acid because it is an organic acid, believing that hydrochloric acid can be added safely only in limited amounts, inasmuch as it is an inorganic acid and must be eliminated. This throws a strain on the acid-base regulating mechanism and leads to an acidosis. According to Faber[2] organic as well as inorganic acids deflect metabolism from the normal, and for this reason acid milks should not be used routinely, but only on specific indication.

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[1] Arch. Intern. Med., 1922, XXIX, 357.
[2] *Loc. cit.*
[3] New York Med. Jour., 1922, CXVI, 623.
[4] Jour. Amer. Med. Assoc., 1923, LXXX, 1896.
[5] Med. Rec., 1921, C, 497.
[6] Jour. Infect. Dis., 1915, XVI, 72.
[7] Jour. Amer. Med. Assoc., 1923, LXXXI, 2007.
[8] Amer. Jour. Dis. Child., 1923, XXVI, 542.
[9] Amer. Jour. Dis. Child., 1923, XXVI, 401.

Vinegar-Milk.—Dunham[3] recommends the employment of vinegar instead of other acids for making an acid milk, as a rule adding 1 oz. of vinegar to each 15 oz. of cow's milk. This gives a hydrogen-ion concentration of about pH 4.2.

Lemon-Juice Milk.—Hess and Matzner[4] have found that the addition of lemon-juice to milk in the proportion of 21 c.c. (0.71 fl.oz.) to the quart (22.18 c.c.(0.75 fl.oz.) per litre) of milk will reduce the buffer-action in the same manner as the addition of lactic acid. The pH of the mixture will vary from about 5.54 to 5.30; the latter figure when diluted milk is used instead of whole milk. The lemon-juice is added to the diluent and then stirred into the milk, or it may be added drop by drop to the undiluted milk. The amount of lemon-juice added is also sufficient to act as an antiscorbutic. If desired the yolk of one egg may be added to the quart of mixture, in this way furnishing the antirachitic factor and iron.

Casein-Milk (Eiweiss-Milk. Protein-Milk. Albumin-Milk).—Although not a translation of the German title "Eiweissmilch" applied by Finkelstein and Meyer,[5] the title "Casein-milk" would appear to express well the composition of the food. It is distinctly a casein preparation; not one of all the proteins of the milk in original amount. Its principal purpose is to furnish a diet with a fair amount of fat; a low percentage of sugar, especially lactose; a reduced percentage of salts and of lactalbumin, and a large amount of casein. The percentage-composition[6] averages approximately the figures given in the following table:

TABLE 53.—COMPOSITION OF CASEIN MILK

Fat	2.5 per cent.
Lactose	1.5 per cent.
Protein	3.0 per cent.
Salts	0.5 per cent.

The amount of the fat is somewhat variable. The caloric value is about 450 per litre (426 per quart). A little saccharin may be used for sweetening if necessary. The formula is based on the belief that the sugar and the salts of the food are the most dangerous elements in many diseased conditions. By diminishing the amount of these the fat is rendered more easily digestible. Later, after the digestion of the infant has improved, the percentage of sugar is raised by the addition of a maltose-dextrin combination. Although the truth of the original theories which led to the production of casein-milk seems repeatedly to have been disproved, the fact remains that the preparation is one of very great value, particularly in many diarrhoeal disturbances in infancy.

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[1] *Loc. cit.*
[2] Amer. Jour. Dis. Child., 1926; XXXI, 395
[3] Amer. Jour. Dis. Child., 1925, XXIX, 200.
[4] Jour. Amer. Med. Assoc., 1924, LXXXII, 1604.
[5] Berl. klin. Wochenschr., 1910, XLVII, 1165; Jahrb. f. Kinderheilk., 1910, LXXI, 525, Münch. med. Wochenschr., 1911, LVIII, 340.
[6] Finkelstein and Meyer in Feer's Lehrb. d. Kinderheilk., 1914, 252.

The preparation of casein-milk is rather difficult and requires close attention to details: One quart of whole-milk is heated to about 100°F. (37.8°C.), ½ oz. of liquid rennet or essence of pepsin added, and the milk then allowed to stand at this temperature in a water-bath for half an hour, by which time it will have been curdled. The mass is then put upon a fine cloth and the whey strained off without pressing, about an hour being allowed for this. The curd is then rubbed through a very fine wire sieve, using the bowl of a spoon to do the rubbing, and 1 pint of water being used in the process; and this sieving repeated 4 or 5 times, using the same water and later adding if necessary enough to preserve the original volume. To the pint thus obtained 1 pint of buttermilk is added. The whole is then sterilized by boiling vigorously, and constantly stirring during the process in order to prevent clumping.

Protein-milk may, if it is desired, be reinforced in its percentage of protein. This may be accomplished by using buttermilk, or other fat-free lactic-acid milk, in place of water for the washing of the curds through the sieve; or by using in its preparation more curds than are obtained from a single quart of milk. It seems to us that there is seldom any indication for increasing the amount of protein beyond that usually employed.

Owing to the difficulty in preparation, or as a result of efforts made at improvement, many substitutes have been recommended and some of them produced commercially and put upon the market. There are two classes of these. In one the powdered milks are so prepared that it is only necessary to mix them in the proportion of 1 oz. (about 3½ tablespoonfuls) to 11 ounces of water to obtain approximately the formula of casein-milk as recommended by Finkelstein. In the other the preparation consists only of casein in the form of powdered flour, which is mixed with milk, buttermilk or lactic-acid milk to increase the amount of protein. When these latter casein-preparations are used, considerable latitude is possible in the manipulation of percentages. The following formulæ serve as examples of those we have employed in the Children's Hospital of Philadelphia. In some of them a portion of the milk used is buttermilk or a fat-free lactic-acid milk produced in any way, and another part is whole lactic-acid milk containing the original fat. Good results may often be obtained by using sweet milk in place of buttermilk or lactic-acid fermented milk.

Table 54.—Composition of Various Casein-milk Formulæ

Buttermilk (no fat), ounces	Lactic acid milk (fat present), ounces	Casein-preparation	Dextrin-maltose	Water, ounces
(1) 10	0	6 grams (about 1 scant level tablespoonful)	18 grams (about 3 scant level tablespoonfuls)	10
(2) 5	5	6 grams (about 1 scant level tablespoonful)	18 grams (about 3 scant level tablespoonfuls)	10
(3)	10	6 grams (about 1 scant level tablespoonful)	18 grams (about 3 scant level tablespoonfuls)	10
(4)	15	6 grams (about 1 scant level tablespoonful)	18 grams (about 3 scant level tablespoonfuls)	5

These give the following approximate calculated percentages and caloric values:

	Fat	Carbohydrate	Protein	Calories per fluid ounce
(1)	0.15	5.25	2.75	10.79
(2)	1.10	5.25	2.75	13.26
(3)	2.00	5.25	2.75	15.60
(4)	3.00	6.40	3.60	20.80

These calculations are based upon the same approximate percentage-strengths employed in using ordinary home-modifications from top-milk (p. 141), and are open to the same criticism of lack of absolute accuracy. The variation is, however, insignificant. The percentage of protein will also vary slightly with the casein-preparation used. The following table shows the relative composition of some of those on the market.

TABLE 55.—ANALYSES OF COMMERCIAL CASEIN MILKS AND CASEIN PREPARATIONS[1]

	Protein, per cent.	Fat, per cent.	Lactose, per cent.	Salts, per cent.	Ash, per cent.	Moisture, per cent.	Lactic acid, per cent.
Hoos' albumin milk	38.5	30.2	21.5	......	5.0	2.0	3.0
Mead-Johnson's powdered protein milk	37.0	30.9	19.1	......	4.6	....	3.0
Merrill-Soule powdered protein milk	38.0	27.0	24.0	......	5.0	3.0	3.0
Casec	89.0	1.6	....	......	5.28	4.12	
Larosan	88.7	2.3	....	......	4.0	5.0	
Protolac	83.65	0.6	....	3.23+	....	4.85	

CHAPTER V

FOODS OTHER THAN MILK

The practise of giving healthy infants articles other than milk, as a regular part of their diet in the latter half of the 1st year, has become of late years very popular with many physicians. It will be discussed in the next chapters (pp. 167, 181). In the present chapter will be considered merely the character and uses of certain foods, independently of any special age-limit for their employment.

PROTEIN-FOODS

It happens constantly that some animal substance other than milk is needed as a temporary substitute during illness in the 1st year; or as a more permanent article of diet after this period. The following may be mentioned:

Albumen-water.—This consists of the white of one hen's egg, averaging from 55 to 60 grams (Pfund)[2] (1.9 to 2 fl.oz.) stirred in enough cool water to make 8 oz., and strained if necessary. It is an excellent temporary substitute for milk in acute cases of failure of digestion, but a very weak one. It is also at times a valuable addition to cream-and-water mixtures, or cream-and-whey mixtures, when it is desired to increase the protein of the food by the use of some substance other than casein. The possibility of it being directly absorbed as a foreign albumin when intestinal disturbance is present is not to be forgotten. A sensitization to egg might be produced in this way. According to Hammarsten[1] white of egg contains 10 to 13 per cent. of protein, a trace of fat, 0.7 per cent. of salts, and 85 to 88 per cent. of water. When mixed with enough water to make 8 oz. a protein-percentage of about 1.3 is obtained. A certain proportion of the egg-white, however, appears in the water in the form of shreddy material, and requires to be removed by straining. The actual protein-percentage may thus be reduced considerably. Albumen-water may be given cool or slightly warm, with or without sugar or with a pinch of salt, and from a glass or bottle according to the taste and fancy of the infant. It may be flavored with a few drops of lemon or orange juice or with whiskey. According to Stutzer[2] 1 lb. of lean beef equals 15 eggs in protein-constituent.

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[1] Analyses from manufacturer.

[2] Zeitschr. f. Hyg., 1900, XXXV, 444.

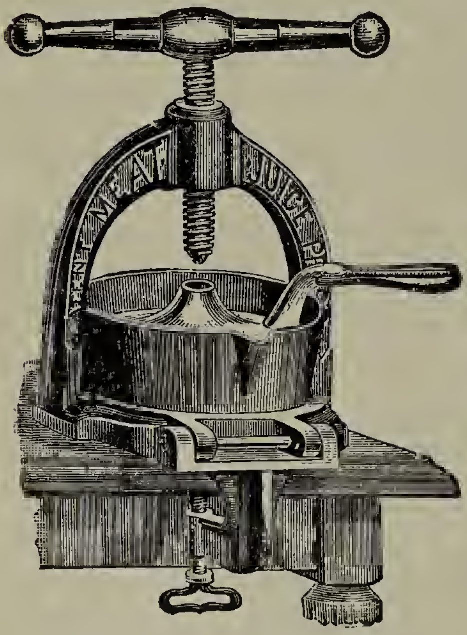

Fig. 25.—Meat-Press.

Fresh Beef-juice.—This may best be prepared in either of two methods:

1. Season with salt and very slightly broil a piece of steak free from fat, then cut it into small pieces and express the juice with a meat-press (Fig. 25). A lemon-squeezer may be used instead, but this method is more wasteful. A pound of beef will make about 2 or 3 fl.oz. of juice. The juice must be kept on ice until needed. It may then be warmed slightly or given cold with a spoon. Some infants prefer it with the addition of sugar.

2. A pound of minced steak is put into 6 or 8 oz. of water and salted slightly. This is allowed to stand on ice over night. It is then squeezed out well with the meat-press or is strained through muslin by twisting it tightly. Juice thus obtained is somewhat thinner than by the first method, but is decidedly larger in quantity.

The protein of beef-juice varies in amount, depending upon the method employed in obtaining it. According to the statistics given by Hutchison[3] it varies from 2 to 7 per cent. Its fat-percentage is low. Beef-juice is of value as a condensed, stimulating food for infants who have difficulty in digesting the protein of milk, but its caloric value is low and it is not sufficient for an exclusive diet in the quantity which the digestion will safely tolerate. Its administration may be alternated with albumen-water or cereal decoctions in cases where it is desired to avoid milk for a time. The daily amount should be 1 or 2 teaspoonfuls at first and gradually increased to 3 or 4 oz. Beef-juice is also useful in cases of anemia or deficient nutrition as an adjuvant to the milk-diet, and may then be added in quantities of 2 or 3 teaspoonfuls to each bottle, or, preferably, given separately in 1 or 2 portions in the twenty-four hours between the regular feedings. In the 2d year of life beef-juice may well constitute a part of the daily food for healthy children.

Minced Underdone Beef.—A thick piece of underdone steak, as free from fat and tendon as possible, is scraped, grated, or minced very

[1] Phys. Chemie, 1904, 435.

[2] Centralbl. f. allg. Gesundheitspflege, 1882, I, 179.

[3] Food and Dietetics, 1917, 100.

fine; pounded in a mortar until it is reduced to a pulp, and seasoned with salt. For infants it may now be rubbed up with a little water until it is of the consistency of thick cream, and fed from a spoon, giving 3 or 4 teaspoonfuls in the course of the day.

Scraped beef is a very concentrated form of nitrogenous food, sometimes useful even in subjects less than 1 year of age. In the 2d year 1 or 2 tablespoonfuls of the scraped meat without water may be given daily when an increase of casein is not desirable. I have more than once seen tape-worm develop from the use of raw beef given in this way, but the danger may be avoided entirely by the slight broiling referred to.

Meat=Broths.—Foods of this class are of practically no value as actual nourishment, if of the class of "clear broth." They are sometimes of benefit during the 1st year as a temporary substitute for milk-mixtures, and in the 2d year may be one of the ordinary articles of diet. The principles and method of preparation are the same for all, and the following description for making beef-broth applies to the others: 1 pound of lean meat is cut into small pieces, and these, together with portions of the cracked bones, put into 1 pt. of cold water. This is heated very slowly, allowing it to simmer for 3 or 4 hours; replacing the water as it evaporates; and then strained, cooled and the fat removed. The strainer used should be coarse enough to allow of the finely divided muscle-fibre passing through, as this increases the nutritive value very decidedly. The addition of a cereal flour also adds to the value as a food, and makes it a very serviceable article of diet in the 2d year, or for temporary use to replace milk in digestive disturbances in the 1st year. (See also p. 185.)

Beef=Tea.—This may be prepared as follows: (1) Scrape or cut fine 1 pound of lean meat and place in 1 pint of cold water in a jar. Let it stand in the cold for 1 hour, stirring occasionally Then heat the jar in a saucepan of water at not over 167°F. (75°C.) for another hour, stirring occasionally. It should then be raised to the boiling point for a moment, strained, cooled and the fat removed. Warm slightly and season before giving it. In place of straining the liquid may be simply poured off. To this should be added the additional liquid obtained by firmly pressing the pieces of meat which remain. (2) Another more rapid method is the following: Place the pound of meat in ½ pint of boiling water. Keep this gently warm for 10 minutes; strain; cool rapidly and remove the fat.

Beef-tea is distinguished from beef-broth particularly by the considerable amount of gelatin contained in the latter. This is derived from the prolonged boiling of the bones and of the connective tissue. Beef-tea has little nutritive value, and practically none at all if sufficient heat has been used to coagulate the protein, and if this has been removed by straining. If the fluid is not strained the food-value is decidedly increased.

Veal=Tea.—This preparation, formerly much in vogue, is very similar in its composition and in its strength to beef-tea. It may be made as follows: Cut fine 1 pound of veal as free from fat as possible, put into 1½ pints of cool water; let stand in the cold for 1 hour; then keep warm without boiling for 3 or 4 hours; strain; let cool and skim off the fat.

Gelatin.—About 40 grains of gelatin are soaked for a short time in cold water, and then dissolved with stirring in ½ pint of boiling water. Gelatin as an element of milk-mixtures was very popular in former years. Its mechanical action in the food is very similar to that of barley-water. As a colloidal substance it tends also to prevent vomiting and the formation of tough curds, and adds to the digestibility of the food. Chemically it possesses but little food-value.

Soy Bean Flour.—This substance may be properly classed among protein-foods serviceable for temporary use when, for any reason, the protein of milk is not desired. According to Ruhräh[1] it contains approximately 35 to 40 per cent. of protein, 4 per cent. of fibre, 9 per cent. of starch, sugar and dextrin, and 18 to 19 per cent. of fat. Made into a flour the protein is over 44 per cent.

AMYLACEOUS FOODS

The employment of starchy foods in the 1st year has already repeatedly been referred to. (See pp. 125, 131, 142, 146, etc.) Although the power of digesting starch is present in early infancy, there is no need for the administration of it to the healthy normal artificially fed infant during the early part of the 1st year. The addition of starch to the diet in the latter part of the year is usually advantageous in order to accustom the child to this food. In the case of illness, however, weak or stronger amylaceous additions or substitutions are often of the greatest value, and this is particularly true when there is difficulty in the digestion of sugar. Starch is also used to prevent the casein from forming tough coagula, approximately 0.7 per cent. of starch in the milk mixture being the best amount to accomplish this (White).[2] For infants in the 1st year the amylaceous food is generally either a cereal flour or arrowroot, and is usually given in the form of a decoction of various strengths. After this period cereal gruels become a recognized part of the diet. (See also p. 182.)

The Weight of Various Carbohydrate Substances. (See also p. 137).—In the making of amylaceous decoctions in the home, measuring by bulk is usually the only method which can be employed; but for better knowledge it is necessary to know the weight also, in order to estimate the percentages obtained. Tablespoons are the measure oftenest used, but as these vary so much in size, it is better to employ the Chapin dipper or other ounce-measure. I have made a series of weighings, with the results shown in the table which follows. They correspond

Table 56.—Approximate Weights of Grains, Flours and Sugars
(Measured by Tablespoon and by Fl.-oz. Dipper)

1 Level tablespoonful of barley or other flour weighs about ¼ oz. Av.
1 Level dipper of barley or other flour weighs about ½ oz. Av.
1 Level tablespoonful of Bermuda arrowroot weighs about ⅖ oz. Av.
1 Level dipper of Bermuda arrowroot weighs about ⅘ oz. Av.
1 Level tablespoonful of pearl barley weighs about ½ oz. Av.
1 Level dipper of pearl barley weighs about ¾ oz. Av.
1 Level tablespoonful of rice weighs about ½ oz. Av.
1 Level dipper of rice weighs about ⅘ oz. Av.
1 Level tablespoonful of rolled oats weighs about ⅙ oz. Av.
1 Level dipper of rolled oats weighs about ⅓ oz. Av.
1 Level tablespoonful of lactose weighs about ⅓ oz. Av.
1 Level dipper of lactose weighs about ¾ oz. Av.
1 Level tablespoonful of dextrin-maltose weighs about ¼ oz. Av.
1 Level dipper of dextrin-maltose weighs about ½ oz. Av.
1 Level tablespoonful of saccharose weighs about ½ oz. Av.
1 Level dipper of saccharose weighs about 1 oz. Av.
1 Level tablespoonful of casein-flour weighs about ¼ oz. Av.
1 Level dipper full of casein-flour weighs about ½ oz. Av.

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[1] Arch. of Pediat., 1909, XXVI, 496; Amer. Jour. Med. Sci., 1915, CL, 502.

[2] Jour. of Bost. Soc. of Med. Imp., 1900, V, 125.

fairly closely with those determined by Chapin[1] and with those obtained at the Children's Hospital of Philadelphia (Mitchell). The figures can be only average ones, especially as regards the weight of the tablespoonful. Barley and oat flours are slightly lighter, and rice and wheat flours slightly heavier than the figures given. In measuring, the tablespoon or the dipper should be filled from a smaller spoon, tapped sharply 2 or 3 times, and the excess scraped off with a case-knife. Too much tapping settles the contents too greatly. Casein-flour is included as a convenience.

Strength of Cereal Gruels.—It is of distinct advantage to know with approximate accuracy the actual amount of the ingredients contained in the various cereal gruels employed. The matter has been studied by Chapin[2] and others. The following table gives the approximate percentage-formulae of some of these gruels.[3] (Table 57.)

The fat and mineral matter are present in such small amounts that they may be ignored for practical purposes, and it is hardly necessary to take the protein into consideration. Simple gruels cannot be made stronger than about 2 oz. of the flour to the quart. If more concentrated preparations are desired they must be dextrinized (p. 165).

Various Starchy Decoctions. Barley-water.—Barley-water is made either from pearl-barley or from barley-flour. With the former 2 level tablespoonfuls of the barley, previously well washed for some time in cold water, are placed in 1 quart of water, let simmer slowly until the liquid is reduced to 1 pint in volume, and then strained. This should be quite fluid in consistency when slightly warmed. In place of the whole grain 1 level tablespoonful of barley-flour may be boiled slowly in 1 pint of water for 15 minutes, with stirring, and then strained, replacing the water which has boiled away. This has the advantage that it can be made much more rapidly and conveniently. As with all amylaceous decoctions it should be prepared fresh daily and kept on ice, as it sours readily.

Barley-water is a very commonly used temporary substitute for milk in cases of acute indigestion where abstinence from food is the best cure. Its nutritive power being extremely slight, it is given in this case not so much for nourishment as for the allaying of hunger at the time. When used as a diluent of milk-mixture it is supposed to have a somewhat constipating action, but this is by no means always the case.

Barley-jelly is the term applied to a thicker barley-decoction which jellies firmly when cool. It is made by using 4 level tablespoonfuls of pearl-barley to the quart of water, or 3 of barley-flour to the pint. A double boiler may be used to advantage to prevent burning.

Oatmeal-water.—This may be made in the following manner: Put 2 level tablespoonfuls of oatmeal into 1 pint of water. Let simmer for 2 hours or more, replacing the water as it evaporates; strain. An *oatmeal-jelly* may be made in a way similar to that employed for making barley-jelly, using 4 level tablespoonfuls to the pint of water, and cooking as for oatmeal-water, although in a double boiler. Oatmeal-water or oatmeal-jelly is used with milk as are the analogous preparations of barley. It is, however, distinctly laxative to many children, and is often a useful addition where there is constipation. In many cases

[1] Med. Rec., 1905, LXVII, 246.

[2] *Loc. cit.*

[3] The analyses are those published by the Cereo Company of Tappan, New York, and based on gruels made of flours prepared by them.

Table 57.—Percentage Composition of Gruel

	Barley		Legume		Oat		Wheat	
	Proteids, per cent.	Carbo-hydrates, per cent.	Proteids, per cent.	Carbo-hydrates, per cent.	Proteids, per cent.	Carbo-hydrates, per cent.	Proteids per cent.	Carbo-hydrates, per cent.
1 Level tablespoonful flour (¼ oz. Av.) to quart of gruel	0.12	0.60	0.19	0.53	0.12	0.60	0.10	0.62
2 Level tablespoonfuls flour (½ oz. Av.) to quart of gruel	0.24	1.20	0.39	1.06	0.24	1.20	0.20	1.25
3 Level tablespoonfuls flour (¾ oz. Av.) to quart of gruel	0.36	1.80	0.58	1.59	0.36	1.80	0.30	1.88
4 Level tablespoonfuls flour (1 oz. Av.) to quart of gruel	0.48	2.40	0.78	2.12	0.48	2.40	0.40	2.50
8 Level tablespoonfuls flour (2 oz. Av.) to quart of gruel	0.96	4.80	1.56	4.24	0.96	4.80	0.80	5.00
12 Level tablespoonfuls flour (3 oz. Av.) to quart of gruel	1.44	7.20	2.34	6.36	1.44	7.20	1.20	7.50
16 Level tablespoonfuls flour (4 oz. Av.) to quart of gruel	1.92	9.60	3.12	8.48	1.92	9.60	1.60	10.00
20 Level tablespoonfuls flour (5 oz. Av.) to quart of gruel	2.40	12.00	3.90	10.60	2.40	12.00	2.00	12.50
24 Level tablespoonfuls flour (6 oz. Av.) to quart of gruel	2.88	14.40	4.68	12.72	2.88	14.40	2.40	15.00
28 Level tablespoonfuls flour (7 oz. Av.) to quart of gruel	3.36	16.80	5.46	14.84	3.36	16.80	2.80	17.50
32 Level tablespoonfuls flour (8 oz. Av.) to quart of gruel	3.84	19.20	6.24	16.96	3.84	19.20	3.20	20.00

oatmeal disagrees decidedly. If amylaceous food is to be added during the 1st year, some other than oatmeal should generally be first tried.

Rice-water.—The method employed is the adding of 2 level tablespoonfuls of washed rice to 1 quart of water, boiling to 1 pint and straining, and then mixing with the food in the same proportions as with barley-water. It is supposed to have a constipating effect, and is often used when there is a tendency to diarrhea. Its actual value for such a purpose is doubtful. In place of the rice-grain, rice-flour may be employed, using 1 level tablespoonful to 1 pint of water, boiling for 15 minutes and straining. Rice is richer in carbohydrate and poorer in protein than the cereals previously mentioned.

Arrowroot.—This is a serviceable food when a starchy addition is required. It is practically a pure starch, containing almost no protein. It may be given in the form of *arrowroot-water*, which is made by adding 1½ level tablespoonfuls of Bermuda arrowroot to 1 pint of water and boiling for 5 or 10 minutes. *Arrowroot-jelly* is made by using 4 level tablespoonfuls of arrowroot to 1 pint of water, and boiling for a few minutes in a double boiler.

Bean-flour.—This has been employed in the form of a gruel when, with the addition of carbohydrate, it is desired to increase the protein-content of the food decidedly by the use of a vegetable product rich in this substance. The Soy bean (p. 162), recommended for infant-feeding especially by Ruhräh,[1] contains so little starch that it cannot be properly classified among amylaceous foods.

Dextrinized (Malted) Starch.—The addition of malt-extract to boiled starch rapidly dissolves it, transforming it into dextrin and maltose and intermediate substances. Starch, as such, ceases to be present if the dextrinizing is complete. Treated in this way it becomes very absorbable and, added to the milk-mixture, often aids greatly in nourishing infants with feeble powers of digestion or assimilation of fat or of lactose. The various commercial maltose-dextrin preparations on the market are of this class. (See pp. 131, 175.) As made at home, 1 fluidrachm of a powerful diastatic malt-extract will transform the starch of 10 oz. of a 10 per cent. amylaceous decoction. According to investigations made at the Children's Hospital of Philadelphia, ⅓ of the starch of such a decoction of arrowroot was converted into a copper-reducing substance in 15 minutes at a temperature of 140°F. (60°C.), and a longer time or a larger amount of the extract would convert all or the greater portion of it. 7 grains of potassium carbonate should then be added to each ounce of the mixture. The dextrinized preparation may replace in part the water of the milk-mixture. The malt-extract should be added to the cereal jelly while the latter is still warm, about 120° to 150°F. (48.9 to 65.5°C.) and maintained at this temperature, with occasional stirring, for 20 to 30 minutes. Dextrinized starch produced in this way—and the same is true of the commercial maltose-dextrin preparations—has an action different from that of the pure sugars. These latter become very promptly assimilated, while the dextrin of the maltose-dextrin possesses a colloidal action resembling that of starch, and is also slower in being transformed and consequently in being absorbed. The relative amount of dextrin and maltose present depends upon the temperature employed. (See p. 131.) If this is below 55°C. (131°F.), there is more maltose resulting; if above 63°C. (145.4°F.), more dextrin. If above 75°C.

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[1] Arch. of Pediat., 1909, XXVI, 496; Amer. Jour. Med. Sci., 1915, CL, 502.

(167°F.) the diastatic ferment is destroyed (Morse and Talbot).[1] The strength of milk-mixtures is very considerably increased in the percentage of carbohydrate when dextrinized gruels are employed. The degree of increase can be determined approximately by estimating the amount of amylaceous flour employed (see pp. 163, 164), ignoring the content of the malt-extract if but little of this has been used.

Malt-soup.—A sharp distinction is to be drawn between the food in which malt-soup takes a part, and that to which simple maltose-dextrin preparations or completely dextrinized gruel has been added. With the latter no starch is present, while with malt-soup there is a very decided proportion of this intentionally remaining unconverted. The dextrinized preparation made at home as just described is a malt-soup if the process is not continued to the complete converting of the starch. The preparation was first made and urged by Liebig,[2] but was later modified by Keller,[3] and came rapidly into prominence as a serviceable form of nourishment for a certain class of sick infants. Its value rests on the fact that a large percentage of unconverted starch, together with maltose and dextrin, is present in the food. The method of preparation as commonly advised is as follows:

1. 1¾ oz. Av. (6½ level tablespoonfuls) of wheat-flour are mixed with 11 fl.oz. of cold cow's milk and rubbed through a sieve.

2. 2½ fl.oz. of malt-soup extract are added to 22 fl.oz. of warm water.

The two are then added one to the other and the whole heated slowly to boiling, with constant stirring, and enough water finally added to replace that which has evaporated. The malt-soup extract should contain 7 grains of carbonate of potash to the fluidounce, the object of this being to alkalinize it and thus to limit its diastatic action, as also to prevent the development of any acidosis in the infant. The caloric value of this mixture is about 625 calories per quart (660 per litre). Most of the malt-soup extracts on the market already contain the alkali; but any which are non-diastatic may be used if potassium carbonate is added in the amount mentioned. Diastatic action may be arrested in malt-extract by heating to 165°F. (73.8°C.) for 2 hours or to 200°F. (93.3°C.) for 15 minutes. The formula given produces percentages of approximately fat 1.33, carbohydrate 11.4, protein 1.58 varying somewhat with the preparation of malt-soup extract used. It is not, however, necessary to use these amounts. The experiments conducted at the Children's Hospital of Philadelphia evolved the following formulæ which will be found convenient.

Table 58.—Percentage-strengths Various Malt-soup Mixtures

			Per cent.	Cal. per oz.
(a)	Wheat-flour, ½ oz. (2 level tablespoonfuls) Skimmed milk, 10 oz. Malt-soup-extract, 1 oz. Water to make 20 oz.	Fat	= 0.5	13.96
		Carbohydrate	= 7.5	
		Protein	= 2.24	
(b)	Wheat-flour, ½ oz. (2 level tablespoonfuls) Whole-milk, 5 oz. Skimmed milk, 5 oz. Malt-soup-extract, 1 oz. Water to make 20 oz.	Fat	= 1.0	15.13
		Carbohydrate	= 7.4	
		Protein	= 2.24	
(c)	Wheat-flour, ½ oz. (2 level tablespoonfuls) Milk, 10 oz. Malt-soup-extract, 1 oz. Water to make 20 oz.	Fat	= 2.0	17.73
		Carbohydrate	= 7.4	
		Protein	= 2.24	

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[1] Diseases of Nutrition and Infant Feeding, 1915, 195.

[2] Suppe für Säuglinge, 1865.

[3] Malzsuppe, eine Nahrung für magendarmkranke Säuglinge, 1898.

Table 58.—Percentage-strengths Various Malt-soup Mixtures (*Continued*)

		Per cent.	Cal. per oz.
(*d*) Wheat-flour, 1 oz. (4 level tablespoonfuls)	Fat	= 2.0	22.88
Milk, 10 oz.	Carbohydrate	= 11.0	
Malt-soup-extract 1½ oz.	Protein	= 2.6	
Water to make 20 oz.			
(*e*) Wheat-flour, 1½ oz. (6 level tablespoonfuls)	Fat	= 2.1	26.81
Milk, 10 oz.	Carbohydrate	= 13.6	
Malt-soup-extract, 1¾ oz.	Protein	= 2.9	
Water to make 20 oz.			

In these calculations the same approximate percentages were employed as adopted for the making of ordinary home-modifications from top-milk. (See p. 141.) Dry malt-soup extract, with or without the addition of wheat-flour, may be obtained on the market. These forms are convenient to prepare, especially those to which flour has been added. For analyses of malt-preparations see p. 131.

Flour-ball.—Flour-ball was formerly much employed as an addition to milk when a cereal was required. It is made by tying a pound of flour tightly in a bag and boiling for 10 hours. When cold it is taken from the bag and completely dried with heat. The outer coating is then removed, and the remaining inner portion grated. 1 or 2 teaspoonfuls are added to each bottle. The advantage claimed for it was that the flour was partially dextrinized and thus rendered more soluble, requiring less cooking. Dextrinized cereal flour can, however, be produced much more easily in the manner already described. Flour-ball in a prepared state may be obtained on the market under the trade name of Cerena.

CHAPTER VI

SPECIAL NAMED MIXTURES AND PROPRIETARY FOODS

SPECIAL MIXTURES WITH PERSONAL NAMES

There have been recommended a very large number of special mixtures to which are often attached the names of the physicians first describing them. A few of these are mentioned below on account of the frequency with which the names are, or have been, met with in medical literature, not because of a desire especially to advocate them. Few of them have been employed to any extent in the United States, and some are now probably entirely out of vogue. It must be remembered that modern scientific pediatrics is opposed to the use of any one preparation as a routine feeding. Rather must the mixture be modified to meet the needs of the case. Some of the preparations have indeed been manufactured and sold commercially, and would be more properly included in the next section.

Meigs Gelatin-Food.—This was a formula found very useful years ago by J. F. Meigs of Philadelphia, and mentioned here because the title is still widely known. It consisted of 1 scruple of gelatin dissolved in 8 oz. of boiled water to which was then added 1 teaspoonful of arrowroot with milk and cream in varying proportions, depending upon the age and digestive power of the child (Meigs and Pepper).[1]

Biedert's Cream-Mixtures (Rahmgemenge).[2]—These consist of modified milks constructed by mixing certain proportions of cream with milk, water, and milk-sugar, thus producing a series of formulæ. The principle is thoroughly scientific and the food was the prototype of many milk-modifications later made in Germany, as well as of all percentage-feeding combinations. The chief objection to the employment of these mixtures is that, according to the formulæ and the percentages given, the altering of the percentage of one ingredient without affecting that of another is not easy. Later a condensed modified cream-mixture (Ramogen) prepared according to Biedert's formula, was placed on the market. It will be found mentioned under Commercial Foods (p. 178).

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[1] Diseases of Children, 1877, 332.

[2] Biedert, Die Kinderernährung im Säuglingsalter, 1900, 189.

Gärtner's Mother=Milk (Fatty Milk).[1]—This is made by centrifugating diluted milk and adding to the richer portion milk-sugar and an alkali. The mixture is then sterilized. The food is in reality a modified milk. It is supposed to possess the same percentages of fat, sugar and protein as are contained in average human milk. It is subject to the disadvantage of all such mixtures, that the relationships of the ingredients to each other are fixed. As the food soon became a commercial one its analysis will be given later (p. 178).

Von Dungern's Renneted Milk.[2]—The milk is treated with rennet and the coagulated mass finely divided by shaking. It is claimed that it is rendered more digestible in this way, since the formation of large curded masses in the stomach is prevented. The rennet used is contained in a preparation with the trade name of Pegnin.

Szekely's Casein=free Milk.[3]—Skimmed milk is heated at about 60°C. (140°F.) in closed vessels under pressure and subjected to liquid carbonic acid which largely precipitates the casein. The whey remaining is mixed with cream and sugar.

Voltmer=Lahrmann's Pancreatized Milk.[4]—This is merely a pancreatized mixture of cream, milk, water and sugar, with potassium carbonate in varying amounts, to which later phosphoric acid is added.

Backhaus' Milk.[5]—This consists of fat-free milk treated with rennet. In this way a portion of the casein is coagulated and then removed. To the whey remaining milk-sugar and cream are then added. More recently maltose and dextrin were used. The food later became a commercial one, three different strengths being sold (p. 178). The preparations are very similar in percentages to those obtained by Biedert's formulæ.

Monti's Whey=Milk.[6]—The food consists in varying mixtures of whey and whole milk rich in fat. In this way the lactalbumin is increased in amount and the casein relatively diminished.

Steffen's Veal=Broth and Milk[7] is a mixture of 100 grams (3.4 fl.oz.) each of veal-broth and milk to which are added 1 teaspoonful (3.9 c.c.) of cream and 3.8 grams (0.13 fl.oz.) of milk-sugar.

Hesse=Pfund's Infant's Food.[8]—Water and cream are mixed in certain proportions and to them is added a sterilized powder, consisting of hen's eggs and milk-sugar with a small quantity of a salt of iron. The amounts of the non-coagulable protein, fat, and sugar are thus increased.

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[1] Wien. med. Wochenschr., 1894, XLIV, 1870.

[2] Münch. med. Wochenschr., 1900, XLVII, 1661.

[3] Wien med. Wochenschr., 1905, XVIII, 341.

[4] Ref. Raudnitz, in Pfaundler und Schlossmann, Handb. der Kinderheilk., 1906, I, 1311.

[5] Berl. klin. Wochenschr., 1895, XXXII, 561; 589. Berl. Molkereiztg., 1905, No 42. Ref. Raudnitz, Monatsschr. f. Kinderheilk., 1906, IV, 583.

[6] Kinderheilkunde, 1899, I, 158.

[7] Jahrb. f. Kinderheilk., 1895, XL, 421.

[8] Arch. für Kinderheilk., 1898, XXIV, 226; 1903, XXXVI, 407.

Vigier's Humanized Milk.[1]—The method of preparation consists in first dividing a quantity of milk into two equal portions. On the first half the cream was allowed to rise and was removed, and from the fat-free remainder whey was prepared. This and the cream were then added to the second half of the milk. The resulting mixture was to some extent like that of some of Biedert's cream-mixtures, but the percentages were inferior.

Lehndorff and Zak's Dialyzed Milk.[2]—This preparation was especially recommended for cases where there was indigestion of sugar. It is made by placing about a pint of milk in a parchment bag and suspending this in a number of quarts of water, renewing every hour. The sugar and salts pass to a large extent through the parchment.

Homogenized Milk. (Raudnitz).[3]—The milk is minutely divided by atomizing under high pressure. This renders the fat-globules exceedingly small. The cream will no longer rise and the coagulum with acid resembles that of human milk. Homogenization is also employed in the synthetic milk advocated by Gerstenberger (see p. 171). Homogenized olive oil added to fat-free milk has been used by Ladd.[4]

Feer's Milk-Preparation.[5]—This is composed of whole milk 500 grams (16.9 fl.oz.), cream (20 per cent.) 50 grams (1.7 fl.oz.), Soxhlet's Nährzucker 10 to 50 grams (0.35 to 1.8 oz. Av.), plasmon 15 grams (0.53 oz. Av.), water 600 grams (20.29 fl.oz.). This gives a preparation consisting of fat 2.3 per cent., sugar 6.2 per cent., protein 2.6 per cent., salts 0.44 per cent. The purpose of the food was to render the fat better tolerated by the removal of part of the whey. The lack of protein caused by the dilution is made up by the plasmon.

Schloss' Modified Milk.[6]—This is a cream, whole-milk and water mixture to which Soxhlet's Nährzucker, mondamin, and nutrose or plasmon have been added.

Friedenthal's Milk.[7]—The principle of the Friedenthal milk is based especially on an effort to render the mineral matter of the food similar to that of human milk. An artificial human-milk serum is made by dissolving in water the required salts. To this white of egg and powdered casein are added, and the mixture used as the foundation for the infant's food.

Czerny-Kleinschmidt Butter-flour Mixture.—Excellent results from the use of this food were originally reported by Czerny and Kleinschmidt.[8] The mixture has been widely used in Europe, where various modifications of it have been proposed; especially to be noted being that of Moro[9] (see p. 149). Our[10] own experiences with it have been so favorable that they have been reported, and a number of articles have since appeared in this country. The principle of the butter-flour mixture, as stated by Czerny and Kleinschmidt, is to supply a food in which boiled butter,

[1] Société de therap., 1893, Jan. 25. Ref. Marfan, Traité de l'allaitement, 1903, 439.

[2] Wien. med. Wochenschr., 1910, LX, 1930.

[3] Pfaundler u. Schlossmann, Handb. d. Kinderheilk., 1906, I, 1, 310.

[4] Trans. Amer. Pediat. Soc., 1915, XXVII, 117.

[5] Jahrb. f. Kinderheilk., 1913, LXXVIII, 1.

[6] Ueber Säuglingsernährung, 1912.

[7] Zentralbl. f. Physiol., 1910, XXIV, 687.

[8] Jahrb. f. Kinderheilk., 1918, LXXXVII, 1.

[9] Monatsschr. f. Kinderheilk., 1920, XVIII, 97.

[10] New York Med. Jour., 1921, CXIV, 137.

because of its low content of volatile fatty acids, is used to raise the fat-percentage to about that of average breast-milk. Carbohydrate is also high, and part of it is as starch, but the protein is reduced to approximately that of breast-milk. In the actual process of preparation, 20 grams (2 level tablespoonfuls) of butter are placed in a pan and heated over a gentle fire until foaming takes place, and until any odor of volatile fatty acids present shall have disappeared. This requires from 3 to 5 minutes. Twenty grams (2½ level tablespoonfuls) of fine wheat-flour are then added and the mixture again boiled over a gentle fire with constant stirring until the mass becomes thin and of a brownish color (4 or 5 minutes). Now 300 grams (10 fl.oz.) of warm water and 15 grams (1½ level tablespoonfuls) of sugar are added. The whole is again boiled and rubbed through a fine sieve. This may conveniently be called the "stock solution." For use it is mixed with the desired amount of previously boiled, cooled milk, and the whole kept cold until needed for use. The addition of salt is not necessary, as this is contained in the butter. For children under 3000 grams (6.61 lbs.) in weight ⅓ milk is added to ⅔ of the butter-flour stock. For those of 3000 grams (6.61 lbs.) or over ⅖ of milk and ⅗ of butter-flour stock are employed. Not more than 200 grams to the kilogram of body-weight (*i.e.*, about 3 fluid-ounces to the pound) should be given daily, and usually smaller amounts than this are required, owing to the high caloric value of the food. The strength of the food may be diminished by reducing the amounts of butter, flour, or sugar, but in any manipulation the amounts of butter and flour should remain equal. Kleinschmidt[1] in a later paper recommended that, in the case of premature infants, the amounts of butter and of flour should be reduced each to 15 grams (1½ level tablespoonfuls). The percentages and caloric value of butter-flour as usually employed follow:

	Fat, per cent.	Carbohyd., per cent.	Protein, per cent.	Cal. per oz.
⅔ stock ⅓ milk	4.6	8.2	1.5	24.6
⅗ stock ⅖ milk	4.6	7.8	1.7	24.3

Butter-flour mixtures may be used in malnutrition, except in the severe stages (athrepsia). Fever and diarrhoea contraindicate its use. A number of our premature infants have done exceedingly well when fed on the weaker dilutions of butter-flour mixture. Kleinschmidt[2] has also recommended that after acute dyspeptic conditions, where the advisability of using the butter-flour food is uncertain, a trial be first made of buttermilk, which can be gradually replaced by the butter-flour food or have increasing amounts of butter-flour mixed with it.

Almond-Meal Milk.—Almonds have been used in the preparation of some proprietary foods, among them, for instance, Lahmann's Vegetable Milk (see p. 178). As a home-made preparation Chapin[3] tried a combination of 100 grams (3.52 oz.) of finely ground almonds, covered with 200 c.c. (6.76 fl.oz.) of water; allowing this to stand on ice over night; pressing out the liquid and adding 300 c.c. (10.14 fl.oz.) more water. The percentage-composition of the food obtained equals fat

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[1] Berl. klin. Wochenschr., 1919, LVI, 673.

[2] Monatsschr. f. Kinderheilk., 1921, XIX, 369

[3] Arch. of Pediat., 1919, XXXVI, 28.

7.02, carbohydrate 1.2, protein 4.4, ash 0.55, water 86.74. A very similar preparation had been used with success by Raudnitz.[1] Moll[2] likewise employed almond-meal, combining it with whey. He has used it extensively and successfully as a temporary food in acute dyspeptic conditions, when it is desired to eliminate casein entirely. The whey is prepared by adding 4 to 5 grams (0.14 to 0.17 oz.) of calcium lactate to 1 litre (1.056 quarts) of milk, boiling and filtering; and then adding from 3 to 5 per cent. of rice-flour or corn-meal, and cane-sugar as desired up to 10 per cent. The almond-meal is prepared by soaking 150 grams (5.29 oz.) of almonds in cold water for from 12 to 24 hours, then grinding them in a mill with the gradual addition of 1 litre (1.056 quarts) of water and straining through a cloth. Equal parts of this and of the whey are mixed and boiled for 5 minutes. The prepared food contains percentages of fat 2.95, sugar 6.50, starch 1.5–2.5, protein 1.85, ash 0.45.

Moll and Stransky's Milk-Free Pudding-Diet.—Like the almond-meal food, this is a casein-free preparation produced in a different way, according to the directions of the authors.[3] Eggs are used to supply the fat and albumin; and whey for the albumin and salts. It is prepared as follows: 80 grams (2.82 oz.) of a finely bolted flour, 1 gram (15.43 grains) of salt, and ½ gram (7.71 grains) of baking soda are stirred in 200 grams (6.76 fl.oz.) of water. The yolk of an egg is then mixed with 40 grams (1.41 oz.) of sugar and added to the mixture. The white of an egg is now beaten into a snow and stirred in with the rest. The whole is next put into a pudding-dish which has been greased with butter and powdered with flour, and is cooked in a water-bath for ½ hour. When the child is to be fed, the pudding is rubbed through a sieve and mixed thoroughly with an equal part of whey. The caloric value of the food is about 750 calories per litre (708 calories per quart). The authors have used it with great success in many cases of different disordered digestive states, spasmophilia, and the exudative diathesis, and find it even better than Finkelstein's casein-milk for diarrheal diseases. It is best adapted for infants older than 3 months. Before this age the almond-meal food seems preferable (Moll).[4]

Gerstenberger's Synthetic Milk Adapted (S. M. A.).—This preparation, advocated by Gerstenberger and his collaborators,[5] is a combination of various vegetable and animal fats resulting in a fat having the same saponification number, iodine number, Polenske number, Reichert-Meissl number, and the same melting point as present in the fat of human milk. Cod-liver oil is one of the fats added. Skimmed milk is used as the protein constituent and lactose as the added sugar, and salts in the correct amounts are included. The whole is mixed and homogenized. The food has become a commercial one, and the percentage-composition will be found later (p. 178). It is often very useful, especially as a substitute-food for young infants or those premature, where breast-milk is not obtainable. It is serviceable also in the milder forms of malnutrition but is contraindicated in diarrhea from any cause. A Protein S. M. A. Acidulated has also been prepared, intended for use where a high protein-intake is indicated, as with other protein-milks. A number of synthetic milks have been put on the market since that of

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[1] Prag. med. Wochenschr., 1914, XXXIX, 13.

[2] Monatsschr. f. Kinderheilk., 1923, XXVI, 250

[3] Jahrb. f. Kinderheilk., 1922, C, 3.

[4] *Loc. cit.*

[5] Amer. Jour. Dis. Child., 1915, X, 249; Amer. Jour. Dis. Child., 1919, XVII, 1.

Gerstenberger, among these Recolac and Franklin Infant Food. The composition of these is given in Table 62, pp. 178, 179.

PROPRIETARY (COMMERCIAL; PATENTED) FOODS

By the title "proprietary" or "patented" food is here indicated a food of this nature directly advertised to the *laity*, usually with what must be considered extravagant claims for its value. There are numerous preparations on the market advertised to the medical profession solely, and consisting entirely of starch or of some form of sugar, or largely of protein, or containing special ingredients and not claiming to be anything else. These foods are, of course, *proprietary*, but not in the sense in which the term is commonly employed and as used here; and since they are advertised only to physicians, and consequently are not liable to be given by a mother without advice, there can be no possible objection to their employment.

The so common and wide-spread employment of the proprietary infant-foods is probably one of the most pernicious factors of the time in the feeding of infants, as indeed it has been for years. It is not because the composition of these foods is necessarily faulty which renders them so harmful, although this is frequently the case, but it is the manner in which they are freely advertised to the laity and freely used by mothers without consultation with their physicians. It is frequently only after an infant becomes ill, having taken a large variety of the foods for weeks or months, that the advice of the physician is asked about the propriety of their use. In other cases physicians themselves are largely responsible for this state of affairs, as they are prone to yield too readily to the temptation to prescribe the foods without due consideration. This saves the physician thought;—and this is the worst thing possible both for him and for the patient.

The proprietary foods are for the most part unreliable and unnecessary:—unreliable, because they are never the perfect substitute for mother's milk in spite of the claims of the manufacturers; unnecessary because it is rare that they cannot be entirely dispensed with. Moreover, in the case of those intended to be used without the addition of milk, the element of freshness, so important in infant-nourishment, is lacking; while in the case of the others the question arises, why use them at all if fresh cow's milk must also be employed in any event? They have further the additional disadvantage that the ingredients bear a fixed, unvarying relationship to each other, and the ready changing of these, which modern infant-feeding considers indispensable, is an impossibility. It is true that many infants have done well upon commercial foods after some milk-modifications had failed; but they would almost certainly have grown as satisfactorily without them and with much less risk. What they needed was a proper milk-modification. There can be nothing of advantage in a proprietary food which cannot be equally incorporated in a home-made modification, and at much less expense. It is still more true that not only have countless deaths arisen from their use, but that countless infants have suffered from rickets, scurvy, and severe gastro-intestinal disorders as a result of their employment. Cases do occasionally occur where the temporary use of a proprietary food may be a necessity because nothing else can be obtained, or advantageous for some other special reason; or beneficial, just as barley-water, beef-juice, or other food than milk is sometimes demanded. These cases form certainly the exception; and it is most important that when the selection of a proprietary food under such circumstances is to be made, the physician should be fully cognizant of its composition and what may be expected from it.

The number of proprietary foods which are now or have been on the market is vast; and it would be a waste of time and space to attempt to consider them all. Only a comparatively few will be referred to, in order that the reader may have some idea of their actual composition and mode of manufacture. The analytical tables are given in order to show how closely, or oftener how remotely, they resemble human milk in composition. Some of the foods are still actively employed; of others little or nothing is now heard.

The various proprietary foods may be classified as follows:

I. *Condensed Milks.*—These consist of whole or skimmed milk condensed by evaporating, and often with the addition of cane-sugar.

II. *Malted or Dextrinized Foods.*—The basis of these is starch which has been completely converted. To some of them milk has been added in the process of manufacture. A few have still other additions.

III. *Amylaceous Foods.*—These are composed entirely or partly of unconverted starch. Quite commonly it is recommended that they be mixed with milk. Milk and milk-sugar have been added in the process of manufacture of some of them.

IV. *Miscellaneous Foods.*—Infant-foods often with milk as a basis with the addition of other substances than, or in addition to, starch or sugar. They are incompletely or not at all dextrinized.

V. *Protein-foods.*—These consist of commercial foods claimed to be especially rich in protein. They are not primarily intended for use in infancy, but are often of temporary benefit.

I. Condensed Milks.—These, including the so-called "dried milks" and "evaporated creams," are made by evaporating the milk by heat to a greater or less degree and then sealing it in cans. All of them need much dilution with water before they can be employed.

They may be divided into:

1. Dried Milk.—This is, in fact, a condensed milk, but not in the usual commercial sense of the term. None of the condensed milks as ordinarily designated have been condensed to the degree of actual dryness. There are various preparations of dried milk upon the market sold under different names. Some of them are made from whole milk; others from skimmed milk. One has 6 per cent. of lactic acid added; and another (lactogen) has been fortified by the addition of both sugar and cream. The analysis given on p. 174 readily shows to which of these two classes each belongs. It should be stated, however, that the analysis of some of these, as given by Bosworth,[1] shows a smaller percentage of fat than should be present according to the advertisements. Dried milk has been used quite extensively in infant-feeding, and generally is well tolerated by infants over 6 weeks of age. It is especially valuable when good, clean cow's milk cannot be obtained; *e.g.* when travelling. Much of the antiscorbutic vitamin may be retained in dried milk (see p. 197). In preparation for feeding the powdered milk is mixed with a little cold water to make a paste. The remainder of the water is then added while warm and the mixture is thoroughly stirred and shaken. Each bottle should be made separately just before use. When given to young infants the strength of the mixture should not be more than 1 level teaspoonful to 1 ounce of water;—the total amount at each feeding varying, of course, according to the usual standard for age. The strength may be increased as indicated, but never beyond 4 level teaspoonfuls (1 level tablespoonful) to each ounce of water.

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[1] Amer. Jour. Dis. Child., 1921, XXII, 455.

2. **Unsweetened and Condensed Whole Milk.**—In this form nothing has been removed by skimming. It is simply a condensed whole milk. It keeps badly after the receptacle is opened.

3. **Sweetened and Condensed Whole Milk.**—In this instance from 40 to 45 per cent. of saccharose has been added in the course of preparation for the purpose of preserving the milk.

4. **Sweetened and Condensed Skimmed Milk.**—Here the milk has had the fat removed, and cane-sugar added as in the previous form.

All the condensed milks are unsuitable for infant-feeding. When properly diluted with water the ingredients still retain at the best the normal relationships of cow's milk. They are all deficient in the amount

TABLE 59.—ANALYSES OF CONDENSED MILKS

	Water	Fat	Protein	Lactose	Saccharose	Mineral matter
Human Milk............	87–88	3.5–4	1–1.5	6.5–7		0.20
Dried Milk.						
Dryco[1]..................	3.00	12.00	32.00	46.00	.....	7.00
Glaxo[1]..................	2.50	20.00	26.00	46.00	.....	5.50
Momilk[1].................	3.00	10.00	33.00	42.00	.....	7.00
Mammala[1]..............	5.00	12.00	24.00	54.00	.....	5.00
Klim[1]....................	1.50	28.00	26.74	38.00	.....	5.76
Skimmed-milk Klim[1].....	3.40	1.40	38.00	50.00	.....	8.20
Albulac[1]................	1.20	18.00	16.00	57.60	.....	7.20
Powdered lactic-acid milk[1]	3.00	28.00	26.00	33.00	.....	4.00
Lactogen[2]...............	1.99	25.00	16.17	53.31	.....	3.53
Mead's powdered milk....	1.50	28.00	26.80	37.70	.....	6.00
Condensed Whole Milk.						
Ideal[8]..................	68.27	10.10	7.36	11.03	.....	1.85
St. Charles[4]............	66.46	9.26	10.49	12.24	.....	1.55
Highland[4]...............	68.75	9.63	9.21	10.89	.....	1.52
First Swiss[5].............	62.15	11.38	9.90	14.44	.....	2.10
Hollandia[6].............	57.00	9.80	11.30	18.50	.....	3.40
Borden's Evaporated milk[3]	73.27	8.10	7.15	10.05	.....	1.43
Sweetened Condensed Whole Milk.						
Rose[7]....................	23.70	11.00	9.70	14.60	38.70	2.30
Red Cross[4]..............	25.97	7.93	8.91	11.93	43.77	1.49
Eagle[4]...................	30.16	9.50	8.10	52.50	52.50	1.70
Anglo-Swiss[6]............	25.60	10.80	8.80	16.00	37.10	1.70
Nestlé's Condensed milk[7].	26.30	11.50	9.70	13.00	37.50	1.90
Sweetened Condensed Skimmed Milk.						
Cowslip[8]	25.68	0.71	10.35	16.85	43.09	2.48
Farm[8]...................	28.48	0.60	7.90	18.76	41.77	2.04
Snake[8]..................	25.88	0.96	10.64	27.38	34.07	2.56

[1] Advertisement.
[2] Advertisement and personal communication.
[3] Thomson, Study and Treatment of Sick Children, 1925, 75
[4] Penna. Dept. of Agriculture Bulletin No. 10.
[5] Cautley, *loc. cit.*, 218.
[6] Hutchison, *loc. cit.*, 463.
[7] Cautley, *loc. cit.*, 220.
[8] Chapin, Infant Feeding, 1902, 79.

of fat required by the normal infant after the protein-percentage has been properly reduced. They may be used temporarily when a very low-fat food is required, but offer no advantages over a dilution of fresh whole milk. The large amount of sugar in the sweetened condensed milk makes the food so sweet that it could not be taken unless diluted to such a degree that the nourishing qualities, except for the sugar, are entirely inadequate. The 4th class in addition to the sweetening contains almost no fat, and is entirely unfitted for administration to infants.

The table on page 174 gives the percentage-composition, derived from various sources, of several of the condensed milks. These examples are drawn at random from the recorded analyses in my possession of over 50 different varieties. Some of them are now used little if at all.

In this, as in the succeeding tables, the analysis of human milk heads the list, for the convenience of comparison.

Condensed milks are generally diluted with from 9 to 20 parts of water before using. Assuming a dilution of 1 in 10 as convenient for calculation, we may readily see what a weak food, except for the sugar, is obtained, and can understand why condensed milk so often agrees with the digestion, but either does not support the infant, or allows it to grow flabbily fat without proper tissue, as a result of the relatively large amount of sugar present and the small amount of protein. A dilution of 1 in 10, for instance, of Eagle Brand Condensed Milk, produces a food containing only fat 0.95 per cent., protein 0.81 per cent., sugar 5.25 per cent. and salts 0.17 per cent. It should be stated that a large number of bacteria may be present in condensed milks, being left alive by the heating process or added through contamination (Park, Schroeder and Bartholow).[1]

II. Dextrinized (Malted) Foods.—The basis of these, often called also Liebig's Foods, is starch usually derived from wheat-flour or barley-flour which has been entirely converted into soluble carbohydrates, generally by the action of the diastase of a malt-extract. These carbohydrates consist of dextrin and maltose and intermediate substances. The foods should not give the iodine reaction for unconverted starch. To some of them milk is added in the process of manufacture. To others it is to be added when the food is mixed for the child. The quantity of sugar present is far too great to resemble in any way human milk, and it is to be noted also that it is not the natural sugar of the milk which is employed. When diluted sufficiently to reduce the sugar to a normal amount the foods are all much too weak in the percentage of fat, and often of protein as well, unless fresh milk or cream is added. These facts can be seen by examining the figures in the following table, which gives the analysis of a few of the foods of this class. Some other dextrinized foods of more complicated composition are placed in the group of Miscellaneous Foods. There is no real need for any of these foods, inasmuch as a milk-mixture with either the addition of starch dextrinized at home or of a commercial maltose-dextrin preparation which is not called a food, can be more conveniently, cheaply and accurately prepared in the house.

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[1] New York Med. Jour., 1915, CII, 1073.

Table 60.—Analyses of Dextrinized Foods

	Water	Fat	Protein	Soluble carbohydrates	Mineral matter	Remarks
Human milk...........	87–88	3.5–4	1–1.5	6.5–7	0.20	
Horlick's Malted Milk[1].	3.06	8.78	16.35	67.95	3.86	Milk; barley and wheat malted.
Liebe's Soluble Food[2]...	22.34	Trace	6.47	68.80	1.71	An extract of malt made from wheat.
Loeflund's Infant Food[2]	25.37	Trace	4.17	68.60	1.47	Wheat and malt.
Loeflund's Peptonized Infant Milk[2]..........	20.39	8.46	10.13	57.53	3.05	Practically a malt-extract.
Mellin's Food[1].........	5.62	0.16	10.35	79.57	4.30	Wheat, malted barley and bicarbonate of potash.
Laibose[1]...............	6.00	17.00	18.00	55.00	4.00	A combination of whole-milk with dextrinized wheat.
Justfood[3]	4.50	.....	1.10	93.95	0.32	Dextrinized cereals.

III. Amylaceous Foods.—All of these contain unconverted starch in larger or smaller amount. In some of them malt-extract, pancreatic extract or some form of sugar has been added; but in none of them, according to the analyses, is the starch entirely transformed. In the case of many of them it is admitted to be present and the food is intended to be added to milk-mixtures. In others the food is recommended without any statement regarding the presence of starch, or even with the claim that none exists in it. The accompanying table gives published analyses of a number of starch-containing foods which have been placed on the market from time to time.

IV. Miscellaneous Foods.—In this class are placed a number of foods in the composition of which other substances than starch or sugar have been used, or which in some way do not properly belong to any of the previous classes. In some the proportion of cream is increased either by direct addition of this or by removal of a part of the protein, and, the food being afterward condensed, the title of condensed cream might with propriety be employed. Some are malted or peptonized; in some unconverted starch is present, and the food might be placed among the amylaceous preparations were it not for the addition of other substances. The accompanying table gives published analyses of some of them.

V. Proteid or Nitrogenous Foods.—These form a mixed group in all of which the nitrogenous element is claimed to be high as compared with other ingredients. They are not intended to be permanent substitutes for human milk and some of them fill a useful place. They may be divided into (1) beef-extracts, (2) beef-juices, (3) peptonized meat preparations, (4) other proteid foods.

1. The Commercial Beef-extracts.—These, although popularly supposed to be highly nourishing from the amount of protein contained, possess in reality but a small percentage of this, while the extractive matter and salts are present in large amount. They are prepared under the influence of water, heat and pressure. Some of the muscle-fibre which remains is added and the liquid then evaporated. Only those in which the fibre has been thus used contain an amount of protein worth consider-

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[1] Advertisement and information from manufacturer.
[2] Blauberg, Arch. f. Hyg., 1897, XXX, 125.
[3] Advertisement.

TABLE 61.—ANALYSES OF AMYLACEOUS FOODS

	Water	Fat	Protein	Total carbo-hydrates	Insoluble carbo-hydrates	Soluble carbo-hydrates	Mineral matter	Remarks
Human milk	87–88	3.5–4	1–1.5	6.5–7	.....	6.5–7	0.20	
Nestlé's Milk Food[1]	2.50	6.50	14.65	74.35	14.00	60.35	2.00	A condensed milk with baked wheat-flour and cane-sugar.
Gerber's Infant Food[2]	4.96	4.60	13.10	77.51	32.93	44.58	1.40	Milk sweetened, with dextrinized flour added.
Kufeke's Infant Food[3]	7.60	0.88	11.17	78.51	48.01	30.50	2.07	Appears to be partly dextrinized and to have salts added.
Carnrick's Soluble Food[4]	3.12	6.26	16.32	71.06	14.44	56.62	3.02	Evaporated milk, partially malted wheat-flour and milk-sugar.[2]
Savory and Moore's Food[5]	5.34	1.06	10.79	81.90	54.09	27.81	0.91	Wheat-flour and malt. Much grape and cane-sugar added.
Wells, Richardson and Company's Lactated Food.[6]	7.76	1.64	11.85	75.43	36.43	39.00	2.61	Partly malted. Contains much cane-sugar and no milk.
Wagner's Infant Food[4]	5.07	10.91	14.81	66.82	28.91	37.91	2.01	
Ridge's Food[4]	8.87	1.67	13.37	74.67	66.35	8.32	0.61	A baked flour.
Imperial Granum[4]	10.57	1.32	19.37	67.30	51.88	15.42	1.13	A baked wheat-flour.
Theinhardt's Soluble Infant Food[7]	6.87	9.58	14.37	64.30	19.77	44.53	3.56	Wheat starch partly malted.
Neave's Food[6]	5.03	1.70	13.20	80.40	74.27	4.71	1.09	Practically all starch.
Robinson's Patent Barley[8]	10.10	0.90	5.10	82.00	.....	.....	1.90	Merely barley flour.
Robinson's Groats[8]	10.40	1.60	11.30	75.00	.....	.....	1.70	Merely ground oats with husks removed.
Allenbury's Malted Food[8]	6.50	1.00	9.20	82.80	.....	.....	0.50	Wheat-flour and malt. Contains unconverted starch.
Milkine[4]	2.74	7.12	13.37	74.82	13.63	61.19	1.32	Roasted oats made into meal. Milk-sugar added.
Hubbell's Prepared Wheat[4]	5.93	1.19	14.81	77.02	60.86	16.16	0.44	
Health Food Company's Barley[9]	10.92	0.89	6.98	.....	80.35	?	0.86	
Rademann's Infant Food[3]	7.67	6.00	15.10	66.43	47.89	18.54	3.69	Roasted oats made into meal. Milk-sugar added.
Benger's Food[5]	11.29	1.10	10.43	76.20	66.30	9.90	0.96	Wheat-flour and pancreatic extract. The large amount of starch becomes for the most part converted when the food is prepared for use.
Cereal Milk[10]	9.33	1.01	11.08	78.42	58.42	20.00	1.16	
Dennos[1]	6.80	1.00	9.70	79.80	61.10	18.70	2.00	A partially dextrinized whole-wheat flour, to which are added small amounts of cane-sugar, dextrin and sodium chloride.

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[1] Advertisement.
[2] Seitz, Kinderheilk., 1901, 27.
[3] Blauberg, Arch. f. Hyg., 1896, XXVII, 119.
[4] Penna. Dept. of Agriculture, Bull. No. 10.
[5] Cautley, Sutherland's System of Diet and Dietetics, 1908, 217.
[6] Ibid., p. 217.
[7] Blauberg, Arch. f. Hyg., 1897, XXX, 125.
[8] Hutchison, Food and Dietetics, 1917, 468.
[9] Chapin, Infant Feeding, 1902, 174.
[10] Leeds, New York Med. Jour., 1883, XXXVII, 449.

TABLE 62.—ANALYSES OF MISCELLANEOUS FOODS

	Water	Fat	Protein	Total carbo-hydrates	Insoluble carbo-hydrates	Soluble carbo-hydrates	Mineral matter	Remarks
Human Milk	87–88	3.5–4	1–1.5	.....	.....	6.5–7	0.20	
Voltmer's Mother's Milk[1]	22.10	9.08	13.26	51.52	.....	51.52	4.04	Milk boiled. Water, sugar, cream and salts added to form average percentages of human milk. Peptonized. Evaporated.
Biedert's Ramogen[2]	.....	16.50	7.00	34.65	.....	34.65	1.50	Milk evaporated. Cream, cane-sugar and an alkali added. Sterilized.
Muffler's Infant Food[1]	4.28	6.47	14.48	72.54	47.62	24.92	2.28	Wheat-flour, milk, eggs, milk-sugar, wheat-gluten.
Allenbury's Food No. 1[3]	5.70	14.00	9.70	66.85	.....	66.85	3.75	Desiccated milk; excess casein removed; vegetable-albumin, milk-sugar and cream added.
Allenbury's Food No. 2[3]	3.90	12.30	9.20	72.10	.....	72.10	3.50	Like No. 1, but with addition of some malted flour.
Liebig's Malto-leguminose mit Zucker[4]	8.44	1.37	20.34	65.10	48.19	16.91	2.97	Leguminous flour partly malted.
Frame Food Diet[3]	5.00	1.20	13.40	79.40	.....	.....	1.00	Baked-flour; cane-sugar; extract of bran. Rich in protein and unaltered starch.
Lacto preparata[5]	5.80	12.35	14.51	63.68	.....	63.68	3.66	Addition of milk-sugar instead of malted starch or cane-sugar.
Frerich's Soluble Infant Food[4]	8.44	5.97	12.98	69.82	27.59	42.23	1.67	Partly dextrinized leguminous flour.
Gärtner's Mother-milk[6]	.....	3.05	2.09	6.00	.....	6.00	....	Milk centrifugated. To the cream-portion milk-sugar and an alkali are added.
Eskay's Albumenized Food[2]	1.70	3.52	6.70	87.02	31.20	55.82	0.99	Digested cereals combined with egg-albumen.
Loeflund's Condensed Cream[7]	20.20	23.00	5.00	50.00	.....	50.00	1.80	Milk and malted flour. Apparently casein has been removed, or a fat milk used.
Backhaus' Infant Milk[8]	I. 88.93	3.16	1.32	6.04	.....	6.04	0.55	Fat-free milk treated with rennet and trypsin. The coagulum removed. Cream and milk-sugar added. Three formulæ of different strengths.
	II. 89.91	3.22	1.74	5.71	.....	5.71	0.42	
	III. 87.93	3.40	2.92	4.99	.....	4.99	0.71	
Lahmann's Vegetable Milk[9]	24.44	24.60	7.05	43.10	.....	.....	1.15	Probably chiefly vegetable albumin. Almonds and other nuts and sugar used in its preparation.
Odda[10]	5.80	6.49	14.57	71.34	.....	.....	2.10	Wheat-flour partly malted; yellow of egg; cocoa-butter.
Biedert's Buttermilk Conserve[2]	.....	0.60	9.60	34.50	4.50	30.00	2.58	Buttermilk, wheat-flour, cane-sugar prepared as for the ordinary buttermilk mixture. Milk condensed to ¼ its volume.
S. M. A.[2]	1.00	28.00	10.00	59.00	.....	.....	2.00	Combination of various animal and vegetable fats with skimmed milk, lactose and salts. Homogenized.

Protein S. M. A. acidulated[2]	2.00	22.00	35.00	28.00	.....	.....	6.00	Prepared as is S. M. A. but with proportions very similar to protein-milk. It is, however, higher in percentage of protein. Contains also fresh lemon-juice.
Recolac[12]	1.00	27.00	16 00	53.00	0	53.00	3.00	Butter-fat replaced by mixture of vegetable and animal fats, including cod liver oil. Dextrine and maltose added. Casein reduced and changed to potassium caseinate.
Franklin Food[12]	3.60	27.10	12.30	53.40	0	53.40	3.60	A homogenized mixture of skimmed milk, lactose, water, cod liver oil, butter, and various vegetable fats. The amount of calcium has been reduced.
Akrelac[12]	3.00	5.00	35.00	46.00	.....	46.00	8.00	Contains 3 per cent. free lactic acid. When diluted has a composition equivalent to that of buttermilk.

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[1] Blauberg, Arch. f. Hyg., 1896, XXVII, 119.

[2] Advertisement.

[3] Hutchison, Food and Dietetics, 1917, 468.

[4] Blauberg, Arch. f. Hyg., 1897, XXX, 125.

[5] Advertisement (Ref. M. and G. C. Lincoln, Artificial Feeding in Children, 1916).

[6] Poole (Fischer, New York Med. Rec., 1897, Dec. 11).

[7] Biedert, Kinderernähr. im Sauglingsalter, 1900, 196.

[8] Hartung, Jahrb. f. Kinderheilk., 1902, LV, 676.

[9] Höck, Wien. med. Wochenschr., 1896, 435.

[10] Mering, Therap Monatsh, 1902, 175.

[11] Amer. Jour. Dis. Child., 1919, XVII, 1.

[12] Advertisement and statement by manufacturer.

ing. A table of analyses of a number of them published by Hutchison[1] shows a total percentage of soluble protein varying in round numbers from 3 to 33 per cent. As only small doses are customarily given, or could, indeed, be tolerated on account of the excess of mineral matter and extractives, the amount of protein received by the infant is in reality trifling; given in doses of a few drops, as is often done, either alone or in addition to the milk in the bottle.

2. **Beef-juices.**—These consist of expressed juice or serum of the beef. According to analyses of a number of the commercial preparations, as published by Hutchison,[2] the percentage of coagulable protein varies from 17 per cent. to 0.3 per cent., nearly all of those in his list containing not over 5 per cent. Although some of them are richer in protein than is freshly prepared beef-juice, yet, as in the case of the beef-extracts, the salts and extractive matter are generally present in too large an amount to permit of the giving them in sufficient quantity to be of real benefit. My preference has always been decidedly for the freshly prepared beef-juice (p. 160).

3. **Beef-powders and Peptonized Beef-preparations.**—Some of these are extensively employed for temporary use in infant-feeding and infant-therapeutics. Prominent among them are a number whose nitrogenous value is slight and whose percentage in alcohol is high. On account of their comparatively pleasant taste they may be used in place of other alcoholic stimulants in infancy and childhood. The following analyses are given by Harrington.[3]

TABLE 63.—ALCOHOLIC STRENGTHS OF LIQUID BEEF-PREPARATIONS

	Alcohol by volume, per cent.	Total solids, per cent.
Liquid Peptonoids	23.03	14.91
Panopeptone	18.95	17.90
Hemapeptone	10.60	19.54
Nutritive Liquid Peptone	14.81	15.20
Hemaboloids	15.81	6.36
Tonic Beef	15.58	18.16
Mulford's Predigested Beef	19.72	10.39

4. **Other Proteid Foods.**—Among other preparations the following may be mentioned:

Mosquera's beef-jelly[4] which contains 28.63 per cent. of protein in a partially digested state.

Somatose.—A meat-powder completely peptonized and containing over 80 per cent. of protein (Neumann.)[5] Another form, Milk-Somatose, is derived from milk and contains about 70 per cent. of protein (Neumann). Somatose is often useful as an addition to milk.

Dry Peptonoids.[6]—This contains 40 per cent. of protein, the remainder of its nutritive value depending entirely on carbohydrates (51.5 per cent.).

Tropon.—A powder rich in protein (90.44 per cent. Fröhner and Hoppe)[7] derived both from animal and vegetable sources. It is not predigested.

[1] Food and Dietetics, 1917, 96.
[2] *Loc. cit.*, 100.
[3] Bost. Med. and Surg. Jour., 1903, CXLVIII, 283.
[4] Wylie, Foods and Their Adulterations, 1911, 565.
[5] Münch. med. Wochenschr., 1898, XLV, 72.
[6] Advertisement.
[7] Münch med. Wochenschr., 1899, XLVI, 46.

Roborat.—A food in powdered form made from various grains and containing about 95 per cent. of vegetable-albumin (Sommerfeld).[1]

Hygiama.—A powder giving 21.93 per cent. of nitrogenous substance as well as a high percentage of carbohydrate, most of which is in soluble form. It is flavored with cocoa (Kraus).[2]

Plasmon represents in powdered form 75 to 80 per cent. of albumin derived from milk, with the addition of 5 to 7 per cent. of carbonate and bicarbonate of soda. It is in reality an alkali-casein (Laves).[3]

Nutrose.—A powdered food formed of a soda-combination with casein. It possesses 73.68 per cent. of albumin and 11.67 per cent. of water (Neumann).[4]

Soson.—A meat-derivative containing 92.5 per cent. of albumin (Neumann).[5]

Eucasin.—A powdered preparation of casein consisting of 77.60 per cent. of nitrogenous material (König).[6]

Fersan.—A powdered product made of beef-blood. It contains 84 per cent. of nitrogenous material (König).[7]

Albulactin[8] is a grey-white powder consisting chiefly of lactalbumin, and having 83.60 per cent. of protein. It may be added in small amounts to mixtures of cow's milk.

Sanatogen (Street).[9]—This is a preparation containing about 90 per cent. of casein, with sodium-glycerophosphate and a small amount of unidentified nitrogenous compound. It would appear to possess no advantage over the very much cheaper commercial casein.

Certain other high-protein foods have already been mentioned in connection with the preparation of casein-milk (p. 159).

CHAPTER VII

DIET AFTER THE FIRST YEAR

Before discussing the feeding to be employed in the 2nd year of life, the question should be considered as to whether or not the diet for this period, which has been in vogue so long and which is about to be described, may not with advantage be commenced decidedly earlier. There has been a growing disposition in many quarters to follow this plan. The arguments in favor of this are to a certain extent fallacious. It has been claimed, for instance, that in the latter part of the 1st year, the deficiency of iron in milk causes the infant to become anaemic. It has also been maintained that milk does not contain sufficient of the vitamins, and that green vegetables are necessary. A complete answer to this would seem to be the fact that countless babies for generations have thrived perfectly during the 1st year on milk-formulæ, with at most the employment of a cereal-decoction as a diluent, without any development of

[1] Arch. f. Kinderheilk., 1903, XXXVI, 341.
[2] Therap. Monatsh., 1902, XVI, 635.
[3] Münch. med. Wochenschr., 1900, XLVII, 1339.
[4] *Loc. cit.*, 72.
[5] Münch. med. Wochenschr., 1899, XLVI, 1296.
[6] Chemie der Menschlichen Nahrungs- u. Genussmittel, 1904, II, 539.
[7] *Loc. cit.*
[8] Advertisement, Analytical Report, Lancet, 1911, I, 34.
[9] Jour. Amer. Med. Assoc., 1914, LXIII, 1831.

anemia or other disturbance of health. Morse[1] in a suggestive paper has shown that in a quart of milk there are all the vitamins needed, especially if orange-juice is given; and that, with a small amount of beef-juice and cereal, there is also more than enough iron; while in the green vegetables there is not as much iron as has usually been supposed. He believes that the older, simpler diet is more digestible, entirely sufficient, and far safer. It would seem that the development of anemia or other manifestation of illness, if dietetic at all in origin, shows a fault in the ability of these infants to utilize the necessary elements from the diet, rather than a fault in the diet itself as adapted for the majority of infants in general of this age.

Yet, on the other hand, the practise of beginning cereal porridges and other foods before the end of the 1st year has much in its favor. Jundell,[2] for instance, fed 382 infants in the last quarter of the 1st year with a diet which contained minced meat, scrambled eggs, minced green vegetables, fruit-pulp, cocoa, cereal gruels, and the like. The daily amount of milk was cut down to 300 to 550 c.c. (10.14 to 18.60 fl.-oz.). While not advocating this diet as the rule, the general impression received was that infants on liberal diets did better on the whole. He thinks that these experiments show that there is evidently no risk in giving this generous diet in the 3rd quarter of the 1st year, and that this is especially to be remembered in the case of infants of 9 months who have not been developing well on the simpler regime. As regards the digestion of meats, Karger and Peiper[3] claim that infants as young as 2½ months can digest meat perfectly well. The writers do not appear, however, to urge its administration.

As a matter of fact, if a strong cereal-decoction has been employed during the 1st year as a diluent, there is manifestly no objection to the giving of the cereal in the form of a porridge and fed from a spoon. Indeed, the procedure has the advantage that it early accustoms the infant to take food in this way, and makes the weaning later from the bottle less difficult in the cases of refusal of spoon-food which are occasionally encountered.

The conclusion that can properly be reached, as far as our own opinion goes, is that, whereas there is no necessity of giving the healthy, thriving infant a diet other than a milk-mixture with cereal diluent, or cereal-gruel and orange-juice, as a routine measure during the 1st year, yet in any case in which the progress in growth and the general condition are not satisfactory it may be well experimentally to begin the diet now to be described at a period decidedly earlier than the end of the 1st year. An exception is made in the case of orange-juice, which may well be commenced for bottle-fed babies at the age of 3 months, if the food is being boiled or pasteurized, in order to prevent the development of scurvy.

The discussion which follows is that of the simpler diet, without the early employment of green vegetables.

DIET FROM 12 TO 18 MONTHS

By the time the infant is 1 year old it will have been weaned and be fed upon cow's milk nearly or quite undiluted, and it should have commenced to take a certain amount of starchy material in the milk (p. 162). From this age its diet is gradually extended. It is better first to increase the amount of starch by having the child learn to eat stale bread or toast moistened with milk and fed from a spoon; a well-cooked porridge of farina, oatmeal, hominy or wheaten grits, arrowroot, sago, tapioca, etc., one not of oatmeal being the first to be tried; or some form of the numerous excellent farinaceous breakfast foods on the market, those which require cooking being much to be preferred. All the porridges must be seasoned with salt in their preparation or before use, a small amount of milk poured over them, and a little cane-sugar added, if necessary, to make the infant eat them. It is better, however, to use no sugar. They may be made from the grain or from the flour, using about 4 level tablespoonfuls to the pint of milk of water. The boiling must be slow and continue for from 3 to 6 hours for broken grain, and at least ½ hour for flours and longer for oatmeal. The water should be replaced as it boils away. The gruels should at first be strained. Later this may be omitted in the case of the finer meals, but oatmeal requires straining until the age of about 18 months. The porridge may with advantage be employed for the midday meal, the other meals still consisting of milk. Very shortly it may instead be given for breakfast, and for dinner the child may receive bread or well-boiled rice, moistened with beef-juice or with dish-gravy as free as possible from fat. The beef-juice should be commenced in small amounts of 1 to 2 teaspoonfuls and then gradually increased up to 2 ounces at most. Soft-boiled eggs may be tried at about the age of 15 months, taking pains that the white is not firmly coagulated, and remembering that many children do not tolerate eggs at all well until after the 18th or 20th month, and others not even then unless given them only occasionally. Throughout early childhood the giving of eggs oftener than twice a week should be tried only cautiously. All through this period milk should remain the principle article of diet, and care with regard to its bacterial content continued. Cream is inadvisable. Yet precautions must be taken that milk is not practically the only food ingested, but that as the amount of solid food is increased that of the milk be reduced somewhat. The more milk there is poured upon the porridge, the less should be drunk from the cup. A total of from 24 to 32 oz. should usually be the outside daily limit. The mild may be slightly or not at all diluted, or may have a small amount of some cereal jelly added. If it is from Channel-Islands cattle the removal of some of the cream is advisable. Very many infants in the 2d year do not tolerate undiluted milk, especially if it is rich in fat. Very frequently trouble arises from this source. As early as possible in the 2nd year the infant should be taught to take most of the milk from a cup or glass, inasmuch as it is very difficult to combine the eating of solid food at mealtime with the drinking of milk from a bottle. The milk from the bottle will be taken without pause and the desire for solid food interfered with; or if the bottle is given last, the child takes too much nourishment for one meal. Indeed, it is well to begin the teaching to drink milk from a cup before the age of 1 year is reached. An exception may be made, if desired, as regards the bottle before the morning nap and that at 9 or 10 P.M. The juice from a small orange may properly be added to the dietary if this has not been done earlier; or from well-stewed prunes if there is constipation. Orange-juice may be given an hour before one of the feedings.

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[1] Jour. Amer. Med. Assoc., 1920, LXXIV, 577.

[2] Acta Paediat., 1921, I, 240.

[3] Jahrb. f. Kinderheilk., 1920, XCI, 235.

The following list may be followed as a guide for the feeding of children from 12 to 18 months of age. The limits of age in this and the succeeding tables are to a certain extent arbitrary, and circumstances may make it best to alter them. A numbered selection of dietaries is given.

Table 64.—Diet from 1 Year to 18 Months

Breakfast (6 to 7 A.M.); (1) 8 to 10 oz. of milk with stale bread, toast or zwieback broken into it. (2) 2 to 3 tablespoonfuls of well-cooked oatmeal, arrowroot, wheaten grits, hominy grits, farina, etc., or one of the numerous good breakfast foods on the market, only occasionally of the ready-to-serve class, made into a well-cooked unstrained porridge, and with 6 to 8 oz. of milk poured over it. (3) A soft-boiled or poached egg (after 15 months) with stale bread, toast or zwieback thinly buttered, and a cup of milk.

Second Meal (10 A.M.): 8 or 10 oz. of milk from a cup or bottle.

Dinner (1:30 to 2 P.M.): (1) Stale bread, toast or zwieback moistened with dish-gravy (no fat), beef-tea, or 1 to 2 oz. of beef-juice; a cup of milk. (2) Rice or grits moistened in the same way; 6 to 8 oz. of milk. (3) A soft-boiled egg and stale bread thinly buttered; 6 to 8 oz. of milk. Sago, tapioca, or rice pudding (no raisins), junket, or cornstarch in small quantities as dessert with any of these diets.

Fourth Meal (5 to 6 P.M.): 8 to 10 oz. of milk; or some bread and milk.

Fifth Meal (9 to 10 P.M.): 8 to 10 oz. of milk from a cup or bottle.

This list is intended as suggestive only. It is also one to which the child must grow accustomed very gradually, and it represents the *extreme* of what may be given rather than what must necessarily be eaten. Many infants do well with a dietary very much weaker than this. Inasmuch as the daily amount of milk is now restricted, care must be taken that the child receive water. The milk should not be used as a drink to replace this in order to quench thirst. If it is desired, a cup of broth may take the place of milk at dinner; but there is usually, in my experience, no advantage in this, and it can well be dispensed with until after the age of 18 months at least in the earlier part of this period. Unless thickened with cereal material it is a weak food, and takes away too much the appetite for more nourishing substances. As already stated, green vegetables in small amount may also be given if there is an indication for their use. The number of teeth which have been cut should have little influence upon the selection of the food during this period, since the infant does but little masticating in any event. The weight and general condition are better guides.

With regard to the hours mentioned in this and the succeeding tables, circumstances may make a modification of them advisable or necessary. The practise often followed of giving a cup of milk at 6 or 7 A.M., and then the heartier breakfast at 10 A.M. makes the two principal and largest meals of the day come too close together; but the household arrangements, or the time when the child takes his daily nap, may require a change in the schedule outlined. If, for instance, the child sleeps in the early afternoon, it may be better to give the dinner at 12 A.M.; or if the early breakfast is impossible, the dinner-time may be fixed at 2 P.M.

DIET FROM 18 MONTHS TO 2 YEARS

During this period but little change is made in the dietary. Milk is still a standby, and should have the same precautions against contamination taken with it as earlier in the infant's life, but the quantity is being diminished by the employment of more solid food. Potatoes may be given occasionally for dinner, although the starch of this vegetable seems often less digestible than some other varieties. Finely minced meat is also advisable. Orange-juice, well-stewed prunes mashed through a sieve, and well-baked apple taken with little or no sugar are useful additions. Green vegetables may now be commenced if they have not, for special reasons, been given earlier. The vegetables should be thoroughly cooked, or, better, well-steamed to favor the retention of the salts (Bartlett),[1] and then rubbed through a fine sieve. Some children assimilate food of this sort readily; others find difficulty in digesting it even at a decidedly later period. The appearance of a moderate amount of some of these vegetables in the stools does not mean that they should be taken from the dietary. The fifth meal is no longer desirable, and, in the case of some children, the omission of the second meal gives a better appetite for the succeeding ones. The following list is, like the last, a guide only.

TABLE 65.—DIET FROM 18 MONTHS TO 2 YEARS

Breakfast (7 A.M.): (1) 8 oz. of milk with a slice of buttered bread or toast, or a soda, oatmeal or other unsweetened biscuit. (2) A soft-boiled or poached egg, with bread and butter and 6 to 8 oz. of milk. (3) Porridge as described in the previous list.

Second Meal (10 A.M.): (1) Bread broken in milk. (2) Bread and butter or a soda or other biscuit with 6 to 8 oz. of milk.

Dinner (2 P.M.): (1) Boiled rice, macaroni, or a baked potato mashed and moistened with dish-gravy or beef-juice; or from ½ to 1 tablespoonful of well-cooked spinach, string-beans, peas, asparagus-tips, celery, or carrots rubbed through a fine sieve, 6 oz. of milk. (2) 6 ounces of mutton or chicken broth cooked with the pulp of green vegetables, with barley or rice in it and the meat-fibre remaining (see p. 161); some bread and butter, zwieback or toast, and some sago, tapioca, bread-, or rice-pudding. (3) 2 or 3 teaspoonfuls of minced white meat of chicken or turkey, lamb, mutton, fish, underdone beef or beef-steak; bread and butter; 8 oz. of milk. A baked apple, or a few stewed prunes pressed through a colander may be allowed as dessert with some of these dietaries at the meals when green vegetables are not given.

Fourth Meal (6 P.M.): (1) Bread and milk. (2) Milk with soda or other biscuit, toast, zwieback, or bread and butter. (3) 2 or 3 tablespoonfuls of a cereal-porridge with 8 oz. of milk.

DIET FROM 2 TO 3 YEARS

Meat which is merely cut up, not scraped or minced, is now added to the list. Stewed fruit in greater quantities can be used, and many fresh fruits if ripe are excellent, such as peaches, grapes without seeds and raspberries. Bananas should be given not at all except for special reasons and if thoroughly over-ripe, and strawberries very cautiously and only if sweet and quite ripe. There is generally not the same need for specially germ-free milk as existed earlier in life, although precautions should still be taken to obtain a good and pure article. A total of 24 ounces daily should be sufficient and it should not be rich in fat. It should have the chill removed, but need not be actually warm.

The following list is a guide for this period.

TABLE 66.—DIET FROM 2 TO 3 YEARS

Breakfast (7 to 8 A.M.): (1) A small portion of beef-steak, with 2 to 3 tablespoonfuls of farina, oatmeal, hominy-grits, wheaten grits, corn-meal, or other cereal porridge with 6 or 8 oz. of milk upon it. (2) A soft-boiled or poached egg; bread and butter, and 6 or 8 oz. of milk.

Second Meal (11 A.M.): 8 oz. of milk, with toast, bread and butter or a soda-cracker or other biscuit. (2) Bread and milk. (3) 6 to 8 oz. of chicken or mutton broth.

Dinner (2 P.M.): Roasted fowl, mutton, or beef cut fine; mashed baked potato, rice, or macaroni, with butter or dish-gravy on it; bread and butter, a green vegetable of the sort mentioned, and well-cooked and mashed. As dessert, tapioca, sago, or rice-pudding, junket, custard or some of the fruits mentioned. Custard should not be given on the days upon which egg has been eaten for breakfast.

Supper (6 P.M.): (1) Bread and butter. (2) 8 oz. of milk, with soda-cracker or similar biscuit, or with bread and butter, toast or zwieback. (3) 2 to 4 tablespoonfuls of a cereal porridge, with 8 oz. of milk. (4) A soft-boiled egg with bread and butter. It makes a variation to give the egg for supper on some days and for breakfast on others; but it should not be allowed twice a day.

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[1] Arch. of Pediat., 1917, XXIV, 436.

Again must be emphasized the statement made, that these tables are a guide only, not an absolute rule, and that they represent rather the extreme than the requirements of the dietary.

No increase of diet should be made in the midst of a heated term unless it is absolutely necessary. Indeed, it is better in very hot weather to return to one suitable for a much younger child, and to let it be largely of milk, diluted more than usual. It is sometimes advised to use a small amount of cream upon the cereal porridge. In my own experience healthy children are better without it. Very many children exhibit great difficulty in digesting much starchy food; others, as stated, show some degree of intolerance for egg, and a very large number are much better off with eggs not more than two or three times a week. More indigestion develops from an excess of starchy food than perhaps from any other cause. When this occurs cereal foods must be much curtailed, and meat may occasionally be allowed for breakfast as well as for dinner. As a rule, however, meat should be permitted but once a day. The ready-to-serve cereals may be given occasionally, but should be avoided as a steady diet. At the outbreak of any acute illness, especially of the digestive apparatus, the diet should be very greatly reduced in variety and amount. Of course, the existence of a chronically weak digestion modifies greatly all the schemes for diet which have been detailed. There is no good foundation for the popular fear of the "second summer," provided the diet be watched then with the same care as in the first summer. A child which has been breast-fed has had the advantage in its first summer of being upon its natural food; and it is on this account that the "second summer" has acquired a bad reputation.

Thorough mastication and slowness of eating must be taught as early as possible, but it is difficult to obtain this. Neither before the age of 3 years nor after it should food be permitted between meals, unless there is actual hunger, and then the habit of irregular eating must not be allowed to form, and candies and cakes are entirely out of the question. Allowing the child to go hungry on a few occasions will often get it in the way of eating more heartily at its regular meal-times. During temporary loss of appetite eating should not be urged, since the child may be merely following Nature's demands for a rest of the digestive organs. Care should be taken, too, as has already been stated, not to put too much sugar upon cereal gruels. A very little may be used, but it is better to accustom the child to take them without sugar.

The meals, up to about the age of 2½ years, are best given in the nursery, but from this time the child can well sit at the table in its high chair, or at a small table close by, provided that it never be given articles of food not suited to its age.

DIET FROM 3 TO 6 YEARS

From 3 years onward the dietary is decidedly increased in variety, approaching more closely, although gradually, that of the adult. All rich, highly seasoned dishes are to be avoided. Milk, not rich, must still form a very prominent article of diet, and should continue to do so up to the age of 5 or 6 years at least, but not more than 24 ounces should be allowed, and sometimes less than this. Cream should be employed with great caution and in small quantity, and not rich. The frequency with which eggs can be given varies with the case and must be carefully studied. The heartiest meal should be in the middle of the day, and the supper should be light. Meat may be given once or twice a day, depending upon the needs of the child and the character of its digestion. Fruits must be allowed with great caution in hot weather. The age when the giving of the mid-morning meal should be stopped and the feedings limited to 3 in the day varies with the hours selected for meals and with the individual child. So, too, the age at which the child may join the family in the evening dinner is a variable one. Certainly until the age of 5 or 6 years at the least the evening meal should be light.

The following lists may be found useful as a guide.

Table 67.—Foods Permitted

Meats.—Broiled beef-steak, lamb-chops and chicken; broiled liver; roasted or boiled beef, mutton, lamb, chicken and turkey; broiled or boiled fish.

Eggs.—Soft-boiled, poached, scrambled, omelette.

Cereal Foods.—Light and not too fresh wheaten and Graham bread, toast, zwieback; plain unsweetened biscuit, as oatmeal crackers, water crackers, etc.; hominy grits, wheaten grits, corn-meal, barley, rice, oatmeal, macaroni, etc.

Soups.—Vegetable and meat soups, broths and purées of nearly any kind.

Vegetables.—White potatoes, boiled onions, spinach, peas, carrots, asparagus except the hard parts, stewed tomatoes, string and other beans, salsify, lettuce, stewed celery, young beets, arrowroot, tapioca, sago, etc.

Fruits.—Nearly all if stewed and sweetened; of raw fruits, used moderately, peaches are one of the best; pears; well-ripened and fresh raspberries; blackberries; grapes without the skin and seeds; oranges without the rind.

Desserts.—Light puddings, as rice-pudding without raisins, bread-pudding, etc., plain custards, wine-jelly, junket, and occasionally ice-cream.

Food to Be Taken with Considerable Caution

Kidney, liver, oysters, muffins, hot rolls, sweet potatoes, baked beans, squash, turnips, parsnips, egg-plant, green corn, cherries, plums, raw apples, strawberries, blueberries, gooseberries, currants.

Foods to Be Avoided

Fried food of any kind, griddle-cakes, pork, sausage, ham, goose, duck, veal, corned-beef, pastry, salt fish, highly seasoned foods, all heavy, doughy, or very sweet puddings, unripe, sour or wilted fruit, bananas, pineapples, cucumbers, radishes, raw celery, raw tomatoes, cabbage, cauliflower, nuts, candies, sweet cakes, preserved fruits, hams, tea, coffee, alcoholic beverages.

After the age of 5 or 6 years the diet may be still further increased in variety, being almost similar to that suitable for adults; but care must be followed, and close supervision given up to the age of puberty.

AMOUNT OF FOOD-ELEMENTS AND NUMBER OF CALORIES REQUIRED AFTER THE FIRST YEAR

A knowledge of the percentage-composition and caloric value of the foods given after the 1st year is less often required than in infancy, but still often very serviceable. This is particularly true when indigestion of some form develops, or when the question of over-feeding or under-feeding arises. The caloric needs of children after the age of 1 year have recently received considerable attention. The matter is reviewed by Locke,[1] as also by Knox,[2] based upon studies by Camerer,[3] Sommerfeld[4] and others. Benedict and Talbot[5] have given a number of figures for basal metabolism. McCann[1] has reviewed the subject of calorimetry and mentioned the caloric requirements of children, and Holt and Fales[2] have summarized in a critical way the work done on the food-requirements for this period of life. The total amounts of the different ingredients necessary vary decidedly, this depending in part upon the fact that the food-elements are to a considerable extent interchangeable, but that if, for instance, a larger amount of fat is given the total quantity of food required is less, on account of the greater caloric value of this. The average amounts given by Sommerfeld, based upon various collected estimations, are shown in the following table:

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[1] Bost. Med. and Surg. Jour., 1913, CLXIX, 702.
[2] Jour. Amer. Med. Assoc., 1916, LXVII, 432.
[3] Der Stoffwechsel des Kindes, 1896.
[4] Pfaundler und Schlossmann, Handb. der Kinderheilk., 1906, I, 1,401.
[5] Carnegie Instit. of Washingt. Publ. 302, 1921.

TABLE 68.—DAILY QUANTITY OF THE DIFFERENT FOOD-ELEMENTS REQUIRED

Age	Protein, grams	Fat, grams	Carbohydrates, grams
2–4 years	40–64	32–62	110–205
5–7 years	50–58	30–43	145–197
8–10 years	60–88	30–70	220–250
10–11 years	68–86	44–85	211–270

In round numbers he places the needs as:

From 2 to 4 years, protein 50 grams (1.76 oz. Av.), fat 50 grams (1.76 oz. Av.) carbohydrates 140 grams (4.94 oz. Av.).
From 5 to 8 years, protein 80 grams (2.82 oz. Av.), fat 65 grams (2.29 oz. Av.), carbohydrates 220 grams (7.76 oz. Av.).
From 8 to 12 years, protein 85 grams (3 oz. Av.), fat 80 grams (2.82 oz. Av.), carbohydrates 275 grams (9.70 oz. Av.).

The actual amount of food needed increases steadily as the child grows older, but the relative amount as compared with the body-weight; *i.e.* the energy-quotient, decreases steadily. This is dependent in part upon the diminished ratio between the surface and the body-weight, and in part upon the relatively more rapid growth in the younger subjects. Taking the weight as our guide, the caloric requirements for the 1st year have already been detailed (p. 46). For the age of 2 years and upward, as determined by Camerer,[3] they may be seen in the following table given by Sommerfeld.[4]

TABLE 69.—DAILY TOTAL AMOUNT OF FOOD-ELEMENTS IN GRAMS, AND OF THE CALORIES, PER KILOGRAM OF BODY-WEIGHT

Age in years	Sex	Total food, grams	Water, grams	Protein, grams	Fat, grams	Carbohydrates, grams	Calories per kilogram of body-weight
2–4	Each	93.1	75.3	3.6	3.1	9.2	75.3
5–7	Girls	84.4	67.4	3.0	1.9	10.7	69.0
	Boys	84.3	66.6	3.5	2.5	10.9	76.6
7–10	Girls	75.5	59.0	2.7	1.3	9.9	59.2
	Boys	70.8	55.5	2.8	1.3	10.4	61.0
10–14	Girls	54.0	41.4	2.1	1.4	8.4	51.4
	Boys	56.1	44.4	2.5	1.0	7.7	47.3

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[1] Medicine, 1924, III, 1.

[2] Amer. Jour. Dis. Child., 1921, XXI, 1; 1921, XXII, 371; 1922, XXIII, 471; 1922, XXIV, 44; 1922, XXIV, 311.

[3] *Loc. cit.*

[4] *Loc. cit.*, 402.

All but the protein may be varied, but this should be approximately the amount given in the table. About 30 per cent. of the food should be of animal origin, and about 50 per cent. of the protein should be of this nature, the rest being of vegetable derivation (Camerer). An examination of the table shows that the protein should be from 20 to 25 per cent. of the total diet excluding water.

This percentage of protein in Camerer's estimations is slightly greater than the very commonly adopted ratio of protein 15, carbohydrate 61, and fat 24, which is favored by E. Müller.[1] Holt and Fales recommend very similar figures, based on a summary of the work on food-requirements done by others, to which they added their own studies of the diets taken by over 100 selected children of both sexes from 1 to 16 years of age. These children were healthy, well cared for, and had normal digestion. These authors emphasize the fact already pointed out by others, that in calculating the total caloric needs there must be considered the requirements for basal metabolism, for growth, for food lost in excreta, and for muscular activity. The average for the first three factors is nearly uniform for children of the same weight living under similar conditions. The requirement for activity varies widely with different individuals. The total caloric needs of children of both sexes during adolescence exceed the requirements of the adult of moderate activity by nearly 1000 calories. The results of the studies by Holt and Fales[2] are shown in the following table.

Table 70.—Caloric Protein, Fat and Carbohydrate Requirements of Children

	At 1 year, average per kilo.	By 6 years, and throughout growth-period, average per kilo.	Per cent. of total calories	Remarks
Total calories......	100	Boys 80 Girls 76		Adult standard of 48 calories per kilo is rapidly approached after 15 years.
Protein............	4.0 gm.	2.6 gm.	15	⅔ of protein is animal. ⅓ of protein is vegetable.
Fat...............	4.0 gm.	3.0 gm.	35	
Carbohydrate......	12.0 gm.	10.0 gm.	50	½ of carbohydrate is sugar. ½ of carbohydrate is starch.

As regards the fat of the diet, the studies of Holt, Courtney and Fales[3] show that vegetable fats, such as nut-butter and corn-oil, are well assimilated in early childhood, and that to a certain extent they can well replace the more expensive animal butter-fat derived from the milk. There must not, however, be a complete substitution, since the vegetable fats are deficient in fat-soluble vitamin. Sherman and Hawley[1] found from an experimental study, that children from 3 to 14 years of age required a diet containing about 1000 grams (33.81 fl.oz.) of milk daily, to provide the best storage of calcium. Green vegetables did not satisfactorily replace this.

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[1] Handb. d. Kinderheilk., Pfaundler and Schlossmann, 1923, I, 425.

[2] *Loc. cit.*

[3] Amer. Jour. Dis. Child., 1919, XVIII, 157.

In closing this discussion of the food-requirements of childhood, it must be emphasized that the estimations of different writers vary widely, and that at most they are largely theoretical. This has been especially well pointed out by Renault and de Tannenberg,[2] who give a table of the caloric needs for different periods, with figures much in excess of those most commonly accepted. They showed by clinical tests that in many cases a lowering of the amounts below the figures given by them was followed by a cessation of gain in weight. The practical conclusion must be that, with good health and good digestion, the amount of food given must vary with the child and depend upon its rate of growth whatever its theoretical demands may be.

PERCENTAGE-COMPOSITION AND CALORIC VALUE OF VARIOUS FOODS IN INFANCY AND CHILDHOOD

In Table 71 may be found the percentage-composition and caloric value of a number of dietary articles likely to be employed in infancy and childhood. They are taken, with a few exceptions indicated by an asterisk, from the "Composition of American Food-materials" by Atwater and Bryant.[3] The edible portion of the food is that referred to in nearly all instances, and always unless otherwise mentioned, when the letters "A. P." (as purchased) are appended, as by the authors. All foods are raw unless otherwise stated. The equivalents of the caloric values for kilograms are given in addition to the avoirdupois estimations of Atwater and Bryant. The caloric values are given in round numbers. The percentage-figures for human milk are those very commonly adopted, and those for, top-milk are the approximate averages derived from a number of sources, and the caloric equivalents for these are calculated by Fraley's formula (p. 124). In the measuring of milk and other liquids, as expressed in ounces, since 1 pint of water weighs practically 1 pound avoirdupois, the difference between the fluid ounce and the ounce avoirdupois is slight enough to be disregarded, both as to percentage-composition and caloric value. A smaller list of the caloric values of certain foods as prepared for use will be given later. (See Table 72.)

Caloric Value of Cooked Foods.—The caloric estimation of cooked articles of diet can be only approximate, depending upon the manner in which the cooking is done. This applies with especial force to the porridges, since the proportions of water and of cereal substance vary greatly according to the recipe employed. Table 72 (p. 193) is one which we have adapted and rearranged from the long and useful lists given by Locke[4] and by Rose.[5] For further details regarding the method of preparation the reader is referred to the latter's book upon the subject.

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[1] Jour. Biol. Chem., 1922, LIII, 375.

[2] Bull. de la soc. de péd. de Paris, 1920, XVIII, 301.

[3] U. S. Dept. Agriculture, Office of Experimental Station, Bull. 28, Revised Edit., 1906.

[4] Food Values, 1916.

[5] A Laboratory Handbook for Dietetics, 1917. Feeding the Family, 1917.

Table 71.—Percentage-composition and Caloric Equivalents of Various Foods

Food material	Water, per cent.	Protein, per cent.	Fat, per cent.	Carbo-hydrate, per cent.	Ash, per cent.	Calories per avoirdupois oz.	Calories per avoirdupois lb.	Calories per kilogram or litre
Milk Foods.								
*Human milk	87.0	1.5	4.0	7.0	0.2	21	330	728
Cow's milk (average)	87.0	3.3	4.0	5.0	0.7	20	326	719
Skimmed milk; commercial fat-free	90.5	3.4	0.3	5.1	0.7	11	170	375
Skimmed milk; after removal of gravity cream	90.0	3.3	1.0	5.0	0.7	13	20.8	429
*Top milk, 32 per cent. fat	61.4	2.5	32.0	3.4	0.7	87	1398	3082
*Top milk, 20 per cent. fat	72.5	2.9	20.0	3.9	0.7	59	936	2064
*Top milk, 16 per cent. fat	76.0	3.05	16.0	4.2	0.7	48	769	1695
*Top milk, 12 per cent. fat	79.8	3.2	12.0	4.3	0.7	39	630	1389
*Top milk, 10 per cent. fat	81.6	3.3	10.0	4.4	0.7	35	554	1221
*Top milk, 7 per cent. fat	84.4	3.4	7.0	4.45	0.7	27	437	963
Buttermilk	91.0	3.0	0.5	4.8	0.7	10	165	364
Whey	93.0	1.0	0.3	5.0	0.7	8	125	276
Butter	11.0	1.0	85.0	...	3.0	225	3605	7949
Meats.								
Beef, lean ribs	67.9	19.6	12.0	...	1.0	54	870	1918
Beef, lean round	70.0	21.3	7.9	...	1.1	46	730	1609
Beef, roasted, A.P.	48.2	22.3	28.6	...	1.3	101	1620	3572
Beef-steak: round, cooked, fat removed, A.P.	63.0	27.6	7.7	...	1.8	53	840	1852
Beef-steak, loin broiled	54.8	23.5	20.4	...	1.2	81	1300	2866
Beef-juice	93.0	4.9	0.6	...	1.5	7	115	254
Beef-liver	71.2	20.4	4.5	1.7	1.6	38	605	1334
Mutton, leg, lean	67.4	19.8	12.4	...	1.1	56	890	1962
Lamb-chop, broiled	47.6	21.7	29.9	...	1.3	104	1665	3671
Fish, Halibut	75.4	18.6	5.2	...	1.0	35	565	1246
Fish, Mackerel	73.4	18.7	7.1	....	1.2	40	645	1422
Bacon, smoked, lean	31.8	15.5	42.6	...	11.0	130	2085	4597
Fowl	63.7	19.3	16.3	...	1.0	65	1045	2304
Capon, cooked	59.9	27.0	11.5	...	1.3	61	985	2172
Turkey	55.5	21.1	22.9	...	1.0	85	1360	2998
Turkey, roast	52.0	27.8	18.4	...	1.2	81	1295	2855
Eggs.	73.7	13.4	10.5	...	1.0	45	720	1587
Eggs, white boiled	86.2	12.3	0.2	...	0.6	16	250	551
Eggs, yolk boiled	49.5	15.7	33.3	...	1.1	107	1705	3759
Sugar and Starch.								
Cane-sugar, granulated	....	....	....	100.0	...	116	1860	4101
*Milk-sugar	....	....	....	....	...	116	1860	4101
Arrowroot	2.3	....	....	97.5	0.2	113	1815	4002
Cornstarch	....	....	....	90.0	...	105	1675	3693

Table 71.—Percentage-composition and Caloric Equivalents of Various Foods (*Continued*)

Food material	Water, per cent.	Protein, per cent.	Fat, per cent.	Carbohydrate, per cent.	Ash, per cent.	Calories per avoirdupois		Calories per kilogram
						Oz.	Lb.	
Sugar and Starch (Cont'd.).								
Sago	12.2	9.0	0.4	78.1	0.3	102	1635	3605
Tapioca	11.4	0.4	0.1	88.0	0.1	103	1650	3638
Cereals.								
Barley, pearled	11.5	8.5	1.1	77.8	0.1	103	1650	3638
Barley-flour	11.9	10.5	2.2	72.8	2.6	103	1640	3616
Oat-meal	7.3	16.1	7.2	67.5	1.9	116	1860	4101
Oats, various preparations	7.9	16.3	7.3	66.8	1.7	116	1855	4090
Rice	12.3	8.0	0.3	79.0	0.4	102	1630	3594
Wheat, cracked	10.1	11.1	1.7	75.5	1.6	105	1685	3715
Wheat-flour (average)	12.0	11.4	1.0	75.1	0.5	103	1650	3638
Wheat, whole, flour	11.4	13.8	1.9	71.9	0.9	105	1675	3693
Wheat, farina	10.9	11.0	1.4	76.3	0.4	105	1685	3715
Wheat, macaroni	10.3	13.4	0.9	74.1	1.3	104	1665	3671
Wheat-bread, average	35.3	9.2	1.3	53.1	1.1	76	1215	2679
Whole wheat-bread	38.4	9.7	0.9	49.7	1.3	71	1140	2513
Bread, Graham	35.7	8.9	1.8	52.1	1.5	76	1210	2668
Wheat-bread, toast	24.0	11.5	1.6	61.2	1.7	89	1420	3131
Zwieback	5.8	9.8	9.9	73.5	1.0	123	1970	4343
Rolls (average)	29.2	8.9	4.1	56.7	1.1	87	1395	3075
Corn-meal	11.6	8.4	4.7	74.0	1.3	108	1730	3814
Corn-bread	38.9	7.9	4.7	46.3	2.2	75	1205	2657
Corn-hominy	11.8	8.3	0.6	79.0	0.3	103	1650	3638
Crackers, various average	6.8	10.7	8.8	71.9	1.8	119	1905	4200
Vegetables.								
Asparagus, A.P	94.0	1.8	0.2	3.3	0.7	7	105	231
Beans, Lima	68.5	7.1	0.7	22.0	1.7	36	570	1257
Beans, string	89.2	2.3	0.3	7.4	0.8	12	195	430
Beets	87.5	1.6	0.1	9.7	1.1	13	215	474
Carrots	88.2	1.1	0.4	9.3	1.0	13	210	463
Celery	94.5	1.11	0.1	3.3	1.0	5	85	187
Lettuce	94.7	1.2	0.3	2.9	0.9	6	90	198
Onions	87.6	1.6	0.3	9.9	0.6	14	225	496
Peas	74.6	7.0	0.5	16.9	1.0	29	465	1025
Potatoes	78.3	2.2	0.1	18.4	1.0	24	385	848
Potatoes, sweet	69.0	1.8	0.7	27.4	1.1	36	570	1257
Spinach	92.3	2.1	0.3	3.2	2.1	7	110	243
Squash	88.3	1.4	0.5	9.0	0.8	13	215	474
Fruits.								
Apples	84.6	0.4	0.5	14.2	0.3	18	290	639
Blackberries	86.3	1.3	1.0	10.9	0.5	17	270	595
Dates, pressed	15.4	2.1	2.8	78.4	1.3	101	1615	3561
Figs, pressed	18.8	4.3	0.3	74.2	2.4	92	1475	3252
Grapes	77.4	1.3	1.6	19.2	0.5	28	450	992
Oranges	86.9	0.8	0.2	11.6	0.5	15	240	529
Peaches	89.4	0.7	0.1	9.4	0.4	12	190	419
Pears	84.4	0.6	0.5	14.1	0.4	18	295	650
Prunes, dried	22.3	2.1	...	73.3	2.3	88	1400	3087
Raspberries, red	85.8	1.0	...	12.6	0.6	16	255	562

TABLE 72.—COMPOSITION AND CALORIC VALUE OF DEFINITE PORTIONS OF FOOD PREPARED FOR USE[1]

Foodstuff	Quantity	Protein		Fats		Carbohydrates		Total calories
		Grams	Calories	Grams	Calories	Grams	Calories	
Meats:								
Beef-juice	1 fl.oz.	1.47	6.02	0.18	1.67	0	0	8
Scraped beef	1 round. tbsp. (20 gm.)	4.18	17.15	2.12	19.72	0	0	37
Beef-steak	1 round. tbsp. (20 gm.)	4.7	19.28	4.08	37.94	0	0	57
Roast-beef (lean)	1 round. tbsp. (10 gm.)	2.33	9.57	0.16	1.54	0	0	11
Fish, such as bluefish	1 round. tbsp. (20 gm.)	5.18	21.24	0.09	8.38	0	0	30
Lamb chop	1 round. tbsp. (15 gm.)	3.25	13.35	4.48	41.71	0	0	56
Chicken	1 round. tbsp. (10 gm.)	2.7	11.07	1.15	10.7	0	0	22
Bacon	1 slice	3.15	12.9	19.44	180.8	0	0	194
Soups:								
Beef soup	1 fl.oz.	1.32	5.4	0.12	1.12	0.33	1.35	8
Bouillon (canned)	1 fl.oz.	0.66	2.72	0.03	0.27	0.06	0.25	3
Chicken soup (canned)	1 fl.oz.	3.15	12.92	0.24	2.22	0.72	2.95	18
Consomme (canned)	1 fl.oz.	0.75	3.07	0	0	0.12	0.5	4
Tomato soup (canned)	1 fl.oz.	0.54	2.22	0.33	3.07	1.68	6.9	12
Vegetable soup (canned)	1 fl.oz.	0.87	3.57	0	0	0.15	0.62	4
Pea soup (canned)	1 fl.oz.	1.08	4.42	0.21	1.95	2.28	9.35	16
Dairy products and eggs:								
Butter	1 ball; or 1 round. tsp.	0.15	0.6	12.75	118.6	0	0	119
Milk, whole, 4–5–3.3	1 fl.oz.	0.99	3.99	1.2	11.16	1.5	6.15	21
Cream, gravity, 16–4.2–3.03	1 fl.oz.	0.91	3.75	4.8	44.64	1.2	5.1	53
Sk. milk, gravity, 1–5–3.3	1 fl. oz.	0.99	3.99	0.3	2.79	1.5	6.15	13
Sk. milk, commercial, 0.3–5.1–3.4	1 fl.oz.	1.02	4.18	0.09	3.84	1.53	6.27	11
American cheese	1 cu. in.	5.7	23.6	7.18	66.7	0.06	2.0	91
Hen's egg	1	6.60	27.1	6.00	55.8	0	0	83
Hen's egg, white	1	4.16	17.1	0.06	0.6	0	0	18
Hen's egg, yolk	1	2.89	11.8	5.99	55.7	0	0	68
Omelette { 3 tbsp. milk 3 eggs 1 h. tsp. butter	½	9.80	40.2	14.01	130.3	1.55	6.4	177
Vegetables:								
Beans, baked (home made)	1 h. tbsp.	3.61	14.8	4.25	39.57	10.95	44.87	99
Beans, baked (canned)	1 h. tbsp.	2.65	10.87	0.96	8.93	7.51	30.8	50
Butter beans	1 h. tbsp.	0.94	3.90	0.06	0.55	2.9	11.9	16
Lima beans	1 h. tbsp.	3.20	13.1	0.27	2.5	11.8	48.4	64
String beans	1 h. tbsp.	0.24	1.0	0.33	3.05	0.57	2.35	6
Beets	1 h. tbsp.	0.8	3.3	0.03	0.35	2.59	10.6	14
Beet tops	1 h. tbsp.	1.10	4.5	1.70	15.8	1.6	6.55	27
Cabbage	1 h. tbsp.	0.20	0.83	0.03	0.3	0.13	0.53	2
Carrots	1 h. tbsp.	0.18	0.73	0.05	0.53	1.13	4.63	6
Celery, creamed	1 h. tbsp.	0.47	1.93	1.66	15.43	1.15	4.73	22
Celery, uncooked	1 small stalk	0.17	0.7	0.02	0.17	0.48	1.97	3
Dandelion greens	1 h. tbsp.	1.19	4.9	0.50	4.7	5.33	21.9	30
Peas, green	1 h. tbsp.	2.05	8.43	1.04	9.7	4.38	18.37	36
Potato { Baked	Medium sized	3.77	15.5	0.20	1.9	32.07	131.5	149
	1 h. tbsp., ⅓ med. size	1.26	5.17	0.66	0.63	10.69	43.83	50
Boiled	1 h. tbsp. ⅓ med. size	1.28	5.13	0.05	0.47	10.45	42.83	48
Creamed	1 h. tbsp.	0.90	3.70	1.29	12.02	4.77	19.55	35
Mashed and creamed	1 h. tbsp.	1.30	5.35	1.50	13.95	8.90	36.5	56

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[1] Tbsp. = Tablespoonful—H. Tbsp. = Heaping Tablespoonful.—Tsp. = Teaspoonful. Home-made soups give amounts about as those in the table; cream-soups also, plus the value of the milk and flour added. The figures for cereals are for these boiled and without the addition of milk or sugar. Other cereals not mentioned (such as cream of wheat, Ralston's, Wheatena and Pettijohn's) may be calculated as for oatmeal. In all the puddings and in practically all the cakes egg is present. For exact ingredients see Locke's "Food Values."

TABLE 72.—*Continued*

Foodstuff	Quantity	Protein		Fats		Carbohydrates		Total calories
		Grams	Calories	Grams	Calories	Grams	Calories	
Vegetables *Continued:*								
Squash	1 h. tbsp.	0.68	2.80	0.41	3.80	6.8	27.9	34
Spinach	1 h. tbsp.	1.05	4.3	2.05	19.05	1.3	5.35	28
Tomato, canned	1 h. tbsp.	0.42	1.7	0.7	0.65	1.4	0.7	8
Tomato, uncooked	1 slice, ¼	0.6	2.45	0.1	0.92	2.0	8.2	12
	of med. size	0.6	2.45	0.1	0.92	2.0	8.2	12
Turnips	1 h. tbsp.	0.22	0.9	0.04	0.35	0.45	1.85	3
Fruits:								
Apple	Average size	0.45	1.8	0.45	4.2	16.20	66.4	72
Orange	Average size	1.5	6.2	0.25	2.3	21.25	87.1	96
Peach	Average size	0.64	2.6	0.13	1.2	9.86	40.4	44
Pear	Average size	0.78	3.2	0.62	5.8	19.81	81.2	90
Plum	Average size	0.32	1.3	0	0	6.69	27.4	29
Raspberries	1 h. tbsp.	0.37	1.1	0	0	3.44	14.1	15
Strawberries	1 h. tbsp.	0.25	1.02	0.15	1.4	1.85	7.57	10
Dates	1	0.16	0.65	0.2	1.93	5.86	24.03	27
Figs	1	0.84	3.44	0.058	0.54	14.47	59.28	63
Raisins	1	0.057	0.23	0.07	0.7	1.71	7.02	8
Apple, baked, 3 tbsp. to apple	1 h. tbsp.	0.2	0.83	0.19	1.8	9.77	40.03	43
Apple-sauce	1 h. tbsp.	0.08	0.33	0.33	3.1	15.5	63.6	67
Apricot, stewed	1 h. tbsp.	0.71	2.9	0.49	4.53	18.22	74.7	82
Jelly, currant	1 h. tbsp.	0.36	1.5	0	0	27.16	111.4	113
Prunes, stewed	1 prune + juice	0.25	1.02	0.05	0.47	11.15	45.72	47
Rhubarb, stewed	1 h. tbsp.	0.2	0.8	0.14	2.2	16.2	66.4	69
Breadstuffs:								
Corn bread	3 × 2 × ¾ in.	3.08	12.6	1.83	17.0	18.06	74.1	104
Rye bread	3½ × 3 × ½ in.	3.51	14.4	0.23	2.1	20.74	85.0	102
Cinnamon buns	1	4.23	17.3	3.24	30.1	26.59	109.0	156
Graham bread	3 ¾ × 2¾ × ½ in.	3.29	13.5	0.67	6.2	19.28	79.1	99
Rolls, French	1	3.32	13.6	0.98	9.1	21.72	89.1	112
Rolls, Vienna	1	3.83	15.7	0.99	9.2	25.43	104.3	129
Baker's wheat bread	3½ × 3 × ½ in.	2.76	11.3	0.39	3.6	15.93	65.3	80
Whole wheat bread	3½ × 3½ × ½ in.	4.07	16.7	0.38	3.5	20.87	85.6	106
Zweiback	3½ × 2 × ½ in.	1.47	6.0	1.49	13.9	11.03	4.52	65
Butter cracker	2 in. diam.	1.10	4.5	0.85	7.9	7.11	29.2	42
Graham cracker	3 in. sq.	0.80	3.3	0.75	7.0	5.9	24.2	34
Saltine	2 in. sq.	0.32	1.3	0.38	3.5	2.06	8.4	13
Soda cracker	3 in. sq.	0.59	2.4	0.55	5.1	4.38	17.7	25
Cereals:								
Farina	1 h. tbsp.	0.82	3.4	0.10	1.0	5.72	23.5	28
Hominy	1 h. tbsp.	1.10	4.5	0.10	0.95	8.9	36.5	42
Macaroni	1 h. tbsp.	1.5	6.15	0.75	7.0	7.9	32.4	45
Oatmeal	1 h. tbsp.	1.4	5.75	0.25	2.35	5.75	23.6	32
Rice	1 h. tbsp.	2.8	11.5	0.10	0.9	24.4	100.0	112
Desserts:								
Gingerbread	2 × 3 × 1 in.	3.48	14.3	5.4	50.2	38.1	156.2	221
Sponge cake	2 × 3½ × ½ in.	1.45	6.0	2.46	22.9	15.16	62.2	91
Apple pie	⅙ pie	3.91	16.0	12.35	114.9	53.93	221.1	352
Bread pudding	1 h. tbsp.	2.76	11.3	2.69	22.3	19.24	78.9	224
Chocolate pudding	1 h. tbsp.	2.49	10.25	3.95	36.75	13.91	57.05	54
Baked custard	1 h. tbsp.	3.65	15.0	3.71	34.4	10.25	42.05	92
Snow pudding	1 h. tbsp.	2.26	9.25	0.01	0.15	5.86	24.05	33
Tapioca pudding	1 h. tbsp.	1.93	7.93	2.04	18.9	7.42	30.4	57
Blanc Mange	1 h. tbsp.	2.38	9.75	2.45	22.85	8.41	34.5	67
Floating Island	1 h. tbsp.	2.49	10.23	2.54	23.63	4.5	18.47	52
Ice cream	1 h. tbsp.	2.6	10.7	5.08	47.25	8.86	36.35	94
Spanish cream	1 h. tbsp.	5.85	24.0	3.68	34.25	10.62	43.55	102
Ginger snap	2¼ in.	0.33	1.4	0.43	4.0	3.8	15.6	21
Ladyfingers	4 in.	1.76	7.2	1.0	9.3	14.12	57.9	74
Molasses cookies	4 × 3 in.	1.44	5.9	1.74	16.2	15.14	62.1	84
Sugar cookies	3 in.	0.77	3.2	1.12	10.4	8.05	33.0	47
Honey	1 tbsp.	0.12	0.5	0.0	0.0	24.36	100.0	101
Sugar granulated	1 h. tbsp.	0.0	0.0	0.0	0.0	30.0	123.0	123
Maple syrup	1 tbsp.	0.0	0.0	0.0	0.0	21.4	87.9	88

VITAMINS

Perhaps best in this connection may be studied the substances known as *vitamins*, or *accessory food-factors*. A very large amount of medical literature has accumulated since the description of these by Funk.[1] Previous work by Eijkman,[2] Hopkins[3] whose studies began in 1906, and others had suggested the presence and necessity of these food-elements in the diet. Satisfactory reviews on the subject have been published by Hess,[4] Moore,[5] Eddy,[6] Sherman and Smith[7] and McCollum.[8] In any discussion of vitamins there should be mentioned also the names of Osborne, Mendel, Mellanby, Ellis, Steenbock, Zilva, Hart, Chick, Park, Shipley, Simmonds, Drummond, Coward, McCarrison, and others.

The exact chemical nature of vitamins is not known, but it is certain that their presence is essential for the satisfactory growth and development of the infant. The lack of them results in deficiency-diseases, often called "avitaminoses." The term "vitamin" has been justified by use; yet, as pointed out by McCollum, these substances are not amines, as first suggested by Funk; and although the name was proposed by him, it was only as a temporary one until their true chemical structure was discovered. The exact way in which the vitamins act is still undetermined, various theories having been advanced; as, for instance, that they regulate mineral metabolism (Park);[9] that their absence impairs the function of the formation of lymphocytes (Cramer, Drew and Mottram);[10] or results in congestive and hemorrhagic conditions in the intestinal tract (McCarrison);[11] or lowers tissue-oxidation (Sajous).[12] It has been claimed, too, that they help to protect the body against bacterial infection, and that they stimulate the glands of internal secretion. Human milk usually contains them in sufficient amount, and certainly so if the mother is being properly fed; but inasmuch as the vitamins appear to be derived from the food and not directly manufactured in the body, it is possible that a mother who is on a diet deficient in these essential substances will supply to her infant a milk lacking in certain of them. This has happened particularly in times of war and famine. Similarly cow's milk, although it ordinarily has a satisfactory vitamin-content, may be lacking or impoverished in this respect, if the food of the cow is inadequate. This is likely to occur, however, only in the case of the water-soluble vitamin B (Osborne and Mendel;[13] Daniels, Byfield and Loughlin[14]), and the water-soluble vitamin C (Hess, Unger and Supplee).[15]

Three distinct varieties of vitamins have been described, and it is possible that others exist. The 3 now universally recognized are named:

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[1] Jour. State Med., London, 1912, XX, 341.
[2] Arch. f. path. Anat. u. Physiol., 1897, CXLVIII, 523.
[3] Jour. Physiol., 1912, XLIV, 425.
[4] Jour. Amer. Med. Assoc., 1921, LXXVI, 693.
[5] Northwest Med., 1921, XX, 246.
[6] The Vitamin Manual, 1921.
[7] The Vitamins, 1922.
[8] The Newer Knowledge of Nutrition, 1922.
[9] Physiol. Rev., 1923, III, 163.
[10] Lancet, 1921, I, 963.
[11] Jour. Amer. Med. Assoc., 1922, LXXVIII, 1
[12] New York Med. Jour., 1923, CXVII, 325.
[13] Jour. Biol. Chem., 1918, XXXIV, 537.
[14] Amer. Jour. Dis. Child., 1919, XVIII, 546.
[15] Jour. Biol. Chem., 1920, XLV, 229.

(1) fat-soluble vitamin A; (2) water-soluble vitamin B; and (3) water-soluble vitamin C.

(1) The fat-soluble vitamin A is present in certain animal fats, such as those of eggs and butter; and is found to a much greater extent in cod-liver oil. It exists also in green vegetables, such as spinach, cabbage and peas, but is usually absent in roots and tubers, except those with yellow pigment such as turnips and carrots. It is not found in vegetable fats. If this vitamin is absent from the diet of the experimental animal xerophthalmia with xerosis of the conjunctiva and keratomalacia will develop. This same condition may occur in the human infant (Bloch;[1] Ross;[2] Mori;[3] Wilson and DuBois;[4] Schwartz[5]). In the early stage of deprivation of this vitamin night-blindness (hemeralopia) may be present (Holm).[6]

(2) The water-soluble vitamin B (antineuritic vitamin) is very widely distributed in foods. Most of such vegetables as cabbage, tomato, spinach, onions, beets, potato, carrots and beans are dietary sources of this food-factor, and it is also found in the glandular organs of animals (kidney, liver, pancreas), in milk, in eggs, and in the germ of the cereal grains. When absent from the diet one of the chief symptoms is a polyneuritis. This is readily produced in the experimental animal, and may occur in infants as noted by Andrews[7] and others.

(3) The water-soluble vitamin C (antiscorbutic vitamin) occurs in certain common fruits and vegetables. Oranges, lemons, grape-fruits, and, to a lesser extent, apples and bananas, are sources of supply, and the berries have a large content. Tomatoes, either raw or canned, are particularly rich in it, and most of the green vegetables, such as lettuce, string-beans and cabbage, will yield a fair amount. While less is present in roots and tubers, yet young carrots, onions, turnips and potatoes have been found to possess sufficient to be protective. Egg-yolk probably contains a limited quantity, while the amount in milk is dependent on the fodder of the cow, as previously stated.

There is another protective substance often called vitamin D which exerts an antirachitic action (McCollum, Simmonds, Shipley and Park).[8] (See Rickets, p. 681.) This, while present in cod-liver oil and to a much less extent in butter, is also found in cocoanut-oil. It can be shown to be different from and independent of the fat-soluble vitamin A. The lack of vitamin D acts only in the presence of a disturbed relation between the intake of calcium and of phosphorus respectively. Bloch[9] maintains that the antirachitic factor is not a substance but is simply the energy of the ultra-violet ray.

Another accessory food-factor called vitamin E or "Substance X" is claimed by Evans and Bishop[10] and by Sure[11] to be necessary for reproduction in the rat. It is found in wheat-embryo, fresh green lettuce oats, polished rice, yellow corn and in very small amounts in milk.

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[1] Jahrb. f. Kinderheilk., 1919, LXXXIX, 405.
Amer. Jour. Dis. Child., 1924, XXVII, 139.

[2] Amer. Jour. Dis. Child., 1921, XXII, 232.

[3] Quoted by McCollum, Jour. Amer. Med. Assoc., 1923, LXXXI, 894.

[4] Amer. Jour. Dis. Child., 1923, XXVI, 431.

[5] Jour. Amer. Med. Assoc., 1925, LXXXV, 2025.

[6] Amer. Jour. Physiol., 1925, LXXIII, 79.

[7] Philippine Jour. Sci., Sect. Trop. Med., 1912, VII, 67.

[8] Jour. Biol. Chem., 1922, L, 5.

[9] Amer. Jour. Dis. Child., 1926, XXXI, 315.

[10] Jour. Amer. Med. Assoc., 1923, LXXXI, 889.

[11] Jour. Biol. Chem., 1924, LVIII, 693.

Pellagra at one time was thought to be an avitaminosis. Later the evidence indicated that it was a deficiency disease only in the sense that it was due to lack of certain amino-acids. More recently the vitamin-deficiency theory has again been sustained. War-edema and coeliac disease have not been proven to be due to vitamin-deficiency. It seems probable, although difficult to demonstrate, that certain states of malnutrition and lack of resistance to infection may develop in vitamin-deficiency, without the actual presence of the classical symptoms of xerophthalmia, polyneuritis or scurvy.

The fat-soluble vitamin A is not easily inhibited or destroyed by such heat as would be produced in the boiling of milk, whereas the water-soluble vitamins B and C are much more readily affected by it. The length of time heat is applied has more effect than its degree. Heat is not the only factor involved in the destruction or inhibition of the vitamins. Oxidation plays a rôle, since if air is excluded during the heating process the vitamin-content is greater than otherwise. Further, if the food is alkalinized before heating there is less vitamin retained than if the reaction were acid. Aging of the food and bacterial contamination also play a part in vitamin-destruction. The effect of drying upon milk and vegetables is of importance, inasmuch as these foods are used at times in a canned and dehydrated state. The antiscorbutic vitamin is especially sensitive to the drying process (Gralka)[1] but a certain amount of it is retained if the process is properly carried out. This is true of milk (Hess and Unger;[2] Cavanaugh, Dutcher and Hall[3]) and of certain vegetables (Givens and Cohen;[4] Givens and McClugage[5]) and fruits (Givens and McClugage).[6]

From the knowledge which we possess of the vitamins, we may deduce the following practical applications: (*a*) When it is consistent with digestibility food should be cooked rapidly at a high temperature rather than by prolonged heating at a low temperature; (*b*) If milk is cooked for any length of time this should be done in a double boiler to prevent oxidation; (*c*) If lime-water or other alkali is added to milk this should not be done until after boiling; (*d*) Milk should be fresh, and only such vegetables used as have been recently gathered; (*e*) An infant fed on sterilized or heated milk should be protected by the administration of fresh fruit-juice. If these precautions are observed there will be little need to fear vitamin-deficiency in the diet of the infant or child. It is not necessary to use commercial vitamin-preparations, many of which are inactive (Hess, Moore and Calvin;[7] McCollum and Simmonds[8]). The extravagant claims made for yeast can be entirely disregarded. This fungus when given in fresh form, as in the ordinary yeast-cake, is capable of doing harm (Ladd).[9]

Appended in tabular form is a list of the vitamins present in various selected food-stuffs.[10]

[1] Jahrb. f. Kinderheilk., 1923, C, 265.
[2] Amer. Jour. Dis. Child., 1919, XVII, 221.
[3] Amer. Jour. Dis. Child., 1923, XXV, 498.
[4] Jour. Biol. Chem., 1918, XXXVI, 127.
[5] Jour. Biol. Chem., 1919, XXXVII, 253.
[6] Amer. Jour. Dis. Child., 1919, XVIII, 30.
[7] Jour. Amer. Med. Assoc., 1922, LXXVIII, 1441.
[8] Jour. Amer. Med. Assoc., 1922, LXXVIII, 1953.
[9] Trans. Amer. Pediat. Soc., 1921, XXXIII, 136.
[10] Hygeia, 1923, I, 62.

Table 73.—Vitamin Content of Various Foods

	"A"	"B"	"C"		"A"	"B"	"C"
Bread, white (water)	?	+	—	Tomatoes (raw or canned)	++	+++	+++
Bread, white (milk)	+	+	?	Beans, kidney	*	+++	*
Bread, whole-wheat (water)	+	++	?	Beans, navy	*	+++	—
Bread, whole-wheat (milk)	++	++	?	Beans, string (fresh)	++	++	++
Barley (whole)	+	++	—	Cabbage, fresh, raw	+	+++	+?
Corn, yellow	+	++	—	Cabbage, cooked	+	++	+?
Oats	+	++	—	Carrots, fresh, raw	++	++	++
Meat, lean	— to +	+?	+?	Carrots, cooked	++	+	+
Beef fat	+	—	—	Cauliflower	+	++	+
Mutton fat	+	—	—	Celery	*	+	*
Pig-kidney fat	++	—	—	Cucumber	*	+	*
Oleomargarine	+	—	—	Dandelion greens	++	++	+
Liver	++	++	+	Eggplant, dried	*	++	*
Kidney	++	++	+?	Lettuce	++	++	+++
Brains	+	++	+?	Onions	*	++	++
Sweetbreads	+	+	*	Parsnip	—?	++	*
Fish, lean	—	+	*	Peas	++	++	+?
Fish, fat	+	+	*	Potatoes (boiled 15 min.)	*	++	+?
Fish, roe	+	++	+?	Potatoes (boiled 1 hour)	*	++	+?
Milk, fresh	+++	++	+v	Potatoes (baked)	*	++	+
Milk, condensed	+++	++	+v	Sweet potatoes	++	+	*
Milk, dried, (whole)	+++	++	+v	Radish	*	+	*
Milk, skimmed	+	++	+v	Rutabaga	—?	++	+++?
Buttermilk	+	++	+v	Spinach, fresh	+++	+++	*
Cream	+++	++	+v	Spinach, dried	+++	++	*
Butter	+++	—	—	Squash, Hubbard	++	*	*
Cheese	++	*	*	Turnips	—?	++	*
Cottage cheese	+	*	*	Apples	+	+	+
Eggs	++	+	+?	Bananas	+?	+?	+
Almonds	+	+	*	Grape juice	*	+	+
Coconut	+	++	*	Grapefruit	*	++	++
Hickory nuts	*	++	*	Lemon juice	*	++	+++
Peanuts	+	++	*	Orange juice	+	++	+++
Pecans	*	+	*	Prunes	*	+	—
Walnuts	*	++	*	Raspberries (fresh or canned)	*	*	+++

+	contains the Vitamin	?	doubt as to presence or relative amount
++	good source of the Vitamin	*	evidence lacking or insufficient
+++	excellent source of the Vitamin	v	variable
—	no appreciable amount of the Vitamin		

CHAPTER VIII

DIET IN SICKNESS

Only general rules can be given here, in addition to, or by way of summary of, what has been said in various places under the different headings of infant-feeding, and of what will be included in the study of the individual diseases.

In the case of breast-fed infants under 1 year, the development of acute gastric disturbance often renders it advisable to stop nursing entirely for 12 or more hours, giving barley-water or other cereal decoction in its place. The mother's milk may be examined and, if faulty, modified if possible. The withdrawal of the breast may continue for a few days if necessary, but be made permanent only if symptoms of indigestion are persistent or repeated and the infant ceases to thrive in other respects. Mixed feedings should be tried before weaning the child completely.

In the case of bottle-fed infants acutely ill with some digestive disturbance, a general rule which can be wisely followed in most instances is to administer a purgative and to stop the milk-mixture entirely for at least 24 hours, giving a weak cereal decoction, such as barley-water, in its place. After this a graduated but fairly rapid return to the former food may be made. It is a mistake to institute any permanent radical change in a diet which has previously agreed well. Should, however, the digestive disturbance constantly recur, and the infant cease to do well in other respects, being a case of "feeble digestion," a very decided alteration of the diet or of other factors connected with it should be made promptly, since the longer the disturbance continues, the greater the effect upon the infant, and the harder it is to restore it to a condition of health.

What change shall be made can be determined only by a careful study of the individual infant. When persistent *vomiting* is the symptom present, it is oftenest the fat which is at fault, and the percentage of this must be reduced very greatly. Food in which skimmed-milk is the basis is frequently very useful in this condition. If reduction of the fat does not succeed, the sugar should be suspected. Sometimes the employment of lactose instead of maltose or saccharose is efficacious; sometimes, contrary to what is to be expected, the reverse is the case; sometimes no sugar is well borne. Buttermilk has the advantage that it is low both in fat and sugar, and is sometimes very serviceable. Even when fortified with wheat-flour and cane-sugar, it is still of value in many cases of vomiting, provided this is not dependent upon a sugar-intolerance. Often the administration of a thick gruel containing from 5 to 10 per cent. of rice-flour or farina is useful under these circumstances. In this connection we must not forget that it is not alone the nature of the nourishment but other factors also which may cause and maintain vomiting. Often the food is given in too large an amount, too frequently, or too rapidly. Whether it is better to give a large quantity of a diluted food or a smaller amount of a more concentrated nourishment is a question to be settled often only by trial. Certainly the intervals of feeding should as a rule be lengthened, and the possible existence of other forms of faulty management sought for. (See Action of Milk-elements, p. 128 and Vomiting, Vol. II, p. 60.)

When *diarrhea* is the prominent symptom, again the fat is first to be suspected, especially if the stools contain large numbers of soft, white curds; or the sugar if the stools are green or yellow-green and have a sour odor. Should the infant be thriving, the condition not acute, and the stools not very frequent in number, no immediate change need necessarily be made, but the condition carefully watched. Diarrheal stools if of a foaming character with irritation of the buttocks depend upon an excess of sugar. As lactose and maltose-dextrin, if in large amount, are liable to produce this disturbance, saccharose may be substituted; but it is better to begin with no addition of sugar, and to make this very gradual. The employment of malt-soup, modified in ways to diminish the amount of converted starch present, gives good results in some instances, but in others increases the diarrhea. Buttermilk-mixture (p. 153) is also useful in many instances, and in others casein-milk (p. 157); both having a low percentage of lactose and a high percentage of casein; the latter a moderate amount of fat and the former replacing this by a high carbohydrate-addition in the form of saccharose and flour, which may be tolerated when lactose is not. The casein-milk, at first without sugar-addition, is to be selected when it is thought that the sugar disagrees; the other when the fat is especially suspected.

Constipation may call for a modification of the diet. (See also Vol. II, p. 128.) Apart from other causes it may depend upon too small an amount of food ingested as a whole, or of one of the elements, usually fat or carbohydrate. Constipated, thriving children may have such complete absorption from the intestine that little waste remains. When the stools are dry and light-colored and perhaps offensive, there is generally an excess of fat and sometimes of protein or a lack of sufficient carbohydrate. The increase of the latter will often change the character of the stools and relieve constipation, and especially so if a combination of starch with a maltose-dextrin preparation be employed, such as exists in malt-soup.

The significance of *colic* is very uncertain. Sometimes it depends in no way upon the character of the food, and it is a common attendant upon constipation. When the food is without doubt the cause, the exact method of production of the colic is still frequently not clear. An excessive carbohydrate-intake, especially of starch, is often a factor, as is also sometimes too high a percentage of protein. This protein-indigestion occurs in probably but a relatively small number of cases. The symptoms are indefinite and not entirely understood, the most suggestive in addition to the colic being the occurrence of offensive stools with a putrefactive odor, either diarrheal or constipated, and sometimes of hard, yellow-white protein curds in the passages if the milk has been given raw. In the way of dietetic modification, the casein may be reduced by the employment of whey-mixtures with cream, or the milk may be boiled or be peptonized. In cases due to excessive carbohydrate fermentation in breast-fed infants Grulee[1] recommends the administration of 1 gm. (15 grs.) of casein in a little water before nursing, while ⅓ of a tube of liquid culture of the lactic-acid bacillus is given twice a day. (See also Colic, Vol. II, p. 94.)

The employment of thin cereal waters in the case of sick children has already been referred to. In some instances, especially in infants past the age of 6 months, benefit in digestive disorders is obtained by giving a stronger cereal-food, entirely without milk at first. This is sometimes digested much better than either fat or sugar. Small and gradually increasing amounts of milk should be added as soon as possible, inasmuch as the diet is generally too insufficient in protein for employment for any considerable time. One of the most trying evidences of malassimilation of the food is that in which, without sufficient vomiting or diarrhea to account for it, there is a persistent failure to gain weight, or even a loss of it. (See Vol. II, p. 57.) This may depend upon an insufficient amount of nourishment, but oftener is the final result of the giving of food which was too strong, especially in fat. The employment of a high carbohydrate-diet with a low fat-percentage is at times one of the best remedies. Where a mere insufficient amount of food offered is the cause, the condition is readily curable. In other instances the appetite is very poor, and food is refused. Here good may come from lengthening the feeding-intervals.

The feeding of all these forms of disturbed digestion in infants will be studied more in detail in considering the diseased conditions in which the symptoms occur.

In children over 1 year of age the presence of persistent weakness of digestion may necessitate the continuance of the use of milk in modifications which would suit younger normal infants. In other cases the best course is to eliminate milk more or less completely for a time. The giving of beef-juice and of scraped underdone meat is often useful under such conditions, while the effects of fat and of carbohydrate, especially starch, must be watched carefully. Each case is a rule to itself, and the diet must be studied and altered as with infants under 1 year. Particularly about the age of 2 years or later there is liable to develop a form of chronic indigestion which depends upon too free a use of amylaceous food and sometimes of fat. This starchy indigestion requires a modification of the diet by which milk, often skimmed, and meat, and, to some extent, green vegetables constitute the principal articles of food, and starch is reduced to a minimum. (See Vol. II, p. 142.) In my own experience an excess of protein in the diet after the 2d year is less often harmful than is an excess of carbohydrate.

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[1] Infant Feeding, 1922, 162.

In many of the febrile diseases in infants or older children vomiting is the first symptom. When this is active, little or no effort should be made to give nourishment. So, too, the dislike for food often seen in acute febrile disorders need be no cause of alarm. It is a natural result of the impaired digestive power always attendant, and nourishment should not be urged. If the attack, however, is prolonged, or when the condition is such that inanition is threatening from lack of food, the physician must feed sufficiently, although cautiously. It may even be necessary to employ gavage. Curiously, in some cases of obstinate vomiting in infants, food given by gavage, immediately following lavage, will be retained when swallowed nourishment is not. (See Gavage, p. 278.) It is seldom that a sick child may not have water as often and as much as it desires. It is chiefly when the administration of water appears to excite vomiting that the amount given must be restricted. Thirst is very intense in the febrile disorders, and it has been the misguided practice of many of the laity to limit the amount of water. This is both dangerous and cruel. The water should be cool, but not cold. Sometimes, for older children, a carbonated water is to be preferred. Nursing infants with fever should have water given from a spoon or bottle, since, if allowed to quench their thirst by nursing, they may readily over-feed themselves. On the other hand, it sometimes happens, where the giving of nourishment is important, that the existence of thirst may be taken advantage of in older children, since, in order to quench it, the child may be induced to drink cool milk if water is withheld; or water may be given as a reward after the milk is taken.

Great regularity should exist in the feeding of sick children. Liquid nourishment should be given every 3 hours, or sometimes every 1 or 2 hours or oftener when very little is taken at a time; but as a rule the interval should be the longer one. The impairment of the digestive power present in the acute febrile disorders, not themselves of digestive origin, often makes it advisable that the food be weaker than that used in health, and especially that the fat be reduced. Infants who have been receiving a modified milk-mixture should have this weakened, and children who have been on solid food require a liquid diet. Of all forms of liquid diet for older children that oftenest serviceable is milk, which should be diluted and may often be alkalinized with lime-water with advantage. Where milk is disliked by or, as in some digestive disorders, disagrees with the child we may employ such foods as egg-water or raw egg given in other ways, beef-juice, commercial peptonized beef-preparations, and broths thickened with starchy food. Clear broths, though appetizing, have little nutritive power. Jellies do not nourish to any extent, but well-made ice-cream is often a valuable food in selected cases for children who have lost appetite and need feeding. It should be given, however, rather as a reward, and not made a constant article of diet, as its sweetness is liable to cause or increase indigestion.

In long-continued illnesses often one of the greatest problems is to maintain the state of the general nutrition in a satisfactory manner. In giving, then, the restricted diet referred to, the greatest caution must be exercised not to overdo the matter. I have repeatedly seen children whose chief ailment after some illness was that they had been, and still were, greatly underfed. In cases of obstinate vomiting food may sometimes be given in the form of nutrient suppositories, or, oftener, of nutrient enemata. These, however, do not fill the place which they do in adult life, owing to the difficulty, especially in infancy, of having them retained and the deficient absorption which occurs. As a rule, they are unsatisfactory at this period of life.

The diet as it relates to specific conditions, such as nephritis or diabetes, will be discussed in detail under the appropriate headings.

CHAPTER IX

CHARACTERISTICS OF DISEASE IN INFANCY AND CHILDHOOD

The diseases of early life vary decidedly from those occurring later. Not only are the causes often different, but the reaction of the growing tissues in early years is not the same as in adult life. Anatomical and physiological distinctions also exist. There is consequently seen a tendency to the development of certain diseases in infancy and childhood, and an immunity toward others. Certain maladies are peculiar to early life; others exhibit clinical manifestations characteristic of that period. The susceptibility of the incompletely developed nervous system is very great, often masking the real nature of the disorder. Trifling factors thus produce general symptoms which are, or appear to be, severe out of all proportion to their causes, similar agencies acting in adults giving rise to no symptoms of moment. The initial effect of deleterious influences is often unusually marked in early life, and the development of symptoms very rapid and apparently severe; while, on the other hand, the recuperative power is great, and convalescence is speedy. Various causes render the examination of a sick child difficult. There are also marked peculiarities at this period in the reaction of the system to certain drugs, some of the materia medica being unusually well-tolerated, and others not at all so.

After early childhood is passed, and particularly after the age of 7 or 8 years, the peculiarities of disease incident to the age of the patient are less marked.

ETIOLOGY

Among the causes predisposing to the development of certain disorders in children direct or indirect *inheritance* plays an important rôle. Syphilis in either parent is directly inherited, as probably are very occasionally such of the acute infectious diseases as typhoid fever, scarlatina, and some others, and not infrequently variola. Tuberculosis existing in the parents certainly predisposes to its development in the offspring Rheumatism and gout exert a similar predisposing influence, as do various nervous disorders, such as epilepsy, insanity, a neuropathic tendency, and some of the muscular dystrophies. Some of these diseases may not actually show themselves until childhood is past, although the seeds of them are present in the system. Of importance, too, is the existence of various *diatheses* (see p. 745), present at birth or developing later, to which the occurrence of certain symptoms seen in early life is to be attributed.

To be mentioned also is the etiological influence upon the diseases of the infant of the various *morbid conditions incident to fetal life and to birth,* which will be referred to again (p. 235). Maternal impressions have frequently been considered a powerful factor in the production of some of these; but although much evidence has been adduced in favor of this view, the element of coincidence is too often ignored, and positive proof of any causal relationship is entirely unsatisfactory.

Imperfect feeding and hygiene are among the most active causes of disease in infancy and childhood. As a result arise the many forms of disturbed digestion and their consequences; such constitutional conditions as rickets and scurvy; the disordered states of the respiratory apparatus so common in children, and the diseases which depend upon lack of proper care of the nervous system. The influence of *school-life* is responsible for many nervous ailments, affections of the eyes, deformity of the spine, and disorders of the general health.

Infection, finally, has unusual etiological power in children, the great majority of cases of acute infectious diseases being witnessed at this period. This is partly due to a greater degree of susceptibility; partly to a much greater opportunity of exposure; and partly to the fact that most adults have already become immune through earlier occurrence of these affections.

GENERAL METHODS OF EXAMINATION AND DIAGNOSIS

There are a few respects in which the study of disease in infancy and childhood is easier than in adult life. Existence having been of shorter duration, there is generally a shorter history to be obtained. There are fewer complicating diseases, at least in the acute affections, and there are fewer previous various bad habits of living to be taken into account.

On the whole, however, the subject presents many and varied difficulties. Many affections exhibit in childhood symptoms different from those of adult life, and the significance of even the same symptom often differs; while there are also many diseases which are almost or entirely peculiar to early life. Again, the infant cannot, without speech, describe its symptoms, and the older child often cannot be trusted to give an accurate account. The inability to control the patient presents another difficulty, and the constant crying of an infant may render a satisfactory examination impossible. Considerable skill, patience and tact are therefore required.

Obtaining the History.—As a sequence to what has been said, dependence must be placed on the gathering of a careful and complete clinical history from the mother or nurse. In infancy there is, of course, no history obtainable from the patient, and even up to the age of 6 or 7 years, the child either refuses to answer questions on account of timidity, or makes replies which are not dependable. The presence of pain, for instance, may be denied when it really exists, or maintained when not existing, and the localization by the patient is very misleading, pain in the chest being perhaps referred to the throat or abdomen, and so on. There should be some definite order followed in the procuring of the history from the attendant. Considerable latitude is, of course, allowable, dependent upon the nature of the ailment; yet, in general, the first point should be a brief statement of the present complaint limited to a very few words; next, the past clinical history of the patient; third, the detailed account of the present illness, and, last, the family history. To reverse this order renders the asking of numerous useless questions unavoidable.

After eliciting very briefly the chief symptoms from which the child seems to be suffering, it is often well to allow the mother to give as full an account as possible of the case, in her own way and undisturbed by interruptions, unless these become necessary. Ordinarily the history may best be heard in the presence of the child, who, meanwhile, is growing accustomed to the physician's presence, and who, unknown to itself, is at the same time under the quiet observation of the physician's eye. Yet there are numerous exceptions to this. If the mother's mind is distracted by the child's crying, she will be unable to answer questions satisfactorily. So, too, if the child is observed to be listening too intently to the questions about itself, it may readily be affected injuriously, and in this event the history should be obtained in another room. While, as a rule, the mother's opinions are of little value, her statements of *what she has noticed* must be listened to and weighed with care; since by her constant association with the child, and by her natural anxiety, her powers of observation have sometimes become very keen, and she is rendered peculiarly able to detect even slight changes from the ordinary condition of health. After the mother has finished her account, she should be subjected to careful questioning by the physician, much, or all, of the child's life being passed in review. Leading questions must be avoided as much as possible, for the suggestions which these offer are very liable to lead to erroneous statements.

Previous History.—In the case of infants this should date from birth; and this applies with equal truth to many older subjects. Among the data concerning which information will be sought are the existence of prematurity, of asphyxia following birth, the nature of the labor, the process of dentition, the time when sitting and walking were begun, the birth-weight and the subsequent alterations, the frequency of micturition, the freedom of perspiration, the character of the sleep, the existence of mouth-breathing, the state of the bowels, the condition of general nutrition and the time when any change in this was seen, the condition of the nervous symptoms in general, and the psychic development, often best shown by the degree of interest taken and the ability to understand and to talk. Particular attention must be paid to the minutest details of the different methods of feeding which have been employed and to the effects of these; whether weaning has taken place, and, if so, why. The statement, for instance, that the baby had been fed on modified milk is not sufficient. The *proportions* of milk, water and the like should be ascertained. Especial care must be given, too, to discovering the date, nature, mode of onset and duration of all previous illnesses. This is often a matter of some difficulty, and may require careful, patient questioning.

Present Illness.—We can next take up the illness from which the child is now suffering. The exact date of onset and the sequence of the symptoms is a matter of importance, and may require time and skill to elicit. At least the mother is generally able to say at what time she considered the child to have been entirely well. A detailed description of the symptoms which have been observed is to be sought for. The nature of the questions varies, of course, with the case. The degree and persistence of fever is important, although the history in this respect is of little value unless the thermometer has been used. Interrogations should be made regarding the state of the general nervous system, as, for instance, the occurrence of restlessness, crying, sleeplessness, drowsiness, coughing, twitching, and the existence of pain; the degree of apparent exhaustion or prostration determined by the previous desire of the patient to be in bed or out and the presence or absence of a desire to play with toys; the position in bed, and the like. In digestive disorders the history should be obtained of the mode of onset and the nature of the food at the time, the history of vomiting or diarrhea and the number of daily occurrences of these, the character of the vomitus or the stools, and the state of the appetite.

Family History.—The nature of the questions asked will naturally depend upon what has already been learned. They should include the clinical history of other children of the family, the parents, and often of the grandparents and other direct or indirect antecedents when the existence of a possible inheritance is involved. Among the matters which need to be investigated are the general health of the family and antecedents; the occurrence in them of tuberculosis, nervous disorders, rheumatism, gout, insanity, alcoholism; the number of children living or dead and the causes of death. The existence of parental syphilis must usually be approached with caution. In place of direct questions information may be gained, for instance, regarding the occurrence of numerous miscarriages, the history of maternal cutaneous eruptions, and the like.

Method of Examining the Child.—Now follows the direct examination of the child—an examination which, as stated, has been all this while quietly going on to a certain extent. Very young infants take no notice of the physician's presence. Older children, however, often have much fear of strangers, and perhaps, from previous experience or suggestion, especially of physicians. A quick glance at the patient on entering the room, or the manner in which a word of cheery greeting is received, will frequently reveal something of the peculiarities of the child in this respect. It is often a good plan to seem to ignore absolutely even the existence of a somewhat timid or irritable child. It quite commonly happens that the confidence which this inspires, as well as a certain degree of pique which it occasions, will soon cause the child itself to make advances toward a further acquaintance; whereupon a skilful response to its overtures will soon establish the most friendly relations between doctor and patient. Avoidance of all hurry, and the use of gentle words and actions, often aid in rendering a child willing to submit quietly to an examination. Sometimes this can be made to appear a game which the physician is playing with his little patient.

In the case of many children whose timidity seems too great to be overcome, and especially with those who have been spoiled by indulgent parents, and who seem to resent in an ugly spirit the physician's presence, nothing whatever is gained by delay, and it is best to proceed quietly, gently, yet firmly with the examination, regardless of any objections made. Many such children, observing that the physician goes on with his work in spite of protest, learn the uselessness of this and behave better at future visits. Very often the difficulty is with the mother rather than with the child.

A sincere love for children, a quick recognition of a child's peculiarities of disposition, a ready adaptability to meet them, and above all, continued experience rapidly lessen the difficulties in the study of disease in early life. Fortunately, practice enables a physician to make an examination of many unruly children almost as satisfactorily as of those who are quiet and docile.

Order of Procedure.—The order of procedure in making the physical examination depends somewhat on circumstances, and the greatest flexibility in the plan is to be allowed. Those examinations are first made either which arouse least objection or which are most important. If the child is sleeping, or often if it is lying awake in its bed, a superficial inspection of it may be made. Under the same circumstances, the time is favorable for determining the character of the radial pulse and for the palpation of the abdomen, for should the infant begin to cry satisfactory examination of the abdomen is rarely possible. Next, the temperature should be taken before its elevation can have been increased by prolonged crying. Following this, the general inspection should be completed, the child being undressed for this. Should crying or coughing occur the character of these may be noted.

The physical examination of the thorax may next be carried on, auscultation being practised first, if the child is quiet, since this often causes less alarm and less tendency to cry than percussion does. At sometime during quiet the reflexes may be tested. Last of all the mouth and throat must be inspected and frequently the nose, eyes, and ears. The urine should be obtained for examination and in many instances the investigation is not complete without a study of the blood and of the cavities of the thorax, abdomen, or spinal canal for the presence of fluid and thecharacter of this. Special cases require a radiological and bacteriological study.

Some of these methods will now receive consideration in fuller detail. The clinical significance of the observations made will be considered in the chapter upon Symptomatology (p. 219).

Inspection.—This furnishes often greater results than any other method for arriving at a diagnosis. It is the first examination to be made, and begins, although at a distance, as soon as the physician comes into the presence of the patient. After a general inspection made of the child while asleep or awake, it should, when possible, be undressed, wrapped in a warm blanket and laid on the mother's lap or on the bed. When a condition of exhaustion, low bodily temperature, the presence of pain on movement, the existence of diseases of the upper respiratory tract, or other cause renders complete undressing undesirable, the body must be examined part by part throughout before the study is ended. Unless the child is thus undressed very valuable data would be necessarily undiscovered; such as differences in the form and degree of motion of the two sides of the chest; the position of a visible apex-beat; the presence of epigastric or episternal pulsation or retraction; the occurrence of important eruptions of the skin; the distention of the abdomen or of the abdominal veins; visible peristalsis; beading of the ribs; etc. In addition to these matters inspection must take cognizance of the state of the general nutrition; the physiognomy; the color of the face and lips; the shape of the head; the condition of the fontanelle and of the eyes; the shape of the abdomen; visible glandular enlargement; the position of the body in bed; the shape and movements of the limbs; the condition of the genitals; the character of the sleep; the degree of restlessness or of extreme quiet; the method of nursing; the existence of nasal or oral discharge; the character of the cough and cry and of the stools, urine and vomited matter. The rate and rhythm of the respiration is to be determined; the occurrence of mouth-breathing noted, as well as the existence of thoracic rattling which can be heard at a distance; and the presence of dyspnea and its degree and method of manifestation is to be studied. The character of respiration and its regularity may be observed well by watching the movement of the thorax and abdomen while the child is asleep. Nothing can be determined if the child is at all excited.

Special inspection of the *ears* is often required, and should be practised in all doubtful cases where there is unexplained fever, pain, restlessness or stupor. Expertness in the use of the otoscope is a necessary accomplishment for the pediatrist. Examination of the *eyes* sometimes gives valuable information. The presence of conjunctivitis is to be noted and of swelling and discoloration of the lids. The use of the ophthalmoscope is possible even in very young children, and important conditions of the eye-ground may often be discovered. Inspection of the *nose* with the mirror and speculum can be carried out in docile children. That of the anterior nares is important and unattended by difficulty. It may reveal the presence of nasal diphtheria, so often unsuspected.

Inspection of the *mouth* and *throat* should always be made as a matter of routine at the first visit, and the possibility of subsequent development of disease here always kept in mind. The condition of the tongue, gums, and teeth can often be seen if the patient is crying. For a more thorough inspection of the mouth in infancy, the fingers, previously well washed, may be pushed gently in between the gums at one or both sides of the mouth. This, or the gently pressing of the chin downward will be enough to open the mouth, and to allow a visual examination of it to be made easily. To view the pharynx satisfactorily demands quickness and a certain degree of dexterity. A tongue-depressor is required, and for this nothing answers better in family practice than a teaspoon with a smooth handle free from sharp irregularities. Serviceable for office use are the wooden depressors (Fig. 26*b*) made for this purpose, and in occasional cases, where considerable force is required, the excellent rigid depressor devised by H. D. Chapin (Fig. 26*a*).

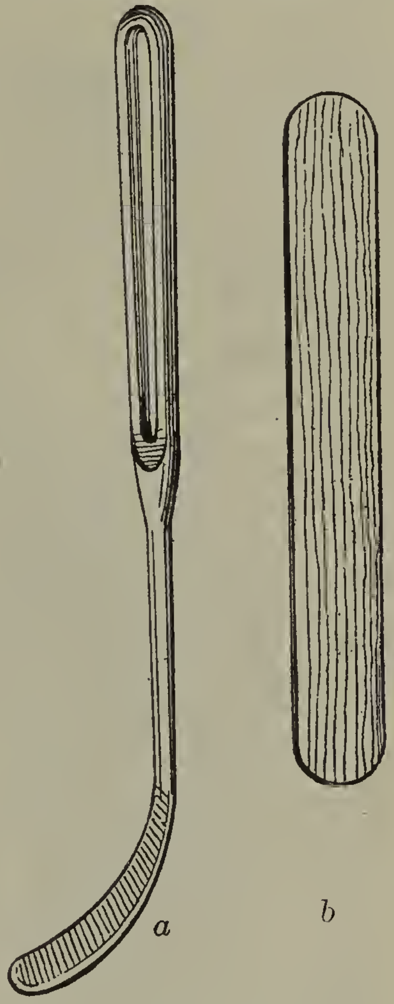

Fig. 26.—Tongue Depressors.
(*a*) Chapin's; (*b*) Wooden.

The infant or young child should be seated in the mother's lap, facing a window and with the back of its head against her right arm or shoulder. Its hands should be held, or better still, a blanket or shawl be wrapped about the arms and body close under the chin. All preparations are to be made before the physician approaches the child. In this way much less fright is occasioned. He now stands slightly to the mother's right side and in front, places one hand upon the head in order that he may steady it and turn it in any direction desired, and with the other hand introduces the depressor, utilizing a moment when the child opens its mouth to cry or to make some remonstrance. With older children who have learned to keep the mouth tightly shut, the depressor can readily be worked in from the side, or the nostrils compressed for a moment to make the mouth open. Every movement must be gentle and without hurry, yet quickly carried out. As soon as the depressor is in the mouth, it is pushed gently backward. When it reaches the base of the tongue the child gags and necessarily opens its mouth widely, the soft palate rising, and the tongue sinking. At this moment, a rapid yet complete inspection of the fauces can be made. All this procedure may and should be accomplished in a few seconds. The only exception to this is in the case of young infants. Here the reflex gagging is less well-developed, and forcible depression of the tongue is required. It is in such cases that the Chapin depressor is serviceable.

In the case of older, vigorous, and very obstreperous children who struggle violently, a somewhat different plan of holding is required. The mother seats herself facing the light, with the child in her lap and its back against her body. Wrapping her knees about its feet and legs she grasps its left hand in her right and its right in her left and draws them toward her. This movement crosses the child's arms over its chest and draws its head against the mother's breast, thus rendering the patient powerless. When the physician wishes to have both of his hands free for the treatment of the throat, a modification of this method can be adopted, as shown in the illustration (Fig. 27).

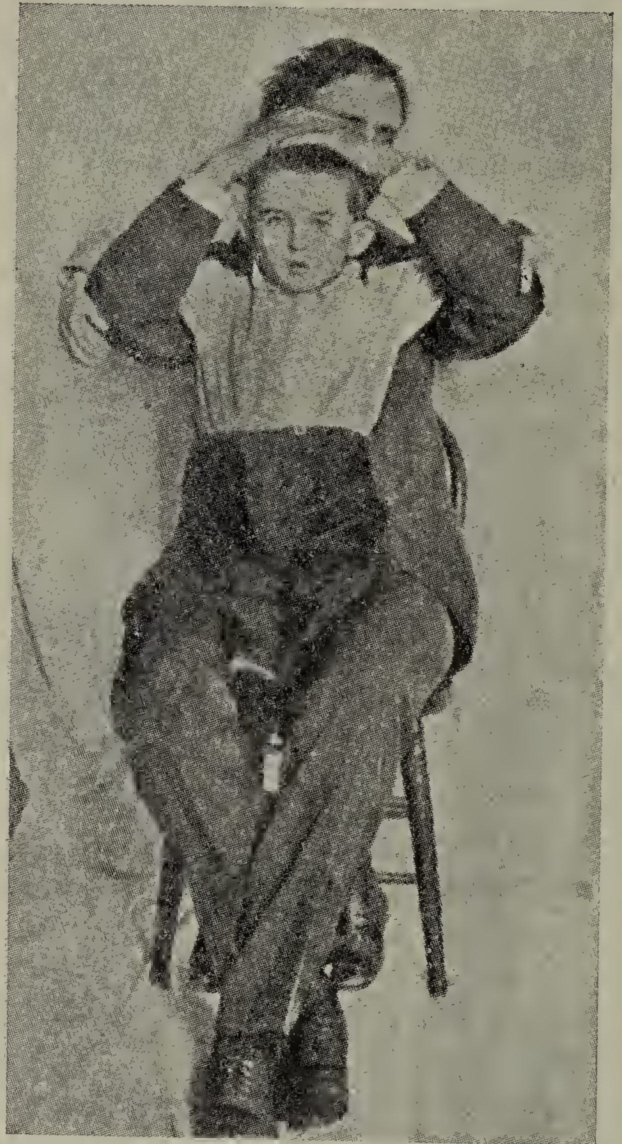

Fig. 27.—Method of Confining the Arms and Legs to Permit of Examination of the Throat.

(*Freeman, Philadelphia Polyclinic,* 1895, *March* 23.)

Occasionally timid little children, who have learned to fear a depressor, will tolerate the physician's little finger pressed for a moment against the base of the tongue. The finger must, of course, be well cleansed immediately before its insertion. The possibility of having the finger bitten must not be forgotten. Some children will themselves depress the tongue with their own finger. Under certain circumstances, as, for instance, in cases of extreme weakness, it is often best to omit the examination of the throat unless there are symptoms pointing to some trouble there. This is true especially of laryngospastic children, in whom a dangerous spasm of the larynx may readily be produced by the examination.

The inspection of the *larynx* in infancy and childhood is often important. It is, however, generally difficult to accomplish satisfactorily in young subjects, even by skilled observers.

Palpation.—In palpating a young child it is important to avoid causing alarm. The hand of the physician should be warm and every touch light. When the child is asleep it is well to attempt to take the pulse or to palpate the abdomen. To examine the pulse of the sleeping child the warm fingers may be applied very lightly over the radial artery, and the physician's hand should readily follow, without restraining, any movements of the hand of the patient. If the child is awake its attention may often be diverted by toys. If it becomes alarmed observations of the pulse are rendered worthless. In infants under 6 months, it is often impossible to feel the radial pulse. In such cases it may be counted at the fontanelle. With similar precautions the rate of respiration may be determined by placing the hand upon the abdomen.

To palpate the abdomen satisfactorily it is necessary that its walls be relaxed. During sleep, or when the child is being diverted in some way, is the time to be preferred. The flat of the warm hand should be laid gently upon the skin under the clothing or blanket. Light palpation is employed first, in order not to awaken resistance of the abdominal walls. Local tenderness may be discovered and the degree of any unusual resistance may be gauged. The edge of the liver or of an enlarged spleen may be detected, and any tumors superficially situated can often be readily felt. Now deeper palpation may be attempted. If this causes resistance and the child cries the hand may sink a little deeper every time a breath is taken, and, by maintaining the advantage thus gained, even conditions deep-seated within the abdomen may sometimes be detected. The value of bimanual palpation is not to be overlooked. Sometimes rectal examination may aid greatly a simultaneous palpation of the abdomen with the other hand. It is often better to do this under anesthesia. When a child continues to scream violently and continuously, abdominal palpation is generally unsatisfactory. At times by keeping the hand in position, and diverting the child, an opportunity to examine may after all be obtained.

In palpating the chest, one seeks for the position of the apex-beat, differences in the expansion of portions of the thorax, the presence of the rachitic rosary or other bony alterations, bulging or retraction of any region, the presence of precordial thrill, and the fremitus communicated by bronchial ronchi and less frequently by the voice. Sometimes bimanual palpation in an intercostal space may detect fluctuation in cases of pleural effusion. Palpation is a most important means of diagnosis in cases of retro-pharyngeal abscess as also of adenoid vegetations in little children. In either condition the finger introduced through the mouth into the nasopharynx will render the diagnosis easy.

Percussion.—This must always be lightly performed. Not only is heavy percussion a cause of discomfort, but it defeats its own object. The chest-walls of the child are so thin that a heavy blow causes a general reverberation and conceals the dullness of a small spot which would otherwise have become apparent. The finger used as a pleximeter should be firmly applied, but the percussion should be done gently and with but one finger, and with a movement of the finger only or the slightest action of the wrist. The feeling of the resistance is as important as the hearing of dullness present. Crying causes no real difficulty, as the tapping can be done at the moments the child stops to draw breath. When there is no crying, percussion should always be made during both inspiration and expiration. In percussing the back or the axillæ, the child may sit in the lap or in the bed; or, in the case of infants, may be held upright in the nurse's arms with its head looking over her shoulder. In either case, however, it is very important to see that its position is straight; not with one arm greatly elevated as compared with the other, or with the spine at all twisted. A faulty position will give untrustworthy results. Indeed, a chest much deformed from spinal disease often cannot be percussed with satisfactory results. A child too ill to be kept in a sitting position may be placed flat on its abdomen. This position may sometimes conveniently be made use of in less feeble patients, since it is one to which healthy infants are accustomed in being dressed. For the examination of the front of the chest the child may conveniently be laid on its back in bed. Percussion should not be too long continued, in order that too great annoyance or fatigue may not result.

Percussion is not as satisfactory in children as in adults, owing to the smaller and more resilient chest and to the variety of sounds produced. It is extremely easy entirely to overlook small areas of consolidation. Even considerable pleural effusion may give no very dull sound. On the other hand, it is not uncommon to discover areas of apparent dullness with which there are neither symptoms nor other physical signs to correspond, and which have no pathological significance, as far as can be discovered. This is especially true for the apices, as has been pointed out clearly by Hamill[1] and by Mielke.[2] Among other peculiarities of percussion in healthy children is the loud, full note obtained over the whole chest, which would be called tympanitic in adults. A cracked-pot sound is often easily obtainable even in perfectly healthy children who are crying, or who are even entirely quiet. (See also p. 225.)

These difficulties and causes of uncertainty in no way militate against the importance of a careful percussion of every chest in which disease is suspected, since this examination, combined with experience in disease in children, will often be of the greatest possible service.

Percussion of the abdomen reveals the presence of unusual gaseous distention, free fluid, enlargements of organs, morbid growths, or inflammatory processes (p. 226).

Auscultation.—Auscultation of the lungs in children, and especially in infants, may well be done with the ear applied next to the chest or with only a thin unstarched garment between. This method is suitable when we are seeking for the presence of the scattered coarse râles of a bronchitis, or for large areas of bronchial or of feeble breathing; but for the more exact localization of sounds, or the discovery of lesions in doubtful cases, a stethoscope is indispensable. Yet both the mediate and the immediate method should always be followed, for it sometimes happens that sounds undiscovered by the one will be revealed by the other. Either the disc double stethoscope or that with the ordinary bell-piece may be employed, according to the preference of the examiner. Children often dislike greatly not only the appearance of the instrument, but also the sensation of the hard, cold bell-piece pressed against the skin. The first difficulty can be overcome by allowing the child to handle or play with the stethoscope before it is used; the second by warming the part which is to touch the chest, or by having it covered with a soft rubber ring or surrounded by a rubber cup. The bell must be small in order to make it adapt itself well to the surface of a child at all thin. The use of the soft rubber referred to is an aid here. The whole chest-piece ought to be short in order to make shifting of the stethoscope easier. The posterior part of the chest should always be auscultated first, not only because it is the more fruitful in auscultatory signs, but because it may be examined more quietly before the child discovers what is going on. Yet all parts must be carefully studied, never forgetting the axillæ. The child may be seated or held in the arms for the examination of the back. Infants may sometimes lie on the abdomen upon the mother's knees. When the child is too ill to sit up, it may be made to lie on the abdomen, or first on one side and then on the other. This last method is, however, less satisfactory, as it prevents immediate comparison of regions on opposite sides of the chest. The lateral portions are best examined while the patient is sitting or held; the front while it is lying in bed. The child must never be so low that the position of the physician is made uncomfortable through stooping, as otherwise the congestion produced interferes with exact hearing and accurate results cannot be obtained.

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[1] Arch. of Pediat., 1907, XXIV, 92.

[2] Berl. klin. Wochenschr., 1914, LI, 1218.

Crying does not interfere materially with auscultation of the lungs, if the ear is at all practised. Inspiration can be listened for when the child draws breath between the cries, unless there is noisy laryngeal inspiration. In fact, crying is often advantageous in making inspiration deeper, and it is frequently only during crying that vocal resonance can be properly studied in infancy. Increase of vocal resonance may sometimes be determined by observing the closeness of the râles to the ear in a certain locality. Auscultation of the heart-sounds is interfered with considerably when the child cries. They can then be heard only when the child inspires. If there is no crying, the rapidity of the cardiac action may be determined by the stethoscope over the apex, in cases where the pulsations at the wrist cannot be counted readily. The irregularity in respiration, especially present in infants, is to be borne in mind, as the very long pauses which occur might deceive the unwary into believing that respiration was inaudible.

Among some of the peculiarities of auscultation in childhood, the loudness and harshness of the respiratory murmur is to be mentioned, a condition which when present in adults is denominated "puerile" and is considered pathological. In infancy, however, respiration is superficial and feeble. The ease with which the heart-sounds can be heard at the back, and in fact all over the thorax, even when there is no consolidated pulmonary tissue intervening, and with which râles and even bronchial respiration produced in one lung can sometimes be heard in the other is to be noted, as is also the facile transmission through the lungs, especially to the apices, of sounds produced in the upper respiratory tract. It is especially, too, in children that respiration approaching a bronchial character is heard under the clavicles and in the interscapular spaces close to the spine, particularly on the right side, yet without pathological significance.

The results of auscultation in pneumonia in early life often present important differences from those obtained in adults. This is so true that the diagnosis of this disease must often rest upon the symptoms rather than the physical signs. The lateness of the development of physical signs is often characteristic, for the attack may sometimes nearly run its course before any signs whatever can be detected. This is perhaps especially true in bronchopneumonia, in which disease the smallness of scattered patches may produce no characteristic alteration of the respiratory murmur. (See p. 225.)

Temperature-taking.—The effort to determine the existence or non-existence of fever by applying the hand to the skin is entirely untrustworthy. High temperature, it is true, can often be detected if the hand is placed under the clothing upon covered parts of the body, but the method has repeatedly led to grave mistakes. The use of the clinical thermometer is the only means to be relied upon. For employment in children it is much better to have one which will record quickly. Yet the instrument should remain in place a little longer than the "one-minute" or "half-minute" would indicate as necessary. The only absolutely accurate method consists in leaving the instrument in position until the maximum temperature is reached, watching the mercury meanwhile to determine this. The dislike of most small children for temperature-taking is, however, often too great to permit of employing this plan. A little experimenting with a quick self-registering thermometer will show how long it generally requires to reach the maximum with approximate correctness.

In well-trained children of 4 years the thermometer may be placed in the mouth, although there is even then some danger of having it bitten. In younger subjects the rectum is the only proper place. The use in the axilla requires very variable times and gives varying results, owing to the difficulty in getting close apposition of the opposing cutaneous surfaces. This is especially true of wasted children. Moreover, this locality demands that the arm be held firmly, and to this children strongly object. It must be remembered that the temperature in the mouth or rectum during fever is at least a degree higher than that in the axilla, even when the latter is accurately ascertained. The first two positions give the correct reading; the axillary temperature must have about a degree added to it. The employment of the groin or of the popliteal space is to be condemned. The results are seldom accurate.

The bulb of the thermometer should be oiled slightly and pushed into the rectum until well out of sight, the infant meantime lying in any position comfortable to it. The nurse should then keep the tip of her finger upon the end of the instrument. There is no necessity of restraining the infant's legs, and it may be allowed without danger to kick all it desires if the hand merely guards the thermometer. It is better that the rectum be empty of feces, but this is not an essential. Only when the thermometer in the rectum produces pain and straining, as in some cases of diarrheal disturbance, should the axilla be used for obtaining the record. The time for taking the temperature should not be after a hard attack of crying, as the height of it may be increased. For the same reason, crying during the observation should be prevented, if possible, by diverting the child.

Examination of the Urine.—The obtaining of the urine is often a matter of some little difficulty in infancy. In the case of male infants it may be procured by applying a condom over the penis, or a small bottle with a sufficiently wide neck which may be held in place by bandages or adhesive strips extending to the waist. For female infants the neck of the bottle may be passed through an oblong piece of adhesive plaster, firmly attached there in a way to prevent leaking, and the plaster then applied over the vulva and perineum. In some cases it may suffice to place a large wad of absorbent cotton inside the diaper immediately under the genitals. The child must then be examined frequently in order that the urine may be expressed from the cotton as soon as possible after it has been passed. The method is not very satisfactory, especially when microscopic examination is important, as in the diagnosis of nephritis or pyelitis. Still another device is to allow the child to lie for a time upon a rubber-cloth and without a diaper, or upon a small circular rubber air-cushion with a hole in the center and a small pus-basin placed under the hole. The surrounding portion of the bed is built up with pillows to the level of the cushion, and the infant, with diaper removed, allowed to lie upon the bed and cushion until the urine is passed into the basin. Children a year old, or sometimes less, may be put at frequent intervals on a chamber in the hope of procuring urine in this way. Urination is most likely to occur at the time of nursing or taking fluid. In the event of none of these methods succeeding, a small silk catheter, No. 9 or 10 French scale of No. 4 American scale, may be employed. Except in very young infants, and sometimes even in these, the instrument can be passed without difficulty. The greatest precautions must be taken against infection, and the method employed only when the obtaining of uncontaminated urine is necessary, as in cases of suspected pyelitis in female infants.

Blood and Blood-pressure.—The examination of the blood is accomplished as in adult life, testing the hemoglobin-percentage, the number of and changes in the erythrocytes and leucocytes, and in some cases the coagulability, fragility of the corpuscles, specific gravity, and any chemical alterations which may be present. The blood-pressure, too, is estimated as in adults, with such modifications of the apparatus as the small size of the patient often demands. The study of the blood-chemistry (p. 54) may well be left to the laboratory expert, but some of the procedures to be described are readily performed by the clinician. The *coagulation-time* (Sabrazes' method)[1] is determined by testing the 2nd or 3rd drop of blood obtained on pricking the finger or ear. A capillary glass tube of 3 or 4 inches (7.6 or 10.2 cm.) in length is filled with the blood, and at intervals of 30 seconds a small section is scratched with a file and broken between the fingers. Coagulation is complete when a long worm-like coagulum is obtained. The normal time for coagulation is 3 or 4 minutes. Another simple method is given by Rodda.[2] The heel of the infant is punctured, and the 2nd drop of blood allowed to flow into a clean watch-glass, having a diameter of 2.5 inches (6.37 cm.), in which has been placed a No. 6 lead shot. A second watch-glass is inverted over the first, and the watch-glasses are gently tilted every 30 seconds until the shot is firmly embedded in the clot. The average coagulation-time by this method is 7 minutes.

The *bleeding-time* is determined by the method of Duke.[3] A deep puncture is made in the lobe of the ear, and at intervals of 30 seconds the drops are taken up on a piece of filter-paper without allowing the paper to touch the skin. Normally the bleeding will stop in from 2 to 3 minutes.

In testing for *increase of fragility* (*i.e.*, decreased resistence) of the red blood-cells, the method of Ribierre[4] modified by Giffin and Sanford[5] is simple and satisfactory for clinical purposes. It consists in preparing accurately a stock solution of 0.5 gm. of chemically pure anhydrous sodium chloride in 100 c.c. distilled water. A series of 12 small test tubes (⅜ by 3 in.) are then marked with figures ranging from 25 down to 14. Using a capillary pipette drawn from soft glass tubing, there is dropped into each tube the number of drops of salt-solution indicated by the figure marked on the tube. With the same pipette distilled water is added drop by drop to each tube in a quantity to make a total of 25 drops. This gives varying percentages from 0.28 to 0.5. Thus the tube containing 20 drops of the solution and 5 drops of water has a percentage-strength of $^{20}/_{25}$ of 0.5 *i.e.* 0.4 per cent. That is to say, the percentage of salt-solution in each tube can be obtained by multiplying the number on the tube by 0.02. The patient is bled by puncturing the vein in the usual manner for obtaining blood for a Wassermann test. One drop of blood should be allowed to fall into each tube, and the tube then thoroughly shaken. The tubes are allowed to stand an hour at room-temperature and the results are then read. Normally hemolysis (laking) begins in 0.42 per cent. to 0.3 per cent. sodium chloride solution, and is complete in 0.36 to 0.32 per cent. Patients with increased fragility show laking with stronger solutions than do those with normal blood; those with increase of resistance hemolyze with weaker solutions than for normal blood.

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[1] Ref. Stitt, Pract. Bact., 1923, 307.

[2] Amer. Jour. Dis. Child., 1920, XIX, 269.

[3] Jour. Amer. Med. Assoc., 1910, LV, 1185.

[4] Thèse de Paris, 1903, 104.

[5] Jour. Lab. and Clin. Med., 1919, IV, 465.

Blood-typing for compatibility is done in different ways. The following is a method advocated by Lee[1] which may be used in emergency:

A small amount of blood is collected from the patient (1 c.c. from the ear or finger is sufficient), allowed to clot and the serum then obtained. One drop of this serum is placed on a slide and mixed with a drop of a suspension of blood of the donor in a 1.5 per cent. sodium citrate solution. To prepare this suspension a few drops of blood are added to approximately 10 times the volume of the citrate solution and shaken. It is very important that the blood be dropped directly into the citrate in order to prevent any partial coagulation. The test will appear in a few moments and is examined under the microscope, where, if the types are different, marked agglutination will be evident. In the event of a negative test it is a wise precaution to raise the cover glass, in order to make sure that the serum and cells are well mixed, and then to examine the preparation again. The only possible source of confusion is the appearance of rouleaux of the red corpuscles, indicating too thick a suspension. If the test is negative, transfusion may be given.

Cutaneous Sensibility and Muscle-power.—The study of both of these is often important. In the case of the first the pain-sense may be tested by means of a needle, but it is necessary that this shall not be seen by the child, or it will be impossible to determine whether the drawing away of the limb is the result of pain or of fear. The presence of a paralytic or pseudo-paralytic condition is often easily discovered by simple inspection. In other cases it is necessary to offer an infant a toy or other object and observe whether this can be grasped by either hand, the other being meanwhile restrained; or to put the arm or leg in a position which is an uncomfortable one and see whether the infant can move it back; or to test the power of moving after sticking with a needle.

The Reflexes.—The knee-jerks may be tested when the child is sitting, diverted, on its mother's lap. A good plan is to support the foot with one hand. Any jerk which occurs can then be felt as well as seen. Another method consists in grasping the thigh with one hand just above the knee. In this way the contraction of the quadriceps extensor muscle can be felt readily. The knee-jerk cannot always be elicited easily, sometimes owing to the flabbiness and weakness of the muscles, sometimes to the difficulty in obtaining a voluntary relaxation on the part of the child. The existence of ankle-clonus and of the Babinski and other reflexes of the lower extremities should be investigated and sometimes those of other parts of the body as well. The matter will be referred to again in the section upon Nervous Diseases (Vol. II, pp. 507, 611). The reflexes under normal conditions in early infancy have already been referred to (p. 61).

Puncture of Serous Cavities.—In very many diseases a diagnosis cannot be made without the puncture of the pleural, pericardial or peritoneal cavity, or of the spinal canal or cerebral ventricles. These procedures are easy to perform and may be done with safety if proper precautions are taken. A glass hypodermic syringe with a stout needle is to be employed, or one of the larger syringes made especially for the purpose. In most punctures in children a needle of about 18 gauge is of proper caliber, although in young infants a smaller needle may be necessary at times. A fairly large needle is preferable in order to avoid clogging of the lumen by fibrin and pus. When inserted it should have a well-fitting stylet, but this may be dispensed with if the needle is attached to a syringe. The apparatus should always be tested before use, to determine that the suction of the piston is good, no air-leaks present, and that the needle is permeable. Great precautions must be used to disinfect the skin, the operator's hands and the instruments, and all aseptic surgical technic followed. For the instruments boiling is to be preferred. For the skin scrubbing with soap and water followed by a 50 per cent. solution of alcohol and water may be employed, or the surface painted with tincture of iodine. If hair is present it should be removed by clipping and shaving. The patient should be held firmly by a nurse or assistant. General anaesthesia is seldom necessary, and even local anaesthesia is rarely required, the discomfort of the freezing being greater and longer continued than that of the puncture. The pain, in fact, is little if any greater than that of an ordinary hypodermic injection. The operation should be done rapidly but never hurriedly. After withdrawal of the needle, the point of puncture is covered with a small piece of adhesive plaster, or a film of cotton may be laid over it and then painted with flexible collodion. Any fluid obtained should be ejected into a sterilized test-tube and then stoppered with scorched cotton.

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[1] Brit. Med. Jour., 1917, II, 684.

Puncture of Pleural Cavity.—The needle should be about 1 mm. (0.04 inch) in diameter, the syringe and glass as described, and the antiseptic precautions mentioned strictly followed. The child should be firmly held by the nurse in a sitting position, with the arms drawn forward and the healthy side of the chest pressed against her in such a manner that any sudden movement will not break the needle. The site chosen for puncture is that where the physical signs are most suggestive, especially where the dullness is greatest, but avoiding the cardiac or hepatic region. Other things being equal, the posterior axillary line in the 7th interspace may be chosen. With the sterilized finger pressed firmly upon the upper border of the rib, the needle is introduced just above this as a guide, thus avoiding both the rib and the blood-vessels running beneath it. A depth of ½ to 1 inch (1.27 to 2.54 cm.) is sufficient. Too short a puncture fails to penetrate the chest-wall and any plastic exudate present; too deep a one may pass through the fluid and enter the lung. The syringe must always be held lightly during the whole process, in order to avoid danger of snapping the needle if the child moves, and the puncture done quickly but carefully. Traction on the piston may be followed by the appearance of fluid, but sufficient time must be allowed, since a purulent effusion often flows very slowly. Should nothing be found at the first puncture in cases where the presence of pus seems probable, a second or a third should be made immediately in another spot, and this, if unsuccessful, repeated in a few days.

Puncture of Pericardium.—The needle used should preferably be smaller than that commonly employed for the pleural cavity or for lumbar puncture, although if thick pus is encountered one with as great a gauge as 18 may be necessary. When inserted the needle is attached to an aspirating syringe. There are several points recommended for aspiration, which will avoid opening the pleura or wounding the internal mammary artery. The usual selection is the 5th left intercostal space 1 inch (2.54 cm.) from the edge of the sternum. If the 4th space is used the needle should be inserted at a distance of about 2 inches (5.1 cm.) from the sternal border. In small infants the distance from the sternum should be less. The 5th right interspace close to the sternum may also be chosen. If the pericardium is approached through the epigastrium, the needle is entered opposite the 7th left costal cartilage or in the midline just below the ensiform cartilage, and, with the patient in the recumbent position, is then pointed upward and to the left and pushed through the diaphragm. The outermost limit of cardiac dullness to the left is preferred by some authors as the point of selection for aspiration. Puncture should never be made over a place where there is a friction-rub.

Lumbar Puncture.—For the obtaining of spinal fluid a needle of about 18 gauge is best, although a one of smaller caliber may be necessary in very young infants, because of the small space between the vertebrae. The child either sits with the body bent forward and firmly held in this position or, we think preferably, lies on its right side (Fig. 28) turned partially upon the abdomen with a small pillow beneath this. The knees and shoulders are then drawn well forward and firmly held by an assistant. In this position the spines and laminæ of the vertebræ become more widely separated from each other. An imaginary transverse line at the level of the crests of the ilia crosses the spinal column at the 4th lumbar vertebra. The needle, unattached to the syringe, is now introduced either in the 3d or the 4th interspace. It should be inserted in the median line and in a direction almost perpendicular. It is only in older children that a slightly upward direction is needed. At a depth varying from 1 to 2 inches (2.5 to 5 cm.) it enters the subarachnoid space below the termination of the spinal cord. As a rule the fluid begins to drop at once, or sometimes, if the pressure is high, even to spurt. If it is purulent in nature, it may be necessary to attach the syringe, and to use a slight suction to start the flow; but after this the syringe should be removed and the exudate allowed to escape unaided. Sometimes raising the

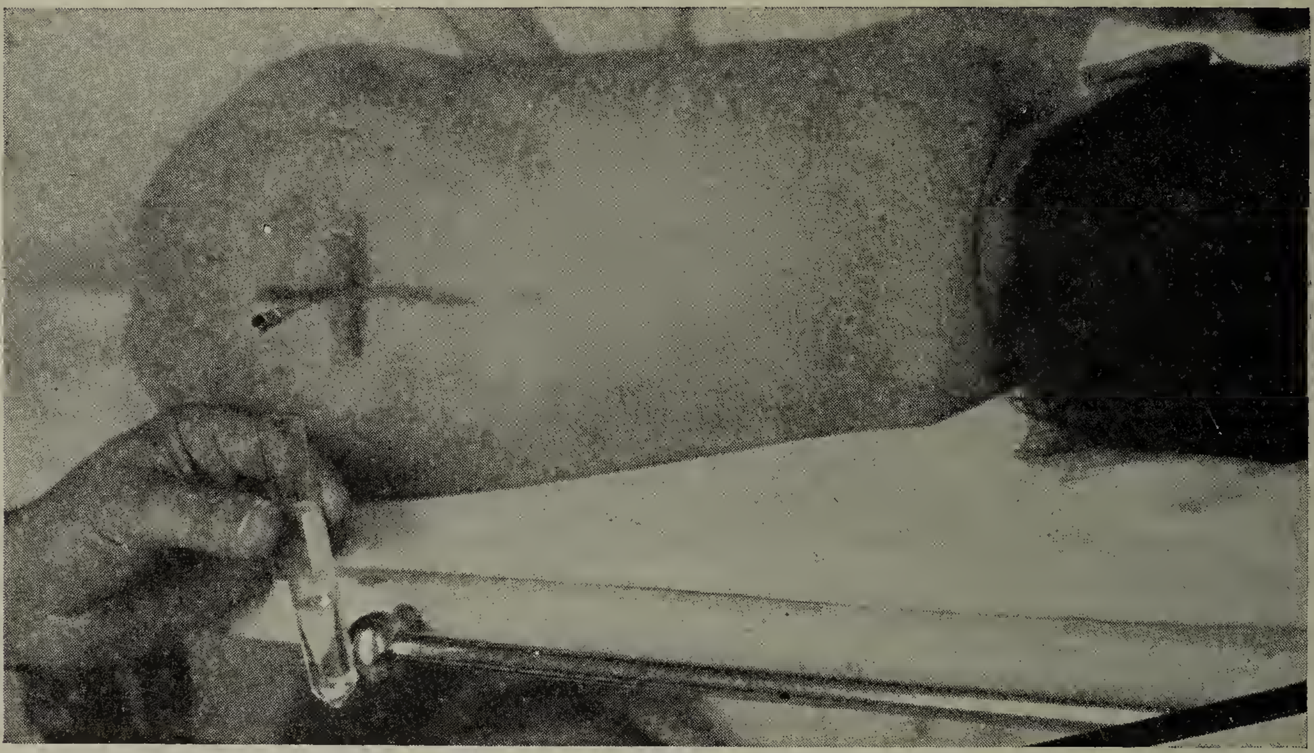

Fig. 28.—Lumbar Puncture.

The lines indicate the vertebral column and the transverse line between the crests of the ilia. Puncture is done at the crossing of these.

head and shoulders slightly will suffice. The fluid should be received in a sterilized test-tube to allow of making cultures. A manometer devised for the purpose may be serviceably employed to measure the degree of intraspinal pressure present. At times a dry tap results, even in cases of undoubted meningitis. This may be due to the failure of the needle to enter the canal, to its being plugged with fibrin, to the thick character of the fluid, or to the fact that the inflammatory process has shut off the brain and the upper portion of the cord from the parts below. If the needle is introduced too far it will impinge on the opposite wall of the spinal canal. This accident does no harm except the wounding of small blood-vessels, as a result of which the fluid which appears is at first bloody, and may continue to be so if there has been much hemorrhage.

The child must be prevented from sudden movement and the needle held loosely to avoid the danger of breaking it in the spinal canal;—an accident which we have seen happen through neglect of these precautions, and when too slender a needle had been employed. As a rule no anæsthetic or local anæsthesia by freezing is necessary, but if the child struggles violently against being held, and is weak enough to make this an objection, a few whiffs of ether may be needed, although this is usually as objectionable to the patient as is the slight pain of the puncture. Lumbar puncture is generally entirely without danger. The pulse should, however, be watched carefully, and no effort made to withdraw all the fluid possible, lest the degree of intracranial pressure be too violently disturbed.

Ventricular Puncture.—The patient is placed on the back and the head is firmly held. When the fontanelle is open trephining is not necessary. The region of the fontanelle is shaved, and the skin rendered antiseptic in the way described. A spinal puncture-needle of No. 18 gauge or smaller is inserted, with the stylet in place, through the right lateral angle of the fontanelle, and pointed parallel to the axis of the body or a little inward. This position is selected in order to avoid any possible injury to the speech-centers. The depth to which the needle must go to obtain fluid varies with the degree of distention of the ventricles. In an infant the undistended ventricle lies 2 to 3 cm. (0.78 to 1.18 in.) from the surface of the skull. It is a good plan to insert the needle 1 centimeter (0.39 in.) at a time, withdrawing the stylet at each stop to see whether fluid can be obtained. Serum may be injected in smaller amounts than that of cerebrospinal fluid removed, or dye may be introduced for the purpose of diagnosing hydrocephalus (see Vol. II, p. 652).

Cisterna-puncture.—The technique of this procedure is described by Ayer.[1] Some modification is necessary for infants and children (Mitchell and Reilly).[2] The patient is placed on the right side, with the neck slightly flexed toward the chest and the head and back kept in alignment, so that there is no scoliosis or torsion. A needle of 18 gauge or less with a short bevel is employed. The thumb of the left hand is placed on the first bony point encountered below the occipital protuberance (the spine of the axis) and the needle, held in the right hand, is inserted in the midline just above the thumb. The needle is pushed forward and upward in such a way that the point is directed toward the glabella. The distance from skin to cisterna magna is about 2.25 to 3.5 cm. (0.88 to 1.38 in.) depending upon the age and size of the child. When the cistern is entered there is a decidedly greater "give," or sensation of entering a cavity, than is experienced in lumbar puncture. Ayer states that it is

[1] Arch. Neurol. and Psychiat., 1920, IV, 526.
[2] Amer. Jour. Med. Sci., 1922, CLXIV, 66.

a good practice to aim the needle a little higher than the external auditory meatus, and if it strikes the occiput to depress the point just enough to pass the dura at its uppermost attachment to the foramen magnum. He[1] reports on collected statistics involving 1985 punctures without serious accident of any sort, and Ebaugh[2] has had the same experience in approximately 1550 punctures in a total of 190 patients. We believe the procedure should be more widely followed.

Radioscopy.—Of recent years the use of the *x*-ray for purposes of diagnosis has been constantly becoming more important. The outlines of the heart and its actual size, the existence of areas of pneumonia or of tuberculosis, the presence of enlarged tracheobronchial lymph-nodes, the outlines of the thymus gland, the presence of pleural effusion and collapse of the lung, the size and position of the liver, alteration in the position of the gastro-intestinal tract and other physiological and pathological conditions may be discovered in this way. Advantage may be taken of the *x*-ray in studying bony deformities in rickets, and also in watching the improvement and cure of this disease. Scurvy will show characteristic subperiosteal hemorrhages and other changes. Intracranial lesions may be demonstrated;—pituitary tumors or enlargement shown by increase in size of the sella turcica; hydrocephalus after air has been injected into the subarachnoid space. A very serviceable purpose of the fluoroscope is the observation of the movements of the heart, lungs and stomach, and, with the aid of barium or bismuth, the rapidity of the emptying of the last-mentioned organ. In the diagnosis of non-opaque foreign bodies in the lungs the fluoroscope is invaluable. In the esophagus, atresia and foreign bodies and their location may be demonstrated by observing the progress down the food-passages of barium in suspension or in capsule. Lipiodol, a vegetable oil containing 0.54 gm. of iodine to the c.c., (32.4 gr. per fl. dr.) introduced into radiology by Sicard and Forestier,[3] may be injected into the bronchi through a tracheotomy tube. The *x*-ray picture taken after this clearly outlines the bronchial tree, cavities, and the like. The procedure is applicable in childhood (Armand-Delille and Gelston).[4] Iglauer[5] has developed a method of injection of lipiodol through an intubation-tube, thus avoiding an incision in the neck. The drug has also been used by intraspinous and cisternal injection to aid in the localization of tumors and blocks in the spinal canal. Iodipin, containing 40 per cent. of iodin in a vegetable oil, may be employed in the same manner.

Great care is needed in the interpretation of *x*-ray pictures, especially those of the chest in children. An extensive knowledge of the findings in the normal chest is necessary, otherwise too much stress will be placed upon shadows which have little significance. An excellent paper upon this phase of the subject is that of Pancoast, Dunham and Baetjer.[6] The pediatrist should familiarize himself with interpretation by examination of the pictures and consultation with the roentgenologist, and he should work in conjunction with the latter and allow him the benefit of the clinical history and diagnosis. Many useless *x*-ray pictures are taken because of lack of care in clinical diagnosis. The *x*-ray is often simply an accessory to diagnosis. In other cases it is the only possible way of making or confirming it.

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[1] Jour. Amer. Med. Assoc., 1923, LXXXI, 358.
[2] Jour. Amer. Med. Assoc., 1925, LXXXV, 184.
[3] Bull. et mém. soc. méd. d. hôp., 1922, XLVI, 463.
[4] Amer. Jour. Dis. Child., 1924 XXVIII, 527.
[5] Jour. Amer. Med. Assoc., 1926, LXXXVI, 1879.
[6] Amer. Rev. Tuberc., 1922–23, VI, 338.

Glucose-tolerance.—This is best tested in the manner described by Janney and Isaacson.[1] A solution is prepared of 1.75 grams (27 gr.) of glucose in 2.5 c.c. (41 m.) of water, and this amount is given for each kilogram (2.2 lbs.) of body-weight. The patient fasts from 7 P.M. until the test is completed. An estimation of the blood-sugar is determined early in the morning and the solution of glucose is then administered. A second determination is made in 1 hour, and a third in 2 hours more. The normal maximum concentration of blood-sugar should be reached in 1 hour and should not be over 0.15 per cent., and the original figure should be regained by the third examination. If the tolerance is normal the difference between the first and the third tests should not be more than 0.01 per cent. Infants have a somewhat greater tolerance than have older children and adults.

Electrical Examination.—This is of great value in many instances of nervous disease for the determination of the extent and nature of paralyses of different sorts, and the reaction characteristic of a spasmophilic state. To obtain the reactions satisfactorily it is sometimes necessary to give an anæsthetic. The subject is referred to later (Vol. II, p. 507).

CHAPTER X

SYMPTOMATOLOGY AND DIAGNOSIS

The characteristics of appearance and development present in health have already been considered in discussing Anatomy and Physiology. Some of the symptoms pertaining to diseased states in early life and the significance of these may now be reviewed briefly. A more complete description of many of them will be found under the headings of the individual diseases.

SIGNIFICANCE OF SYMPTOMS

Position and Movements.—A child with a commencing illness which is attended by pain or fever usually no longer exhibits the quiet, motionless sleep of health, but tosses from side to side. When awake, too, the natural restlessness is in like manner increased, the patient wishing to be taken from bed, put back, rocked, or carried about in many ways, indicating the excited state of its nervous system. On the other hand at the beginning of an infectious disease and sometimes in digestive disturbances it may happen that the patient will lie unusually still, sleeping constantly, the evidence apparently of the toxic state existing. All movements are slow in debilitated states, and a child afflicted by very profound exhaustion may lie for hours without motion, with its face directed upward instead of to one side, as it commonly is in health. The same position and lack of motion is seen in coma from any cause.

Restlessness in infancy especially during sleep, with an unusual tendency to kick the covers away, is an early symptom of rickets. Restless sleep may also depend upon hunger, pain, nervousness, great fatigue, noises or light in the room, or unusual excitement before going to bed. Later, restlessness at night may accompany certain forms of chronic gastroenteric indigestion. An intense degree of restlessness, called "jactation," may occur in some respiratory diseases, especially those of the larynx; in a state of acidosis with air-hunger; in severe chorea; great cerebral anemia, and sometimes in severe attacks of infectious disease including sepsis.

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[1] Jour. Amer. Med. Assoc., 1918, LXX, 1131.

Orthopnea may attend diseases of the respiratory apparatus or of the heart, the child resting comfortably only when propped up in bed or when held upright in the nurse's arms with its head against her shoulder. Sleeping with the head thrown back and the mouth open is often the result of obstruction to respiration by adenoid growths. A rocking of the head from side to side on the pillow may be observed in infants with rickets, sometimes in meningitis or headache, and is frequently a natural expression of an intensely nervous state. Keeping the head bent backward is seen in basilar meningitis of different forms, and to a less degree in headache. A fixity of the head and neck also attends cervical caries or torticollis, the head in the latter condition being generally turned to one side. Boring the head into the pillow may indicate meningeal disturbance. Lack of power to hold it erect denotes great general weakness, or may be an evidence of congenital or acquired torticollis, of idiocy, or of some other nervous disorder, such as, in later childhood, advancing Friedreich's ataxia. Inability to walk may be the result of idiocy, of actual paralysis, or of a pseudo-paralysis dependent upon syphilis or, oftener, rachitis. Failure to move one or more limbs properly may denote paralysis, but may equally well be due to the pseudo-paralysis of rickets or syphilis, or depend upon congenital dislocation of the hip-joint, or indicate that motion is avoided because painful, as in infantile scurvy, or be due to the existence of a spastic rigidity or to an ataxic condition. Lying upon one side sometimes occurs in pleural effusion of that side. Lying with the head retracted, the back hollowed, the knees and elbows flexed and the arms crossed over the chest—the so-called "gun-hammer position" (*en chien de fusil*)—is frequently a symptom of meningitis. Lying upon the abdomen may indicate abdominal pain, but with many children it is only a habit.

Often an infant will repeatedly put the hands fretfully to the head when there is headache, to the mouth when pain exists there, or to the ear when earache is present. In the latter condition the side of the head is frequently held pressed against the pillow or the mother's breast. Pulling at the ear accompanied by fretfulness may, however, be only a nervous habit in a child suffering from rachitis or other debilitating disease. Rubbing or picking at the nose indicates coryza or gastro-intestinal disturbance or is a neurotic habit merely. It is not a special indication of the presence of intestinal parasites. Pulling at the throat sometimes occurs when there is much dyspnea. The violent alternate flexion and extension of the limbs upon the trunk, and of the trunk itself, accompanied by clinching of the hands and the characteristic cry, denotes the pain of colic. The keeping of the thumbs drawn into the palms and the toes flexed or rigidly extended often indicates impending convulsions, and it is also present in tetany. At the same time it is to be borne in mind that every very young infant has a tendency to keep the thumbs thus inverted much of the time. Rigidity of the limbs may occur, as in meningitis, cerebral paralysis, and spinal caries. More or less extensive tonic spasm may be seen in tetanus. Irregular, jerking, incoördinate movements in older children occur in chorea. Clonic, to-and-fro movements with unconsciousness are characteristic of convulsions, while true ataxic movements are seen in Friedreich's ataxia and allied conditions. Tremor is rarely observed in children, except as a result of chorea, some organic nervous disease especially encephalitis, or of such weakness as develops after fever. The shaking of a true rigor is uncommon in early childhood and infancy, being replaced by coldness, pallor, drowsiness, unusual quiet, or a convulsion.

Surface of the Body.—A yellowish tint of the cutaneous surface and of the sclera is seen in icterus. In well-nourished infants there is, however, normally a sufficient deposit of fat under the conjunctiva to give a slightly yellowish tint. Flushing of the face is common in fever, and is also often observed in chronic gastro-intestinal indigestion of older children, and from the action of belladonna. Slight eczema or chapping of the cheeks simulates flushing to some extent. A flush which comes and goes slowly, on the face or on a part of it, or on the trunk when exposed to the irritation of the air, is a characteristic symptom frequently present in meningitis. The broad red line which develops after drawing the finger over the abdomen in cases of meningitis (*tache cérébrale*) is of the same nature. This symptom is, however, not pathognomonic but only suggestive of this disease. Very marked blueness of the whole face, the fingers and toes, and the mucous membrane of the mouth is present in congenital cardiac affections, less often in the intense dyspnea arising in laryngeal stenosis, and sometimes in severe pneumonia. A slightly bluish tint of the lips and cheeks is of common occurrence in cases of post-natal affections of the heart. The red flush present on the cheeks in many cases of pneumonia quite commonly has a bluish tint to it. Moderate distention of the veins running over the scalp and at the root of the nose occurs in debilitated children, but especially in rickets. The veins of the scalp are also much distended in hydrocephalus. Great distention of the veins of the face and neck attends any decided degree of dyspnea. Dilatation of the veins over the abdomen and lower part of the thorax is witnessed in cases of malignant abdominal growth or sometimes of tuberculous peritonitis. A faintly purplish tint of and under the eyelids and above the mouth is often seen in infants with debility or even with any slight disturbance of health. In very many children, however, a purplish tint or, still oftener, a shadow under the eyes is an individual peculiarity, the health in general being unaffected.

Marked pallor of the skin accompanies nausea, anemia of any sort, rickets, chronic diarrhea, chronic suppurative processes, nephritis, some severe cases of pneumonia, and frequently heart disease. Very little can be estimated about anaemia, however, by the appearance of the skin. The color of the mucous membranes furnishes a much better index. Combined with coldness pallor may replace in the infant the chill of adult life. An earthy color is frequently observed in severe chronic diarrhea and a brownish-yellow color of the skin, especially of the projecting portions of the face, in congenital syphilis. A marbled appearance of the surface of the body is often present in debilitated infants with feeble circulation. This becomes particularly noticeable if the skin is unduly cold. The various eruptions of the exanthematous fevers are oftenest witnessed in children, since these diseases are far commonest at this age. Infants show an especial tendency to inflammations of the skin, such as miliaria, eczema and forms of erythema. Profuse sweating, especially of the head, is an early symptom of rickets. When general over the body it is very frequently the result of an undue amount of clothing. Coldness of the extremities is present in weakly babies with poor circulation and in infants suffering from colic. Clubbing of the fingers and toes occurs in congenital cardiac disease and in chronic affections of the lungs and pleura. A shining red appearance of the palms and soles in young infants is a symptom of inherited syphilis, while peeling of the skin in older children, seen especially about the fingers, suggests convalescence from scarlet fever.

Edema of the skin, especially of the face and feet, may indicate nephritis or valvular disease of the heart, or may be an evidence of extreme malnutrition and feeble circulation. Angioneurotic edema sometimes occurs in children. A localized asymmetrical atrophy of the muscles points to poliomyelitis or neuritis, while an undue development, especially of the calves, may indicate pseudo-hypertrophic muscular dystrophy. Local swellings of the joints are observed in all forms of arthritis, and the swelling about the joints and of the shafts of the long bones in scurvy is not to be forgotten. Curvature of the spine of various forms may be dependent upon disease of the vertebræ, old pleurisy, unequal length of the limbs, rickets, or faulty positions in being carried or in sitting. General wasting in infancy with the skin in loose wrinkles and folds is oftenest the result of insufficient nourishment, of persistent diarrhea or vomiting or of chronic intestinal indigestion. In some cases, however, it is a sign of tuberculosis or of congenital syphilis.

Face and Expression.—The mouth is open during sleep in cases of adenoid or tonsillar hypertrophy, or when the nose is obstructed by secretion. Chewing movements occur when there is indigestion or inflammation of the mouth. A general puffiness of the whole face, with redness of the eyes, is often present during pertussis or measles. Puffiness is also witnessed in the edema of advanced marantic conditions, and especially about the eyes in nephritis. A pale circle about the mouth accompanies nausea.

Pain is shown during sleep by contortions of the face of various sorts. Thus there is sometimes an expression of pain with contraction of the brows in headache, while the smiling of very young infants during sleep often signifies abdominal pain. Discharge from the nose occurs in coryza, and is often, also, one of the first symptoms of nasal diphtheria, and in these cases the discharge is likely to be bloody. The "snuffles" of new-born infants suggest congenital syphilis. In older children persistently reddened eyelids, combined with a swollen upper lip, wide nostrils, thick nasal discharge, muddy complexion, and enlarged cervical lymphatic glands, may indicate the existence of the lymphatic-exudative diathesis with tuberculosis, a combination to which the title "scrofulosis" was formerly applied. Wide-open nostrils moving with every inspiration and accompanied often by an anxious expression of face are observed in dyspnea, most commonly from pneumonia or from stenosing affections of the larynx. Occasional movement of the nares is, however, of frequent occurrence in healthy infants. A flattened, somewhat sunken bridge of the nose is characteristic of congenital syphilis, but by itself is not sufficient to warrant this diagnosis. In atrophic conditions in infancy, when the fat has largely disappeared or there has been great loss of fluid through diarrheal discharge, the face becomes lined in a marvellous manner, especially when the child cries, suggesting the features of a very old man. The most prominent of these lines is that called the "nasal," extending from the alæ of the nose and running in a half circle around the corners of the mouth. Other special lines have been described but have little importance.

In severe acute disease, atrophic states, or when there is pain or indigestion, the eyes may be only partially closed during sleep. Twitching of the lids and crossing or rolling upward or outward of the eyes indicates impending convulsions. The eyes in hydrocephalus are depressed downward, with the lower part of the iris covered by the lower lid and the sclera above the iris visible. After a severe acute attack of diarrhea or vomiting the tissues about the eyes shrink, leaving them peculiarly large and staring (Fig. 29). The pupils are dilated or unequal, or sometimes contracted in meningitis or other intracranial disorder. Nystagmus may often be seen under these conditions, but may be a purely functional disturbance combined with spasmus nutans or gyrospasm. Strabismus, too, is frequently an attendant of intracranial disease, but is equally well a congenital defect. Keeping the eyes shut or turned from the light or buried in the pillow indicates photophobia from conjunctivitis, keratitis, or headache dependent oftenest on meningitis. A film-like appearance of the cornea develops in children who are moribund. Ulcers on the cornea often occur in syphilitic or tuberculous children. Tubercle of the choroid, choke-discs, and other important interocular conditions may be discovered by ophthalmoscopic examination.

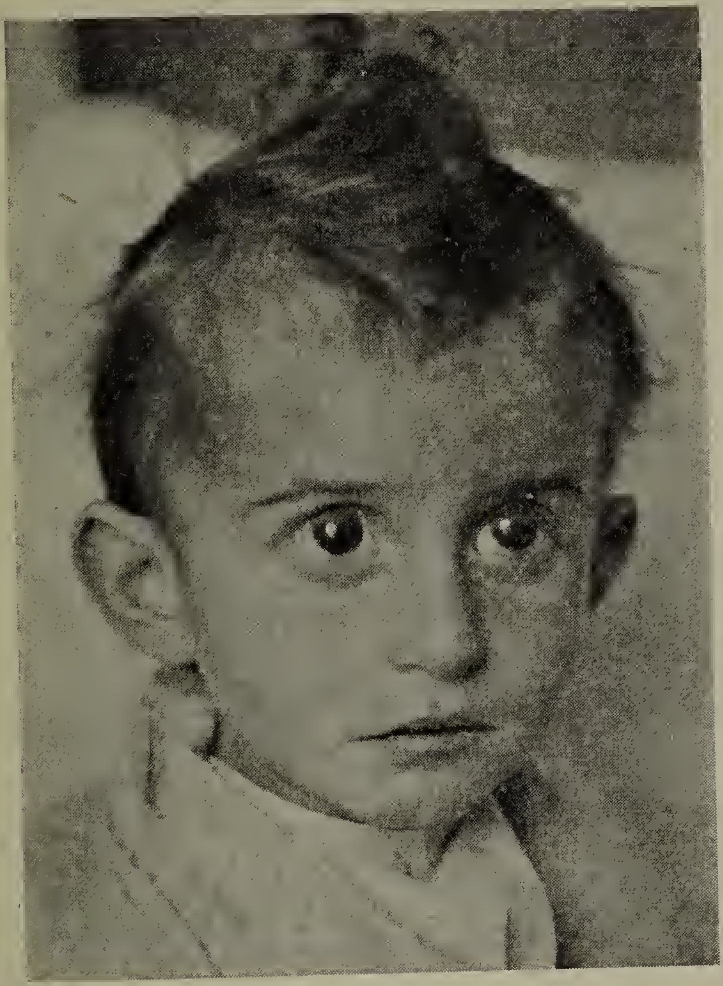

FIG. 29.—SUNKEN TISSUE ABOUT THE EYES.

Child of 19 months in the Children's Medical Ward of the Hospital of the University of Pennsylvania. Chronic intestinal indigestion with troublesome vomiting, mucus in stools, and loss of weight. Improved.

Head and Neck.—Various alterations of the head appear, some of which add to the altered expression of the face. In chronic hydrocephalus the head is globular, the forehead overhangs, and the face looks small. In rickets the head has in general a square or oblong form, with the top flattened and the frontal and parietal eminences unusually large.

The face looks small and is given a somewhat square appearance through the widening of the lower jaw. Asymmetrical heads are due to the pressure by forceps or oftenest to the existence of rickets, the lying too much on one side producing the deformity in the latter case. Faulty position may, however, produce the deformity even when rickets does not exist. Flattening of the occiput may be due to pressure, the result of the rachitic infant lying too constantly upon its back. Asymmetrical, microcephalic, and other deformed states of the head may be found in idiocy. Spots of thin membrane-like bone (craniotabes) may occur in the occipital region of infants in the early months of life. The fontanelle is unduly prominent and tense in hyperemia of the brain from any cause, meningitis, tumor, and chronic hydrocephalus. It is much enlarged with the sutures open in the latter disease, as it is to a less extent in rickets. It is depressed in conditions of inanition or after profuse diarrhea, and especially in collapse. Not infrequently the bones of the skull overlap under these circumstances. The fontanelle closes very early in microcephalus and late in rickets. A systolic murmur is sometimes audible in the neighborhood of the anterior fontanelle, especially in cases of rickets. It may also, however, sometimes be heard in healthy children. Tenderness over the tragus and over the mastoid may indicate otitis. Swelling of the occipital and superficial cervical glands is often the result of inflammation of the scalp; while that of the glands below the body of the jaw commonly attends affections of the pharynx and nasopharynx. A fluctuating swelling in the neck may be due to abscess of the glands or sometimes to a retropharyngeal abscess pointing here. The hair is worn from the back of the scalp in cases in which there is much rocking of the head, as in rickets.

Mouth and Throat.—Blueness of the lips has already been alluded to (p. 221). Fissuring around the mouth may be a symptom of congenital syphilis. Grinding of the teeth occurs especially in children with cerebral disease or suffering from convulsions, but it is also heard in those with slight digestive disturbance, and in some cases seems to be only an insignificant although disagreeable habit. Notching of the permanent upper incisor teeth is seen in congenital syphilis. The mucous membrane of the mouth may exhibit mucous patches in syphilis, and is one of the earliest sites for the appearance of the eruptions of some of the infectious fevers. A hemorrhagic swollen condition of the gums is seen in infantile scurvy. The tongue is coated in most disorders of digestion, but in some instances is bright-red and smooth. It is coated in many fevers; bright-red with prominent papillæ ("strawberry tongue") in scarlatina; thick and protruding in cretinism; furrowed and dry in Mongolism; worm-eaten in appearance in the so-called "geographical tongue;" cyanotic in congenital heart-disease and slightly so in pertussis, and may, like the rest of the mucous membrane of the mouth, exhibit the lesions of some of the forms of stomatitis. In children with severe cough, and especially with pertussis, who have cut the lower incisor teeth ulceration of the frænulum linguæ is not infrequent.

Cleft palate is a congenital defect, while perforations are generally the result of congenital syphilis. High arching of the palate may attend some forms of idiocy, but may also be present in children with perfect mental condition. It is not infrequently associated with deviation of the septum and the presence of adenoids.

Thorax.—The chest in rickets is small and exhibits the rachitic rosary in front and bulging of the ribs behind, with marked depression in the lateral regions. On horizontal section this gives the well-known "violin-shape." A horizontal depression beneath the nipples is also characteristic of rickets, especially where there has been much disturbance of the respiratory apparatus, and may be well-marked in obstruction by adenoid growths. A typical pigeon-breast is due often to obstruction to respiration by adenoids. This and other very great deformities of the thorax may be the result of curvature of the spine. A very unusual prominence of the precordium occurs in cardiac hypertrophy in heart-disease in children. Diminished expansion of one side with lack of movement of the intercostal spaces is dependent upon pleural effusion of that side, adhesions from a former pleurisy, or, in a lesser degree, upon pneumonic consolidation. Contraction of one side results from old pleural adhesions. Bulging of the intercostal spaces, with lack of movement, occurs in large pleural effusions; yet effusion may sometimes be present and the interspaces still move. Decided dyspnea from any cause produces in children great retraction of the interspaces with each inspiration. The degree of retraction of the epigastrium and of all the lower portion of the thorax, with tugging of the sternocleidomastoid muscles, and sinking of the episternal notch, is often remarkable at this time of life. It is generally greatest in cases of stenosis of the larynx. Yet a considerable amount of moving of the epigastrium combined with lower thoracic retraction is a normal accompaniment of respiration in healthy infants, and still more so in rachitic subjects. Displacement of the heart's apex by pleural effusion is to be noted. Dullness over the manubrium of the sternum or between the scapulæ may indicate the presence of enlarged bronchial glands. In infancy the substernal dullness may be produced by the thymus gland. Other regions of dullness of small size may be occasioned by old pleural thickening, encysted empyema, areas of collapsed lung, or small pneumonic patches. More extensive dullness may be due to pleural effusion, pneumonia, or widespread pulmonary collapse.

An unusually deep tympanitic percussion note or a Skodaic tympany may indicate in children the presence of pneumonic consolidation. It may persist throughout nearly the whole attack. A cracked-pot sound is especially often heard in advancing or receding pneumonia, although it is often, too, present in healthy lungs. Localized ringing râles of unusual loudness and nearness to the ear frequently signify pneumonic consolidation. Feeble respiration over one side of the chest may indicate pulmonary collapse or pleural effusion. It may, however, be the only discoverable evidence of pneumonia in some instances. This has repeatedly led to the erroneous supposition that that side was affected on which the loudest respiration was heard. In other cases of pneumonia a slight harshness of respiration is the only physical sign. The frequency with which numerous râles occur in bronchitis in children as compared with adult life is another interesting feature. Bronchial respiration generally denotes consolidation, but only when accompanied by other symptoms. As already stated it may often be heard through a pleural effusion, and is normally present to a certain extent in certain parts of the lungs. (See p. 211.) In the supraspinous fossæ it may at times be a sign of enlarged bronchial glands.

The various cardiac murmurs heard offer nothing peculiar in childhood to be mentioned in this connection, except that the characteristics of the murmurs of congenital disease of the heart are to be borne in mind, as well as the frequent occurrence of accidental murmurs. These will be referred to in considering Diseases of the Heart (Vol. II, pp. 378, 420). A very distinct precordial thrill occurring in a young infant points strongly to congenital heart disease.

Abdomen.—Unusual gaseous distention of the abdomen is a common symptom of rickets and is also often associated with colic. It is also present in wasting disease due to chronic digestive disorders, is a constant attendant upon idiopathic dilatation of the colon and upon some forms of indigestion in the later years of early childhood and is often a serious symptom in pneumonia. Great flatulent distention with much tenderness may occur in peritonitis, and to a less degree in inflammatory diseases of the intestine. Distention by liquid is noticed in tuberculous peritonitis and in abdominal dropsy from cardiac, renal and, more rarely, hepatic disease. Irregular distention by solid masses occurs in tuberculous peritonitis, fecal accumulation, enlargement of the liver and spleen, morbid growths, intussusception, and in localized inflammatory processes. Marked retraction of the abdomen is seen in cholera infantum, in meningitis, especially of the tuberculous variety, and in many exhausting diseases. Absence of movement during respiration may be due to inflammation or to paralysis of the abdominal walls. The outlines of the stomach and of the coils of intestine may often be detected in atrophic children with distention and great thinning of the abdominal walls. Quite active peristalsis is noticeable in cases of pyloric stenosis and of intestinal obstruction.

Method of Sucking and Swallowing.—Sucking may be almost impossible when the nares are occluded, as by severe acute coryza, congenital syphilis or unusual adenoid growth, since the infant cannot breathe while the mouth is closed on the nipple. Harelip or cleft palate likewise renders sucking difficult or impossible on account of the interference with the production of the necessary vacuum in the oral cavity. Refusal to nurse after making a short effort may indicate soreness of the mouth. In other cases it shows that little or no milk is obtained from the breast. Swallowing with a noisy gulping sound and with a grimace or a cry of pain occurs in soreness of the throat. Sucking for a moment and then stopping to breathe attends pneumonia, while entire refusal to suck may accompany extreme weakness or coma. It is an unfavorable sign. Inability to swallow, even when fed by a dropper or spoon, is seen in tetanus, eclampsia, stricture of the esophagus, and in children extremely ill from exhausting diseases. Choking over the food, with inability to swallow, occurs in severe cases of retropharyngeal abscess. Regurgitation through the nose indicates pharyngeal paralysis, oftenest after diphtheria.

Respiration.—Acceleration of breathing is very common in children, and the rate is out of all proportion to that which similar causes would occasion in adults. It is seen in fever and, very markedly, in pneumonia, and is constantly present to a variable degree in rickets, even when there is no catarrhal disturbance of the respiratory apparatus. The increase in the respiratory rate for each degree of temperature is approximately the same as in adult life, *i.e.*, about 2½ :1, with the rate of increase slightly greater than this in infancy and slightly less in later childhood (M. S. Cohen).[1] Any excitement will accelerate respiration greatly in infancy. Dyspnea, *i.e. labored* breathing—which may or may not be rapid as well—may occur in any condition which interferes with proper aeration of the blood. It is most typically seen in stenosis of the larynx from diphtheria, sometimes in retropharyngeal abscess, and in cases of foreign body in the trachea or bronchus. It may occur under other circumstances also, such as pneumonia, pleurisy, diseases of the heart, severe anemia, uremia and acidosis. In moderate dyspnea the inspiration is labored, prolonged and noisy; but in bad cases the expiration has the same character as well. Sometimes the rhythm of the respiration is altered, and instead of the inspiration being the louder and longer, with the pause following expiration, the latter is the louder and more accentuated and the pause follows a short inspiration. This is oftenest seen in pneumonia. It may, however, sometimes occur in healthy infants if excited. A catch in the respiration is frequently observed in abdominal or thoracic pain. It is witnessed very characteristically in pneumonia and pleurisy, in which the inspiration is short, and "catchy," and is followed by a moaning expiration, the so-called "expiratory moan." Snoring at night points strongly in children to occlusion of the nasopharynx by adenoid vegetations. A curious spluttering, gurgling respiration is heard in retropharyngeal abscess. The natural great irregularity of the respiration in young children, and especially in infancy, is much increased in cerebral affections. This greater irregularity may also sometimes be seen in painful affections, especially of the chest. Sighing, with unusual intermissions in the respiration, is a common symptom of meningitis, yet it may at times occur in healthy infants. A respiration approaching the Cheyne-Stokes type is frequently observed in early life in disorders of the brain, even though only functional in character. Although commonly of grave import, it is by no means so much so in infancy as in adult life. Great slowing of the respiration may take place in cerebral diseases, as in coma. It also occurs in narcosis from opium. Frequent yawning may indicate serious failure of the circulation or sometimes impending syncope.

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[1] Arch. of Pediat., 1905, XXII, 917.

Vital Capacity.—The vital capacity as measured by the spirometer (Emerson and Green;[1] Edward and Wilson;[2] Stewart and Sheets[3]) is diminished in heart-disease in proportion to the degree of respiratory embarrassment dependent upon loss of compensation. There is temporary reduction of it in acute bronchitis; very great in pneumonia; temporary immediately after an attack of asthma, and none at all in tuberculosis unless there is extensive involvement of the lung. Dyspnea on exertion is much more marked when reduced vital capacity results from cardiac disease than when it is caused by disease of the lungs.

Pulse.—The pulse becomes more rapid in febrile conditions and under the slightest excitement. This last is so true that it is almost impossible to come to any conclusions regarding it when an infant is awake, unless entire placidity is obtained. Other things being equal, and all undue excitement being removed, the increase for each degree of temperature is not so great as in adult life, and the younger the child, the less is the relative augmentation, the increase for 1 degree Fahrenheit being about 4 beats of the pulse during infancy. This has been pointed out by Cohen.[4] The truth of this statement, which is contrary to the opinion generally held, will be readily admitted on slight consideration. If an infant of a year with a normal pulse-rate of 110 to 120 showed an increase of 10 beats for each degree of temperature, as does an adult, an elevation

[1] Amer. Jour. Dis. Child., 1921, XXII, 202.

[2] Arch. Int. Med., 1922, XXX, 638.

[3] Amer. Jour. Dis. Child., 1922, XXIV, 83; 451.

[4] Arch. of Pediat., 1905, XXII, 915.

of 105 degrees would produce a pulse-rate of 180 to 190, which is clearly more than is usually obtained for fever in otherwise healthy and quiet infants. The relationship of the rate of the pulse to that of the respiration, which is 4 : 1 in adults unaffected by any disease which unduly influences either one or the other, is altered somewhat in infancy, being then about 3 : 1 (Squire).[1] The natural irregularity of the pulse in young children (see p. 49 and Vol. II, p. 419) becomes much intensified in many affections of the brain. In such disorders irregularity is often combined with decided retardation. The pulse is to a less extent irregular in pericarditis and often in chorea. It is unusually rapid in scarlatina, out of all proportion to the severity of the attack, and slower than would be expected in many cases of typhoid fever. It may be slightly retarded in nephritis and the arterial tension is increased. The "trip-hammer" pulse of aortic regurgitation occurs in childhood as in adults; the capillary pulse of this disease may be readily obtained in the finger-nails or in the lips, and the pulsus paradoxus is found in adhesive exo-pericarditis.

Temperature.—Most important in this connection is the observing of the ease with which abnormal alteration of temperature takes place in early life. High elevation may result from slight causes, such as constipation of the bowels or even excitement. Moro[2] found that 22 out of 25 children experimented upon showed elevation of the rectal temperature after vigorous exercise. Temporary fever is not uncommon in the new-born. (See p. 340.) More or less irregular elevation may be seen in children with moderate debility, especially during convalescence from an acute disease. The variations in the course of a febrile temperature, including the difference between morning fall and evening rise, are liable to be greater in children than in adult life, and the thermolabile condition of certain children must not be forgotten in estimating the importance of elevation of temperature. In fact in some children, especially those of a highly neurotic nature, the temperature is persistently elevated during at least a part of the day, even with the child at rest, without this possessing any significance. Very high temperature may attend the infectious fevers, some of the cases of milk-poisoning, and the heat-exhaustion occurring in very hot weather. High fever of short duration is borne, as a rule, better in childhood than in adult life.

Depression of temperature is witnessed in such conditions as after the crisis in such diseases as pneumonia; severe diarrheal diseases; collapse; hemorrhage; sclerema neonatorum; congenital heart-disease; premature birth, and in very many cases where insufficient nourishment is taken or assimilated. In all weakly children the temperature is readily depressed by external cold.

Cry.—The observation of the cry constitutes one of the most important methods of diagnosis in infants. A healthy, comfortable and contented infant does not cry. A cry of any sort always has a meaning, even though it indicates nothing more than some slight dissatisfaction. Persistent violent crying, rather fretful than sharp, is often due to hunger. It is unappeasable by anything except the giving of food, when it ceases at once and permanently. Sometimes a cry of this nature is in reality dependent on thirst, especially if there has been severe diarrhea. A similarly continuous cry, but more high-pitched and piercing, attends persistent severe pain, most commonly earache. The offering of food quiets it only momentarily if at all. Pain of a less severe nature, the existence of the intense itching of eczema, the pain from the pricking by a pin concealed in the clothing, the presence of a wet diaper, and many other sources of discomfort produce obstinate crying, but of a less piercing and violent character. The cry of colic is very violent but more or less paroxysmal, a momentary pause being followed by a sudden renewal without discernible reason. It is attended by the movements of the body already described as characteristic of colic (p. 220). The giving of food may quiet it for a time, the warm milk lulling the pain, but it soon returns as bad as before. It may cease suddenly after the expulsion of gas from the stomach or bowel. A similar cry sometimes attends the passage of gravel or may be observed in intussusception. A sudden acute pain, such as results from a fall or other slight accident or the touching of some tender part, produces violent but temporary crying, soon appeased. Crying just before, with, or after the evacuation of the bowels indicates intestinal pain, or sometimes pain at the anal opening. Crying may also attend the passage of urine and may denote pain in the bladder or the irritation of scalded areas by the secretion.

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[1] Transac. Obstet. Soc. Lond., 1868, X, 280. Ref., Cohen, *loc. cit.*

[2] Monatsschr. f. Kinderheilk., 1917, XIV, 214.

A weak, peevish, fretful cry, sometimes almost constant, is heard in many conditions attended by much debility. Under such circumstances speaking to or even looking at the child may start the cry. A louder but fretful cry in a healthy child, often attended by rubbing of the eyes with the fists, indicates sleepiness. An almost inaudible cry occurs in severe stenosis of the larynx and in cases of great exhaustion. Puckering the face into the position for crying, but absolutely without sound resulting, occurs in these conditions when extreme, as also after tracheotomy or intubation. The absence of crying is also witnessed in comatose states. There is likewise very little crying accompanying decided dyspnea, such as attends severe pneumonia or pleural effusion, on account of the lack of air for it. The cry of pneumonia is suppressed and short, and the expiratory moan described in considering Respiration is often heard (p. 227). Yet children with pneumonia sometimes cry loudly if the dyspnea is trifling. As a rule, however, loud crying indicates that there is little wrong with the lungs. Hoarseness of the cry is heard in laryngitis, and a hoarse, whimpering, and somewhat nasal cry occurs in congenital syphilis. A nasal cry is present also in coryza of other nature. A characteristic "brazen" cry is heard in spasmodic croup. A short cry of pain after coughing denotes pain, as in pneumonia or pleurisy. Crying is usually unattended by the production of tears until about the 3d month. After this date crying without tears indicates a condition of dangerous debility. A sudden shriek at intervals, without ordinary crying, uttered by a child in a stuporous state, suggests tuberculous meningitis ("hydrencephalic cry"). Sudden crying out at night may, however, be produced by the night-pains of disease of the bones. After infancy is passed violent, unappeasable crying, with which a child suddenly starts from sleep, is indicative of night-terrors. In later infancy and childhood the cry of anger is often witnessed. It is loud, violent, without any piercing character, unattended by any evidence of pain, and generally associated with some evident reason for wrath. The infant while crying from this cause often stiffens itself all over, or throws its head backward; while the older child may stamp its feet, throw itself upon the floor, and even beat its head against the floor or wall. Finally, there is the very common and very deceptive cry occurring in infants who have in various ways not been well trained, which is merely an expression of discontent with their condition, although without anger or pain. A baby, for instance, wakens from sleep and cries violently. As soon, however, as it is taken up by the nurse its crying ceases, and smiles replace the tears.

Cough.—A short suppressed cough followed by a facial expression of pain is heard in pneumonia and pleurisy; a peculiar barking, brazen cough in spasmodic croup or the early stage of laryngeal diphtheria; a tight hoarse cough in laryngitis and tracheitis. Long, hard paroxysms of dry cough sometimes causing pain in the chest occur in the early stages of severe bronchitis, and a loose rattling cough in bronchitis after secretion is established. The long paroxysms of rapid, short expiratory efforts, continuing until suffocation seems impending and followed by a crowing inspiration, are characteristic of pertussis. In this disease mucus is often driven from the mouth. A very similar cough may occur in enlargement of the bronchial glands, yet not often accompanied by a whoop. It may be particularly troublesome at night, as the cough in pertussis is likewise. A peculiarly severe, ringing, brazen cough, in some respects resembling that of croup but often paroxysmal, is sometimes caused by the existence of glandular or other tumors or abscesses within the thorax, or the presence of a foreign body in the air-passages. An annoying "tickling," hacking cough occurs in pharyngitis, especially when the uvula is elongated. When the pharyngitis is severe the cough causes pain in the throat. A hard dry cough, often severe, is heard in passive congestion of the lungs produced by disease of the heart. Indigestion is frequently attended by a hacking cough, the so-called "stomach cough." Asthma has a short dry cough, not paroxysmal.

Voluntary expectoration of *sputum* following cough does not, as a rule, take place in any disease until 6 or 7 years of age. Sputum for examination must be obtained by introducing an elastic catheter or a pledget of cotton or cloth on an applicator to the base of the tongue. This occasions coughing, whereupon the sputum may be aspirated by the catheter or caught on the cloth.

Pain.—Pain, either subjective in origin or produced by handling, is of the most varied form and significance in early life. It has already been discussed to some extent under different headings. The determination of the seat of pain in infants and young children is often very difficult. Frequently tenderness can be ascertained by handling various parts, and observing whether a cry or a grimace is produced. Children under 5 years seldom locate pain exactly in attempting to describe it. Pain in the head is very common. It may indicate the onset of fever, whether this be of short or of more prolonged duration, being a frequent early symptom of typhoid fever and still oftener of meningitis. It is common in intracranial tumor. In other cases it is dependent upon coryza, anemia, dyspepsia, eyestrain, otitis, fatigue, excessive mental work at school, migraine, dental caries, nephritis or heart-disease. Pain referred to the mouth may indicate stomatitis of various sorts, or toothache. In the throat it may attend many forms of inflammation there. Pain in the neck may be due to inflammation of the lymphatic glands, mumps, tonsillitis, or the tenderness of the muscles in torticollis. It may also be a symptom of caries or of basilar meningitis, especially when the pain is increased by motion. Pain in the thorax depends most frequently upon pleurisy and pneumonia. It may then be present only during cough. Occasionally pain in one side is produced by herpes zoster. Heart-disease is sometimes attended by severe attacks of precordial pain. Tenderness of the chest on grasping the child under the arms to lift it may occur in pleurisy, but is most marked in rickets, probably due to a scorbutic complication. The symptom may be deceptive, as the pain can be in reality located in some other part of the body, which the lifting has disturbed. Pain in the abdomen depends upon digestive disturbances, peritonitis, appendicitis, or intussusception, and is not infrequently a symptom of spinal caries. In the form of "umbilical colic" it is most frequently of nervous origin. Inflammatory affections of the chest quite frequently produce a pain which is referred to the abdomen. Pain with stiffness on moving the back strongly suggests caries of the spine. In the limbs it may depend upon rheumatism or other form of arthritis, or on poliomyelitis, but in infancy is far oftener a symptom of scurvy. It becomes particularly evident when the child is handled. Pain is liable to attend inflammatory affections of the bones, among which may be mentioned periostitis and osteomyelitis. The existence of undiscovered fractures in infancy is a fruitful and puzzling source of severe pain. Hip-joint disease occasions pain which is referred to the thigh or, commonly, to the knee. Caries of the spine may produce pain in the lower limbs. An unusual degree of local or general hyperesthesia is not uncommon in different nervous disorders.

Breath.—A rancid, butyric-acid odor may be present on the breath of infants suffering from the vomiting of gastric indigestion. Acute febrile conditions or acute indigestion may produce that of acetone, or give rise to other odors of an unpleasant character. In ozena, ulcerative stomatitis, the accumulation of secretion in the tonsils, and the sputum from pulmonary abscess and from bronchiectatic cavities the odor may be very offensive, while in gangrene of the lung and in noma the sickening odor is almost unbearable. A stercoraceous breath is exceptionally noticed in intestinal obstruction.

Vomiting.—Vomiting is a symptom so frequent in early life and due to such varied causes that it will receive independent consideration later (Vol. II, p. 60). In this connection a few of its diagnostic indications may be mentioned. Simple regurgitation, without effort, of food which is little if any changed, occurs in the case of healthy infants who have taken more milk than they can comfortably hold, or who have been carelessly handled after feeding. True vomiting is accompanied by more effort and by evidences of nausea, such as pallor of the face and perspiration. It is very common at the beginning of acute febrile diseases in early life. When acute and of brief duration, accompanied by nausea and coated tongue, and perhaps followed by diarrhea, it is generally the sign of acute gastro-intestinal disturbance. Very obstinate vomiting, frequently with much mucus, occurs in chronic gastric indigestion and gastritis in infancy and in cases of stenosis of the pylorus. Vomiting may be the evidence of a toxic state, as seen, for instance, in uremia and in acute milk-poisoning. If repeated frequently for days, and attended by retracted abdomen, headache, moderate fever, and some degree of constipation, it is very suggestive of meningitis, or, in the absence of fever, of brain-tumor. Recurrent vomiting is a disorder not to be forgotten. Vomiting is common after violent paroxysms of coughing, especially in pertussis and severe bronchitis. Very intractable vomiting with distention of the abdomen and some degree of constipation occurs in peritonitis. When obstinate constipation is combined with obstinate vomiting, the existence of obstruction of the bowels, sometimes congenital, may be suspected.

Finally, the extreme ease with which vomiting is brought about in infants and in children must be borne in mind. This is especially true when once the tendency to vomit has been developed. Thus in many infants slight moving of the body after a meal is sufficient to cause the loss of it. The mere taste of the food may occasion vomiting, both in infants and older children, everything being ejected the taste of which is not liked. This is probably the reason why nourishment given to infants by gavage may be retained, when that entering the mouth in the usual way may be vomited.

Bowel-movements.—Alteration of the character of the feces is common in disease in infancy. A large amount of white, lumpy material in the stools, especially of bottle-fed infants, generally indicates too large an amount of fat in the food. Mucus appears very readily in the stools of infants. It may indicate merely a catarrhal process, but in large amount and accompanied by fever and straining suggests an inflammatory condition. It is generally present in considerable quantity after the administration of a purgative, such as castor oil. Blood in the passages may denote a purely local process, such as fissure, hemorrhoids, congestion, or constipation. Mixed with mucus and attended by straining efforts it may be the evidence of enterocolitis or intussusception; and when in larger amount, without mucus, may be one of the symptoms of purpura hæmorrhagica, severe ulceration, or rectal polypi. Black stools suggest the existence of hemorrhage in the upper intestinal tract. A pea-green color is often physiological, but the presence of dark-green slimy masses is very frequent in intestinal indigestion and in enterocolitis. Putty-colored stools may be due to deficiency in the secretion of bile, but oftener in infancy white or grey, formed or unformed stools are seen in those who are taking much more butter-fat than they can digest. Hard, scybalous masses coated with mucus occur in older children with forms of intestinal indigestion of a chronic nature. Intestinal parasites of different sorts or their ova may frequently be discovered.

The *odor* of the stools during disease varies. In cholera infantum the passages are nearly odorless; in other forms of diarrhea in infancy they may be sour-smelling and irritating to the adjacent skin, have a peculiar "mousey" odor or may be very offensive, the difference depending upon whether indigestion of carbohydrate or of protein is present. The giving of such substances as beef-juice and egg-water often produces exceeding unpleasant odors. The action of certain drugs and food upon the color of the passages is to be remembered; iron producing a black, and bismuth a greenish-black hue, and such substances as hematoxylon and krameria, formerly much in use, a reddish color. Infants fed upon a high carbohydrate-diet, such as malt-soup or buttermilk-mixtures, exhibit smooth, brownish stools. A fuller discussion of the character of the feces in disease will be given later. (See Vol. II, p. 97.)

The *number* of passages varies greatly; and in estimating the importance of this matter, the character and amount of the food taken must be considered carefully. Constipation in infancy is sometimes a sign of a very thorough digestion which leaves little waste-material to pass; in other cases it may depend on the great length of the sigmoid flexure in early life; in others upon impaired general health, and in still others, when combined with a failure to gain in weight, it may be caused by insufficient nourishment. On the other hand, an undue number of movements, unless diarrheal in character, may be merely a natural method of getting rid of an excess of nourishment. Frequent, diarrheal stools may be due to inflammatory conditions of the mucous membrane, or tuberculous or other ulceration; toxic influences, as in the infectious fevers; local irritation such as by improper articles of food; or reflex causes, as seen in diarrhea after surface-chilling.

Urine. (See also Vol. II, p. 426.)—*Retention of urine* may be dependent upon the pain which urination causes, as in vulvitis, inflammation of the prepuce or glans, or the smarting produced by very acid urine. Sometimes it is the result of obstruction, as in cases of calculus in the urethra, very narrow foreskin, malformations of the urinary tract, or stone in the bladder. *Suppression* or very *great diminution* in the amount of urine may attend acute nephritis, acute fever, profuse diarrhea, severe vomiting, renal calculus, and sometimes intestinal obstruction. *Great increase* in the amount of urine occurs in diabetes, diabetes insipidus, after crisis in fever, after attacks of abdominal pain or of convulsions, and in some forms of chronic Bright's disease. *Albuminuria* is occasionally physiological. It occurs also in Bright's disease, in severe cardiac affections, in many febrile states, sometimes in scurvy, and from the admixture of pus or blood. In old specimens of urine it may depend upon the growth of bacteria. *Blood* in the urine, unless in very small amount, makes it appear smoky, muddy or bright-red in color. It may occur in Bright's disease, scurvy, in some grave cases of infectious fevers, purpura, stone in the bladder, and especially in stone in the kidney. Often the presence of blood manifests itself most noticeably by a stain on the diaper. Sometimes the altered color is due to hemoglobin and not to the presence of corpuscles. This is true of infectious hemoglobinuria of the newborn. A paroxysmal hemoglobinuria is occasionally seen in older children. The urine is of a reddish-yellow color in jaundice. Uric acid sand may appear on the diaper of infants, while in other cases an excess of urates makes the urine of infants milky in color. Pus in the urine is found in inflammation of any part of the urinary tract, as pyelitis, cystitis or urethritis. It also results from contamination by the discharge in vulvovaginitis.

Blood.—The significance of the alterations of the blood in disease depends upon the variation in the amount of hemoglobin, and in the number and character of the erythrocytes and particularly of the leucocytes, the presence of pathological corpuscles and not infrequently the alterations shown by chemical examination. The matter can best be considered in connection with the various diseases of the blood (Vol. II, p. 757).

Serous Cavities.—By the puncture of the pleural cavity the existence of effusion is made known and the character of the fluid determined. The diagnosis between pneumonic consolidation and pleural effusion often hinges upon the result of this procedure. By lumbar puncture the existence of forms of meningitis can be positively determined in most cases. The diagnosis of the nature of an abdominal disorder may, in like manner, be aided by the employment of puncture in some instances.

The appearance and character of the fluid obtained by the puncture of serous cavities, including the kind of formed elements present in it, is shown by microscopical and chemical examination. The presence and nature of any bacteria found must receive careful consideration. The matter can better be studied in another connection. (See Puncture of Serous Cavities, p. 214 and Pleurisy, Vol. II, pp. 357, 369.)

Mental and Nervous Symptoms.—In what has already been said incidental reference has been made to various diseased states of the nervous system. What follows is by way of addition or recapitulation. Further discussion will be found in the section upon Nervous Diseases (Vol. II, p. 505).

Various forms of mental defect are to be recognized. The mother often wrongly supposes that the child's failure to take notice, walk, or talk is due respectively to defective sight or hearing, paralysis of the legs, or tongue-tie. To determine whether failure to notice objects at the proper age is due to idiocy or to blindness is often a matter of difficulty. An ophthalmoscopic examination may settle the question. Slowness in learning to speak, when not dependent upon imbecility, can be the result of defective hearing or of a general slowness of development caused by ill-health. Often, too, it seems to be merely a peculiarity of the individual, irrespective of other conditions, and need then cause no anxiety. A marked tendency to allow the saliva to dribble out of the mouth is a characteristic of the idiotic state. A condition of confusional insanity, almost maniacal at times, may develop after fevers, especially typhoid. Simple anger becomes so uncontrollable in many children that it occasionally seems almost maniacal. Delirium is a frequent attendant of high temperature in children, and does not necessarily indicate the presence of serious disease. It may occur even in infancy, although at this age convulsions tend to replace it. Convulsions, indeed, are very prone to develop in early life. They may be due to many causes, often of the most trivial nature. Pneumonia and all the infectious fevers, especially scarlatina, may be ushered in by them, and even high temperature from other sources is likely to produce them in some subjects. Spasmophilia is a peculiarly frequent predisposing cause. They may result, too, from peripheral irritation of various sorts, particularly of the gastrointestinal tract, diseases of the brain, and uremia. Occurring in the new-born they are frequently the sign of intracranial trauma or disease.

Hysteria may be witnessed even in early childhood, and the presence of malingering dependent upon this is often to be recognized. In fact, the power of imagination is so great in childhood that pains are very liable to be complained of if the suggestion of pain is made to the child. Aphasia in children has not the grave import or the diagnostic value of the symptom in later life. It sometimes occurs temporarily after typhoid fever.

Psychic and nervous symptoms are much more easily evoked in early than in adult life. Great restlessness or excessive talking during sleep, somnambulism, and such semi-delirious states as are seen in night-terrors illustrate the readiness with which the mental condition of children is affected by such slight causes as temporary fever, indigestion and overexertion. Extreme excitement, with crying, may be due to disease or may be dependent only upon accidental causes. Great irritability is evidence of an unhealthy state. Sometimes it may be merely the result of indigestion or malaise, but in other instances it points strongly to the beginning of meningitis of the tuberculous form. Indifference to surrounding objects and the ceasing to play with toys indicates a serious affection of health. It may depend upon great weakness or upon intracranial disorder. An unusual desire to go to bed, or an unwillingness to get up at the usual time may be a distinctly suspicious indication. Stuporous and comatose states with the attendant symptoms occur in actual organic disease of the brain, or may be the result only of severe functional disturbance of it. Thus the condition of "pseudo-hydrocephalus," very closely resembling meningitis, may develop as an attendant upon great weakness, especially after severe diarrhea. Absence of the patellar reflex may occur in poliomyelitis, the muscular dystrophies, diphtheritic and other neuritic paralyses and in Friedreich's ataxia. Increase of it may be a symptom of cerebral palsy, Pott's disease, disseminated sclerosis and of some forms of cerebellar disease, or may be without serious import.

CHAPTER XI

MORBIDITY AND MORTALITY

TENDENCY TO DISEASE IN INFANCY AND CHILDHOOD

The greatest tendency to disease in general appears to exist in the 1st year of life, and especially in the new-born; the least at the age of from 10 to 15 years. There is besides, as has already been indicated (p. 202), a predisposition to certain diseases during infancy and childhood as contrasted with adult life, and a comparative freedom from others. A difference exists, too, in the susceptibility to particular diseases at the various periods of early life, different stages of development being attended by different maladies. Thus in the new-born exists a group of affections, almost characteristic, which are due either, first, to disease or defects of development persisting from fetal life, or, second, to injuries or disease arising at birth or very shortly after it. In the first class are all the malformations and congenital disorders, such as those of the heart, intestines and brain, as also the different forms of imperfect development visible to the eye, and the inherited affections, especially syphilis. There is also the state of congenital asthenia with which very many infants appear to be born and which in no way depends necessarily upon premature birth, although a common result of this. In the second group are the injuries occurring at birth, which result from parturition, notably the forms of paralysis dependent often upon meningeal hemorrhage or direct injury of the brain or nerves, hemorrhage into some of the other viscera, the various dislocations and fractures, and injuries connected with the surface of the body, such as cephalhematoma. In this group, too, are a number of diseases which are largely peculiar to the new-born, such as forms of septic infection, atelectasis, pyloric stenosis, certain types of pneumonia and icterus, ophthalmia of the new-born, etc.

From the end of the first 3 or 4 weeks of infancy, up to the end of the 1st year of life the prevailing diseases are those connected with the digestive apparatus, over one-half of them belonging to this category and being the result in most instances of artificial feeding. The nervous system is also unusually sensitive, and convulsive disorders are prone to occur. The general feebleness of the young infant and its deficient power of reaction render it less able to resist cold, excessive heat, and other debilitating influences. It is particularly in this year that children seem to fade away under the influence of torrid weather. Diseases of the respiratory tract are very common, and pneumonia is frequent and liable to be of the catarrhal form, and often fatal. Scurvy is seen especially at this time, as are the earlier manifestations of rickets. Affections of the thymus gland are to be noted. Acute infectious diseases, as a class, are not so liable to occur, with the exception of pertussis and the septic infections, and to a less extent measles. Diseases of the skin are common, thrush is a frequent affection, and tuberculosis is often encountered, usually in the form of a general tuberculous infection. Inflammation of the lymphatic glands begins to be frequent at this age, particularly of the internal glands, and may be either simple or tuberculous in nature.

The 2d year of life sees the respiratory diseases continuing to prevail, while the digestive disorders, though still frequent, are, on the whole, less so, and less fatal in their results. Convulsions still occur very readily from the slightest causes. Meningitis is common, rachitis extremely so, and pyelitis often seen. Diseases of the lymphatic glands, especially in connection with other disorders, continue to prevail with a frequency characteristic of early life. Adenoid growths of the nasopharynx and hypertrophy of the tonsils become frequent. In this period, too, the marked disposition to the occurrence of acute infectious fevers, especially diphtheria, begins to be manifested. Throughout the first 2 years of life acquired affections of the heart are very uncommon and rheumatism rare. Tuberculosis shows itself as tuberculous peritonitis, adenitis, meningitis, in the form of tuberculous bronchopneumonia, and very often as a widespread general tuberculous infection. Typhoid fever is relatively much less frequent than later, and its symptoms often obscure.

During early childhood; *i.e.*, from the age of 2 years to that of 6 years, there is a predisposition to certain forms of digestive disturbances, particularly disorders of the stomach and small intestine. Diarrhea is less troublesome than in infancy. More or less malnutrition is of great frequency, often dependent upon mild digestive disorders. Tonsillitis, pharyngitis and various forms of stomatitis increase in frequency. Respiratory affections are very common, and spasmodic croup becomes one of the terrors of the household. Rheumatism and acquired affections of the heart begin to be observed although still not common. The tendency to the acute infectious diseases appears to be approaching its height. Appendicitis, rare under the age of 2 or 3 years, begins to be often observed.

In later childhood; *i.e.* after the age of 6 years, chorea becomes frequent and rheumatism and disorders of the heart also. Diseased conditions of the tonsillar tissue are common. Functional neuroses dependent upon school-life are to be noted, as are also myopia, scoliosis, headache, anemia, and the like. The infectious diseases continue extremely frequent. Meningitis is of common occurrence, and various psychoses appear as puberty is approached.

Chronic affections of the kidney, with the exception of pyelitis, are infrequent during the whole of infancy and childhood, unless as a result of scarlet fever. Acute nephritis may readily attend the infectious diseases, and often in infancy is associated with severe diarrheal disturbances. Disease of the bones and joints, generally of a tuberculous nature, is frequent in all periods of early life, although less so in infancy than in childhood. Disease of the brain-substance is rare except when secondary to meningeal disturbance, and that of the spinal cord, nerves, and muscles likewise so, the exceptions being poliomyelitis, which is common in infancy and early childhood, the neuritis following diphtheria, the transverse myelitis resulting from spinal caries, and the muscular dystrophies and certain systemic nervous affections which are characteristic of childhood.

FETAL MORTALITY. STILL AND PREMATURE BIRTHS

Fetal mortality, including miscarriages and still-births, shows a high percentage of the total number of conceptions. Whitehead[1] calculates the proportion of miscarriages to pregnancies as 1 :7, and Priestly[2] as 1:4⅓. Ingraham[3] observed a fetal mortality of 4.6 per cent. in 3000 births; ½ of these deaths being still-births. In 28 cities of the United States in 1890[4] the number of still-births bore to the number of children born alive a ratio of 62.65:1000; *i.e.* 5.9 per cent. of the total births were still-births. Howard[5] in 1921 found the average percentage of still-births to the total births in the Registration Area of the United States for 1918 equalling 3.44. An earlier estimate of the European states made the still-births 3.79 per cent. of the total births (Wappäus).[6] Ruppin[7] gives the still-births of Prussia during 25 years as 3.69 per cent. of the total births and Chambrelent,[8] for different parts of France during 3 years, estimated from 2.6 to 7.3 still-births for every 200 born alive.

Premature birth is also a frequent cause of death during infancy. This might, in a sense, be included in fetal mortality, since death finally occurs because the organism is not in a state fully prepared for life outside the uterus. The Census statistics of the United States for 1890[9] make 5.68 per cent. of all the deaths under 1 year dependent upon premature birth. This is probably below the actual figures, since many deaths attributed to inanition and similar causes might properly have been classified with those resulting from premature birth. From a study of the statistics of 6 English hospitals, Eden[10] found that from 30 to 45 per cent. of all premature infants died while still in the hospital.

MORTALITY IN INFANCY AND CHILDHOOD

Death-rate Especially in the First Year.—The conditions shown by some of the statistics which follow have been distinctly improved in more recent years. (See Diminution in the Death-rate, p. 240.) It is a matter of common observation that the younger the child the more dangerous, as a rule, is any disease affecting it. The exhaustive studies of Eröss[11] based on the vital statistics of 13 European States for a number of years, showed that an average of 18.33 per cent. of all children born died in the 1st year, equaling 26.89 per cent. of the total mortality for all ages. Of the deaths in the 1st year, more occurred on the 1st day than on any other. Of the fatal cases in children under 1 year, 31.86 per cent. took place in the 1st month, and 73.13 per cent. in the first 6 months. Nearly ½ of the fatal cases of the 1st month were in the 1st week. Later investigations support these figures, those by Holt and Babbitt,[12] showing that of 100 deaths in the 1st year, 13 occurred on the 1st day, 22 in the 1st week, 28 in the first 2 weeks, and 33 in the 1st month. In Philadelphia in 1917, 39 per cent. of the infants who died in the 1st year did so in the 1st month (Mitchell).[1] All this shows a rapidly decreasing mortality in proportion as the infant grows older. This diminishing mortality, with subsequent increase as adult life advances, is illustrated graphically in the accompanying curve (Fig. 30) after Westergaard.[2]

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[1] Ref. Ballantyne, Disease of the Fetus, 1892, I, 8.

[2] Ballantyne, *loc. cit.*

[3] Amer. Jour. Obst. and Gynec., 1921, I, 821.

[4] U. S. Census, Vital Statistics, II, 142.

[5] Amer. Jour. Hyg., 1921, I, 197.

[6] Allg. Bevölkerungsstatistik, 1859 Ref. Pfeiffer, Gerhardt's Handb. d. Kinderkr., I, 551.

[7] Schmidt's Jahrbücher, 1902, CCLXXIII, 233.

[8] Le Nourrisson, 1920, VIII, 321.

[9] *Loc. cit.*, IV, 666.

[10] Lancet, 1921, II, 127.

[11] Zeitschr. f. Hygiene, 1895, XIX, 371.

[12] Jour. Amer. Med. Assoc., 1915, LXIV, 287.

The statistics of the United States for the census-year ending in 1890[3] give very similar results regarding the high mortality of the 1st year. A percentage of 18.29 deaths occurred in the 1st year of every 100 born alive in the Registration area.[4] During 6 years' observation in this area 251,424 deaths occurred under 1 year, a percentage of 29.71 of the total number of deaths for all ages. Of these deaths in the 1st year 55.77 per cent. were in the first 3 months, 18.19 per cent. at from 3 to 6 months, 14.35 per cent. from 6 to 9 months and 11.62 per cent. from 9 to 12 months. In the Registration area in 1918, ⅔ of all the deaths in the first 5 years occurred in the 1st year (Reuben).[5]

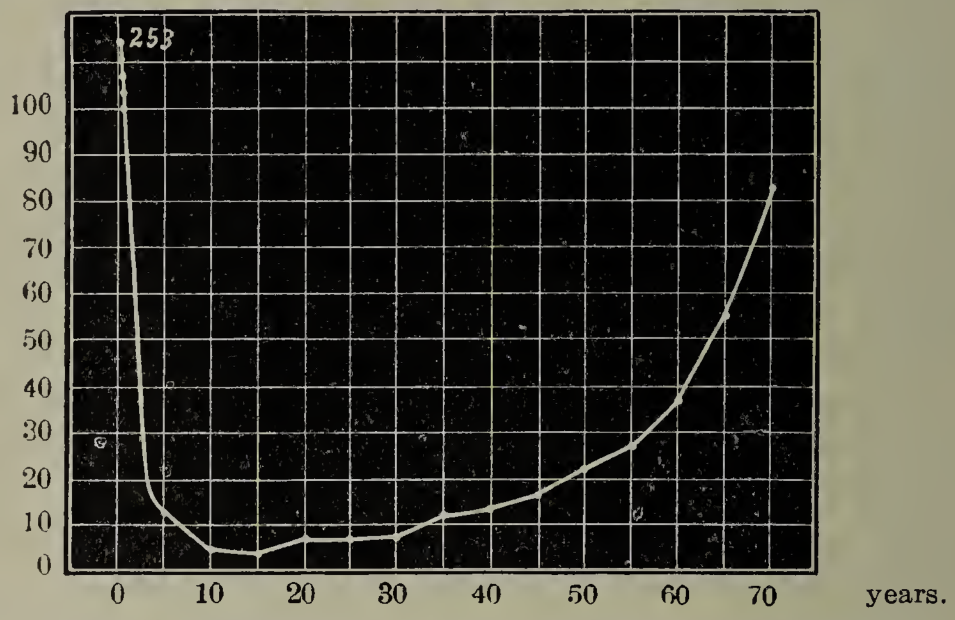

Fig. 30.—Graphic Chart Representing the Number of Deaths for Different Years for Every 1000 Persons of Each Age.

The figure 253 represents the number of still-births. (*Westergaard, Die Lehre v. d. Mortalität, und Morbidität.*, 1901. *Ref. Prausnitz, in Pfaundler and Schlossmann's Handb. der Kinderheilk.*, 1906, I, 1, 279.)

There is considerable variation in the mortality-rates according to *geographical distribution*, the relative number dying in the 1st year in the countries studied by Eröss being lowest in Ireland (9.4 per cent.) and Sweden (9.7 per cent.) and 3 times greater in Saxony (28.1 per cent.) and Bavaria (28.7 per cent.). It was still higher in Russia (32.6 per cent.) (Gundobin).[6] Johannessen's[7] statistics for Norway gave a mortality of 9.8 per cent. The influence of geographical distribution probably depends, at least in part, upon climatic conditions and upon the attention paid to preventive measures by the local authorities or the public.

The influence of *crowding and bad hygiene* is very positive. The infant-mortality is higher in the poorer classes and is constantly greater

[1] Arch. of Pediat., 1920, XXXVII, 151.

[2] Die Lehre von der Mortalität und Morbidität, 1901. Ref. Prausnitz, Pfaundler und Schlossmann's Handb. der. Kinderheilk., 1906, I, 1, 279.

[3] *Loc. cit.*, I, 22.

[4] The "Registration area" for that date included the registered states, Massachusetts, Connecticut, New Hampshire, Vermont, Rhode Island, New York, New Jersey and Delaware; the District of Columbia, and 83 registration cities in non-registration states.

[5] New York Med. Jour., 1922, CXVI, 462.

[6] Jahrb. f. Kinderheilk., 1898, XLVII, 368

[7] Jahrb. f. Kinderheilk., 1902, LVI, 259.

in cities than in the country. Many of the larger cities of Europe and America have shown a mortality in the 1st year of from 20 to 30 per cent. of those born (Deutsch).[1] The mortality is nearly always greater among illegitimate children as a class, for various reasons not dependent on the innate vitality of the child. Deaths in homes, asylums, and hospitals for infants are extremely numerous, in spite of all efforts to prevent this. In some of the poorly managed institutions the mortality has reached, or even exceeded, 90 per cent., and in the best it has often averaged 50 per cent. or 60 per cent. The causes of the high mortality depend partly on the character of the infants in these institutions, partly on the improper care given in some of them, partly on the spread of disease from one child to another, and largely on a general, imperfectly understood, deleterious influence which attends the keeping of many infants together in one place, and to which the title "hospitalism" is often applied.

If the production of large families increases the death-rate among the infants, as claimed by Hers[2] and others, it probably can be only because of crowding, insufficient nourishment and bad hygiene. Vercoe[3] has found no deterioration in the average child with succeeding pregnancies, even in very large families.

The influence of *artificial feeding* on mortality in the 1st year is very striking. Of 34,325 infants under 1 year dying in Berlin, 17.7 per cent. were wholly or partially breast-fed, and 82.3 per cent. entirely artificially fed (Eröss). Undoubtedly the feeding was in most instances improperly managed. The statistics for Münich, as published by Frank[4] gave very similar results: 85.22 per cent. of 8329 deaths under 1 year of age being in artificially fed infants, with but 14.78 per cent. among those breast-fed. Studies upon this subject by Woodbury[5] show that among 22,422 live-born infants in 8 cities in the United States the mortality among the artificially-fed infants was from 3 to 4 times as high as among the breast-fed.

The influence of *season* upon the mortality is decided. Both the coldest and hottest seasons of the year are attended by high mortality, especially in the 1st year of life. In the United States the mortality under the age of 5 years is very much the greatest in the hot summer months.[6] The increase of infantile mortality during the hot weather is not only dependent upon the deleterious changes which more readily take place in the milk at this season, but to a considerable extent upon the direct influence of the torrid weather upon the infant. This has been carefully demonstrated by Rietschel,[7] Schereschewsky[8] and others. The plotted curve shown in the figure (Fig. 31), after Rietschel, is of the infantile mortality from digestive diseases in Münich compared with the air-temperature in centigrade. A partial exception to the fatal effects of hot summer-weather exists in the 1st month of life, greatly debilitated children of this age being often favored by hot weather.

Sex exerts some influence. In 12 states of Europe 55.59 per cent. of the deaths in the 1st year were among males and 44.41 per cent. among females (Eröss). In the 6-years' record of the Registration Area of the

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[1] Centralbl. f. Kinderheilk., 1903, VIII, 269.
[2] Nederlandsch. Tijdschr. v. Geneesk., 1920, LXIV, 371.
[3] Lancet, 1922, I, 758.
[4] Ref. Jacobi in Gerhardt'sHandb. der Kinderkrankh., I, 319.
[5] Amer. Jour. Hyg., 1922, X I, 668.
[6] XI Rep., U. S. Census, 1890, II, 63.
[7] Ergebnisse der inn. Med. u. Kinderheilk., 1910, VI, 369.
[8] Arch. of Pediat., 1913, XXX, 916.

United States[1] 54.83 per cent. of the deaths under 1 year were among males and 45.17 per cent. among females. As, however, there are about 95 females borne for every 100 males, the death-rate of females as compared with the males born is a trifle greater than the figures express.

It is difficult to determine the influence of *race* apart from attendant climatic, hygienic, and similar conditions. In the United States the mortality of the white races in the 1st year is greatest in the Italians, Bohemians, and Hungarians. Among the dark races of the United States (Negroes, Chinese, and Indians), the mortality in the 1st year exceeds greatly that of the white infants. Knox[2] gives some interesting figures for the State of Maryland. Out of every 1000 children of each race born alive there survived at 1 year 910 white and 838 Negro children; at 5 years 862 white and 731 negroes. At 40 years 733 of the white individuals were alive and only 505 of the negroes.

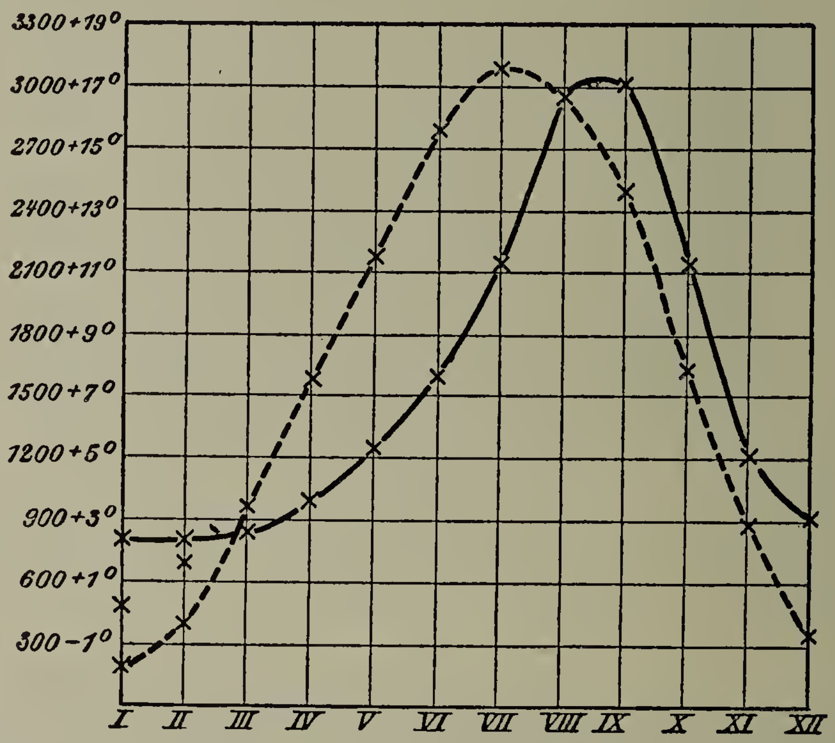

FIG. 31.—DEATHS FROM DIGESTIVE DISORDERS IN INFANTS IN MUNICH FROM 1895 TO 1904. The interrupted line indicates the mean air-temperature; the other the number of fatal cases for each month. It is to be noted that the height of the death-curve occurs about 4 weeks later than that of the temperature-curve. (*Rietschel, Ergebn. d. inner. Med. u. Kinderh.*, 1910, *VI*, 416. *Based upon statistics of Fürst, Vierteljahrsschr. f. öffentl. Gesundheitsflege*, 1907, *XXXIX*, 417.)

Diminution in the Death-rate.—There is every reason to believe that a decided lessening of the mortality-rate has occurred in recent years, particularly among infants; and this applies even to infant-homes and similar institutions where previously the mortality had been so high. The diminution is dependent in large part upon improved methods of hygiene and diet and increased knowledge of infantile diseases, especially of the digestive apparatus. Rich[3] in a comparison of the statistics of the United States Census of 1900 with that of 10 years earlier, pointed out that the infantile death-rate in the Registration-Area had decreased from 20.58 per cent. per 100 births in 1890 to 16.54 per cent. in 1900; and that while the death-rate of children from 1 to 5 years of age in New York City in 1891 equalled 96 per 1000 individuals of this age, in 1896 it had fallen to 77.5 and in 1900 to 67 per 1000. Further, according to the statistics of Baker[1] the death-rate in infants in New York City, which was 289 per 1000 infants living in 1880, gradually but uninterruptedly fell to 134 in 1910. This history of a falling death-rate in infancy and early childhood is borne out by the experience of many other cities of the United States, the varying degree depending largely upon the amount of civic and professional care observed. In 1917 the infantile death-rate was 9.8 per cent. in the Birth-Registration Area, and by 1922 there had been a decrease to 7.9 per cent.[2] Improvement has taken place in other countries as well. Thus in Naples in 1915, according to de Capite,[3] the death-rate in the 1st year was 15.4 per cent., which fell in 1922 to 12.6 per cent. In Buenos Aires recent statistics give the mortality at this period as 8.66 per cent., (Eyzaguirre).[4] DePorte[5] after an extensive study of European and American statistics concluded that in the 6-yr. period from 1916 to 1921, there had been a definite decline of infantile mortality in all racial groups. On the other hand, among the native Mohammedans in Tunis, where hygiene and treatment in general are very imperfect, Dinguizli[6] in 1923 reported the mortality under 1 year of age as 24 per cent.

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[1] *Loc. cit.*, IV, 666.
[2] Arch. of Pediat., 1925, XLII, 243.
[3] Arch. of Pediat., 1905, XXII, 762.

Causes of Death at Different Ages.—Of cases dying in the 1st month of life prenatal causes of various sorts account for probably from 60 to 75 per cent. (Mitchell).[7] Death incident to birth and occurring shortly after it is very frequent, and perhaps the majority of instances of this depend upon gross intracranial trauma (Crothers;[8] Schwartz[9]). In 200 autopsies reported by Wolcott[10] intracranial hemorrhage was found in from 43 to 50 per cent. Among the most frequent causes of death in the 1*st year* various conditions appearing to indicate an inborn state of debility occupy one of the most prominent places. Of the 174,614 deaths in this period occurring in Ireland during 10 years "Debility, Atrophy, Inanition" headed the list with 29,136 deaths, or 16.69 per cent. "Convulsions" came next with a percentage of 11.89 (Langford Symes).[11] The latter, however, and to some extent the former, represent terminal diseases or methods of dying rather than the real cause of death. The mortality-statistics of Massachusetts during 5 years (1892–1896) made diarrheal diseases responsible for 29.49 per cent. of the total deaths occurring in the 1st year of life (S. W. Abbott),[12] and respiratory diseases, classified as "pneumonia" and "bronchitis," for 11.97 per cent. The deaths attributed to "atrophy" and "debility," many of which were probably the results of gastrointestinal disease, equalled 10 per cent. The statistics of the Registration Area of the United States for 1913, as analyzed by Woodward[13] made diseases of the digestive system responsible for deaths in the 1st year of life in 27.12

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[1] 15th Intern. Cong. on Hygiene and Demography, 1912, III, 3, pt. 1, 139.

[2] Statistical Report of Infant Mortality for 1921 in 573 cities of the U. S. Same for 1922 in 635 cities. Published by the Amer. Child-Health Assoc.

[3] La Pediat., 1922, XXX, 990.

[4] Anal. de la Facultad. de Med., 1921, IV, 175. Ref., Jour. Amer. Med. Assoc., 1921, LXXVII, 207.

[5] Amer. Jour. Hyg., 1925, V, 454.

[6] Bull. de l'acad. de méd., 1923, XC, 613.

[7] *Loc. cit.*, 152.

[8] Arch. of Pediat., 1923, XL, 266.

[9] Münch. med. Wochenschr., 1922, LXIX, 1110.

[10] Amer. Jour. Dis. Child., 1921, XXI, 488.

[11] Dublin Jour. Med. Sci., 1898, CV, 479.

[12] Jour. Mass. Assoc., Boards of Health, 1898, Dec.

[13] Amer. Jour. Obstet., 1916, LXXIII, 362.

per cent. and diseases of the respiratory system in 15.85 per cent. Of the latter 96 per cent. depended upon bronchitis or pneumonia.

The mortality from digestive diseases is especially large in cities. 38.5 per cent. of the deaths under 1 year occurring in the civic population of France were due to gastroenteritis (Budin).[1]

Of the chronic infectious disorders syphilis is more fatal in the 1st year than at any other time, about ⅓ of all the deaths from this affection occurring in this year. Tuberculosis is as common a cause in the 1st year as later.

The number of deaths from the acute infectious fevers is generally less than later in infancy and childhood owing to the much smaller incidence. To this, however, pertussis and erysipelas form exceptions, both of them causing more deaths in the 1st than in any other year. The mortality from measles is also high at that period. The cases of the other infectious fevers, although much fewer, have a relatively unfavorable prognosis.

In the 2d *year* the mortality is much less. The Massachusetts' statistics (S. W. Abbott)[2] for 5 years gave about ⅕ as many deaths for the 2d year as for the 1st year of life. Gastrointestinal diseases and respiratory affections are the most prominent causes, the latter gradually growing in importance and the former lessening. The deaths from gastrointestinal affections equalled 19.79 per cent. of the total deaths between 1 and 2 years of age, and respiratory diseases (pneumonia and bronchitis) 21.27 per cent.

Infectious fevers, particularly scarlet fever and diphtheria, play a more important part in this year, rubeola being about as frequent a cause of death as before. The deaths from tuberculosis in various forms are numerous, especially from general tuberculosis and tuberculous meningitis. Convulsions still remain a very prominent cause, or, rather, method, of death.

In *early childhood*, from the age of 2 to that of 5 or 6 years, deaths are chiefly due to the infectious fevers. According to the XI Census Report of the United States[3] the number of cases dying of scarlet fever from the age of 1 to 5 years, as compared with the total deaths at all ages from this disease, equalled 66.87 per cent. The corresponding percentage for measles was 64.36 per cent., for diphtheria and croup 64.09 per cent. and for pertussis 43.31 per cent. By far the largest actual number of deaths from these diseases was due, however, to diphtheria and "croup," the latter being without doubt generally diphtheritic.

Viewing infancy and early childhood together it is noticed that the mortality for different diseases reaches its maximum at different *seasons* of the year. A general average irrespective of the nature of the disease shows the proportion of deaths in children to be greatest in the cool season of the year (Uffelmann).[4] In some regions, however, the mortality is much higher in summer. This is very decided, for instance, in the United States[5] for children under 5 years of age. The nature of the disease, however, has an important influence. Thus in the Metropolitan District of the United States during the 6-year record there were 16,413 deaths from diarrheal diseases in July against only 553 in December, while from respiratory diseases there were but 1921 deaths in August against 5102 in January. *Sex* continues to exert a slight influence after the 1st year to the end of early childhood, rather more males than females dying.

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[1] Ann. méd. et de chir. inf., 1903, VII, 181.
[2] *Loc. cit.*
[3] 1890, IV., 666.
[4] Handb. d. Hyg. des Kindes, 1881, 85.
[5] U. S. Census, 1890, II, 63 and I, 195.

In *later childhood; i.e.* from the age of 5 or 6 years to that of 10 or 12 years the mortality is lower than at any other period of life. The principal causes of death at this time are the acute infectious diseases, especially diphtheria and scarlet fever. Respiratory disorders also constitute a very common cause; diarrheal diseases much less often so. Tuberculosis is still a frequent factor, showing itself often as tuberculous infection of the bones, lungs, and peritoneum; as well as of the meninges and in the form of general tuberculosis as before. Diseases of the heart now become a more prominent cause than before. The relative importance of certain of the maladies affecting early life, in their influence upon the death-rate, is well shown in the accompanying table given by Veeder[1] (Fig. 32) based upon 930,067 deaths in subjects under 10 years of age.

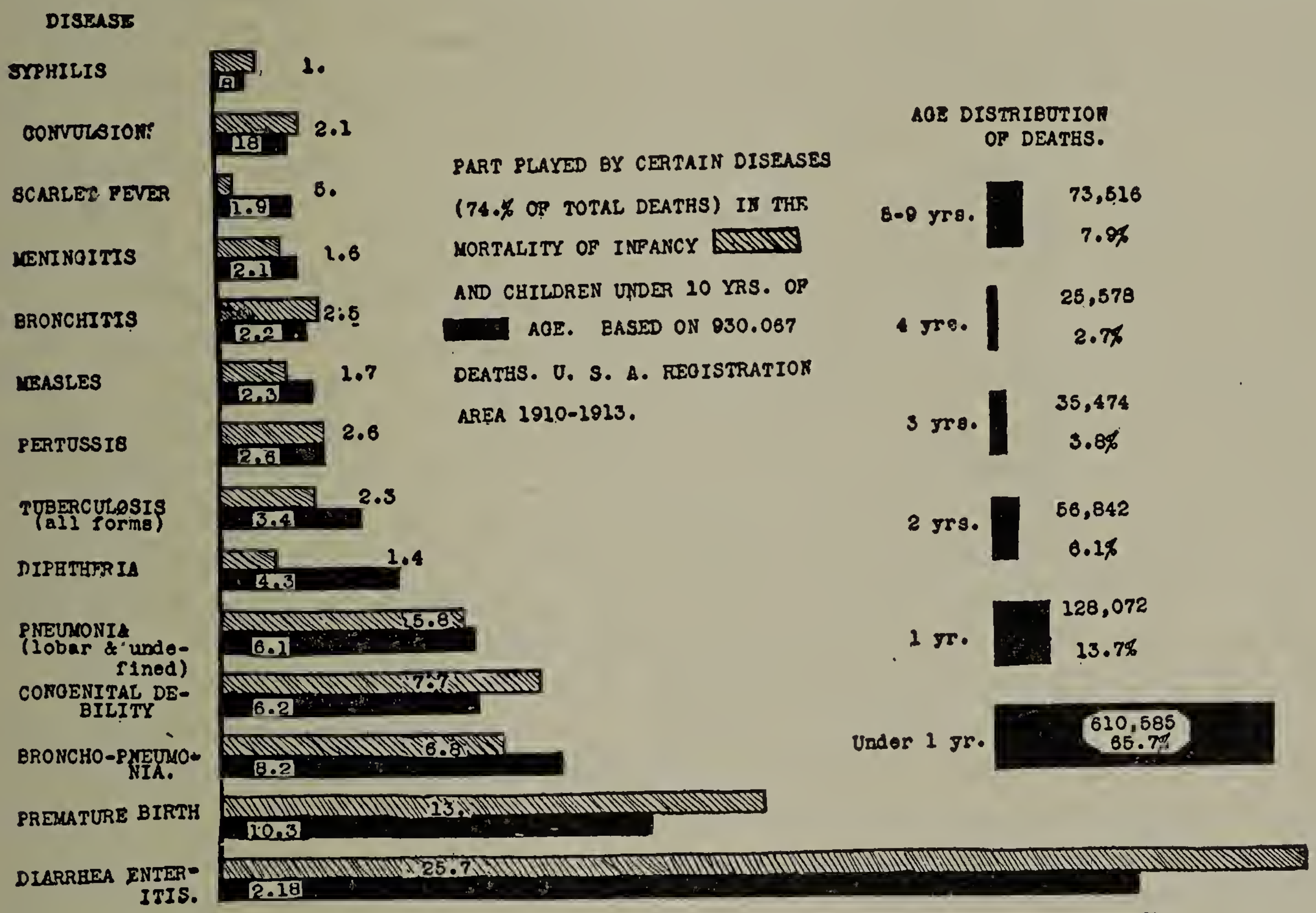

FIG. 32.—RELATIONSHIP OF CERTAIN DISEASES TO THE MORTALITY OF INFANTS AND CHILDREN.

(*Veeder, Arch. of Pediat.*, 1917, *XXXIV*, 322.)

Sudden Death.—Death occurring suddenly and unexpectedly is not infrequent, especially in the first 2 years of life, but the cause is often very obscure. In some instances the child has appeared in perfect health; in others the death occurred during some debilitating disease, yet without any discoverable reason for the sudden fatal ending. Sometimes there had been some easily overlooked symptoms, which would probably have made the diagnosis possible and accounted for the death had a physician been in attendance. In some cases an autopsy reveals the cause, but in many it does not. The feeble resisting power of infancy and the great excitability of the nervous system are the principal reasons

[1] Arch. of Pediat., 1917, XXXIV, 322.

for predisposition to sudden death at this age. The various causes have been reviewed by Thiemich,[1] Vipond,[2] and others.[3]

Conditions affecting the respiration are generally considered as holding the most prominent etiological place. Coryza in the new-born may rarely be the cause through what has been described as "aspiration of the tongue" (Bouchut).[4] By the violent efforts at breathing through the mouth the tongue is drawn backward and its under surface and tip become pressed against the hard palate, cutting off more or less the entrance of air. Death in the same way is stated occasionally to occur in young infants with pertussis. Asphyxia from over-lying probably does not happen as frequently as once supposed. Although a considerable number of infants die in this way, most of the cases assigned to this category probably belong elsewhere. Enlargement of the uvula has been designated a cause of fatal closure of the glottis, but the occurrence of the accident seems doubtful. Spasm of the glottis is perhaps one of the most frequent apparent causes; certainly so in those infants who had seemed to be in perfect health. This laryngo-spasm depends on the very great irritability of the nervous system which, although oftenest seen in debilitated, and especially rachitic children, exists sometimes quite independently of these conditions and is a symptom of spasmophilia (Vol. II, pp. 526, 537) and probably closely associated with lymphatism. It is likely that many cases of death attributed to other causes in reality depend upon this state. Thus it seems likely that the sudden death assigned to enlarged thymus gland is not in fact connected with this, but with the lymphatism of which the thymus enlargement is but a symptom. (See Diseases of the Thymus Gland, Vol. II, p. 839; Lymphatic Diathesis, Vol. I, p. 747.) Whether or not the death is actually due to laryngo-spasm or depends upon sudden cardiac failure is still an open question. Perhaps each factor may be the cause in different cases.

Central respiratory involvement, or what seems probably such, sometimes occurs in cases presumably with the neurotic tendency which might in other instances lead to laryngeal spasm. In these cases the child may die as though from sudden heart-failure. In other instances respiration becomes more and more rapid or difficult without other symptoms or discoverable reason, and the infant dies in a few hours. I have seen this accident occasionally. The cause of the condition is entirely obscure. A rapidly developing bronchopneumonia, especially in the new-born, may sometimes kill with apparent suddenness, the attendants having noticed no symptoms.

Sudden death in atrophic infants is of comparatively frequent occurrence. Thus it often happens that an infant who has been ill for days or weeks with unchanged evidences of debility will unexpectedly be found dead in bed, without there having been any alteration in symptoms. This is common also in cases of premature birth, autopsy often showing no lesions whatever. Yet in many of these cases, the cause is found to have been atelectasis, to which very young infants in a debilitated state are greatly disposed. In many other instances of atelectasis, however, the process comes on much more gradually and with evident symptoms. In some cases the death in marantic states may be due to the depressing effect of chilling or to a fall of body-temperature without discoverable cause.

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[1] Vierteljahrssch. f. gerichtl. Med., 1901, XXI, 300.
[2] Montreal Med. Jour., 1901, XXX, 23.
[3] See also article by the Author, Amer. Med., 1903, V, 989
[4] Mal. des nouveau-nés, 1885, 279.

Death with hyperpyrexia is often very unexpected in children who have been perfectly well or only moderately ailing but a few hours before. This may be seen in heat-stroke in infants, in malignant eruptive fevers, acute sepsis, and, still more frequently, in pneumonia in early life. Rarely sudden fatal asphyxia may result from the rupture of a caseous bronchial gland or of a retropharyngeal abscess into the respiratory tract. In other cases it is due to pressure of intrathoracic growths, abscesses, or enlarged glands upon the pneumogastric nerve. The aspiration of food into the windpipe after vomiting has been assigned as a cause. It probably occurs much less often than has been supposed, and only in infants so weak that the ability to cough has almost disappeared. Instances of asphyxia from the entrance of ascarides into the larynx have been reported, but in most of these it is probable that the worms entered the respiratory tract after death. Edema of the glottis is also an occasional cause.

Sudden death from heart-failure is not infrequently seen in convalescence after diphtheria. It may also occur in debilitated states or in respiratory diseases, especially pleural effusion, following too sudden a movement, improper position, or excitement. The same sudden cessation of the heart's action may take place in acute nephritis on account of the strain of increased arterial tension. Sudden stopping of the heart may occur in chronic valvular disease or as a result of distant nervous influences. It is probable that the sudden death which has occasionally followed quick movement or excitement in apparently healthy children, such, for instance, as the tossing of the child into the air, or which has occurred after exploratory puncture of the pleura, the giving of a hypodermic injection, or the administration of an anæsthetic, has been in reality caused by inhibition of the heart's action in subjects with lymphatism. Many of the deaths assigned to spasm of the larynx are very possibly, as stated, the result of this sudden stopping of the heart.

Gastrointestinal affections seldom occasion sudden death except indirectly. A number of cases are reported where it appears to have been induced by the irritation produced by large numbers of ascarides in the intestine. This is probably, however, of very rare occurrence.

Convulsions are a very common cause of sudden death in children already ill or in those in whom no disease has been detected. Nervous reflex irritation from various regions of the body may be responsible for them. Congenital syphilis is a not infrequent cause of sudden death, even in infants who appear perfectly healthy (Fournier).[1] Various malformations and accidents not already mentioned may also produce it, among them perforation of the intestine, entrance of foreign bodies into the larynx or trachea, injuries from forceps, strangulated hernia, external hemorrhages from the stomach or bowels, and internal hemorrhages, especially in the new-born, into the suprarenal bodies and occasionally into the brain or elsewhere.

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[1] La sem. méd., 1901, XXI, 20.

CHAPTER XII

THE THERAPEUTICS OF INFANCY AND CHILDHOOD

Therapeutics in infancy and childhood possesses a few characteristics which sometimes render the subject easier and more satisfactory than in later years. All the tendencies of life are toward recovery, and the system generally responds well to remedial measures. Drugs when given should be in sufficient strength to do good, but never to do harm, and they should not be administered at all unless distinctly indicated. Under-dosing is futile; over-dosing harmful. To the latter there exists a wide-spread tendency among physicians. In the large majority of cases little medicine of any sort is required, and the careful attention to hygiene and diet is sufficient, with possibly such slight aiding of nature as the giving of a laxative, a diuretic, a warm bath, rest in bed, and the like.

Despite what has just been said the therapeutics of childhood for the most part presents many difficulties to the inexperienced. These depend chiefly on the fact that the child does not react toward remedial measures merely as a small-sized man would do, but has in many respects its own susceptibilities. Some methods of treatment influence the system more powerfully than in adult life; others, useful in adults, are not so in childhood. The difficulty in giving medicines to many children is another factor. Numerous children, as a result of whim, or of the unpleasant taste or smell of a medicine, refuse this altogether. The administration of it may do harm through the struggling which arises, or may be always followed by vomiting. The taste of the same medicine may be not at all disagreeable to another child.

The therapeutics of early life is thus conveniently divided into A, *Administration of medicines by the mouth.* B, *Treatment other than by drugs by the mouth.* The order of consideration by no means indicates the relation of their importance, but rather the reverse of this. It may be said in passing that the older method of measuring by apothecaries weights and measures will be chiefly referred to, not as preferable, but because it is at the present time much the most familiar, and because there seems to be no practical advantage in writing prescriptions in the metric system and ordering the administration of the medicine in drops or fluidrams. Metric equivalents are given in the parentheses whenever this seems best. A fluidram is equivalent to practically 4 c.c., a fluidounce to 30 c.c., a grain to 65.0 milligrams, and a dram (apothecary) by weight to nearly 4 grams. We are usually obliged in family-practice to regard a teaspoonful as equalling 1 fluidram and a tablespoonful as the equivalent of ½ a fluidounce; although the great variation in the size of teaspoons and tablespoons renders this inaccurate. A small glass graduate should always be recommended. Locke[1] gives the teaspoonful as equalling 5 c.c. and the tablespoonful as 15 c.c.

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[1] Food Values, 1916, 24.

A. ADMINISTRATION OF MEDICINES BY THE MOUTH

1. Method of Giving Medicine.—In this, as in all forms of medical treatment in early life, we must as far as possible avoid the occasioning of fright, pain, or great excitement of any sort. Previous good training by the parents will have rendered the administration of medicines much easier. The physician, on his part, must see first, that the doses are small; second, that the taste is as pleasant as it can be made; and third, and most important, that the giving of any drug is really necessary. There is no question that children are often much over-dosed. It is often impossible to conceal a disagreeable taste. In such an event force may become necessary in giving the dose. Whether this shall be employed, or whether the treatment by drugs shall be foregone, depends upon the needs of the case. Judgment must be exercised, remembering, as stated, that the harm from the fear and excitement may much exceed the good which the medicine might do. This scarcely applies to the cases of less severe ailments in vigorous children, when the refusal depends merely on ill-temper or obstinacy.

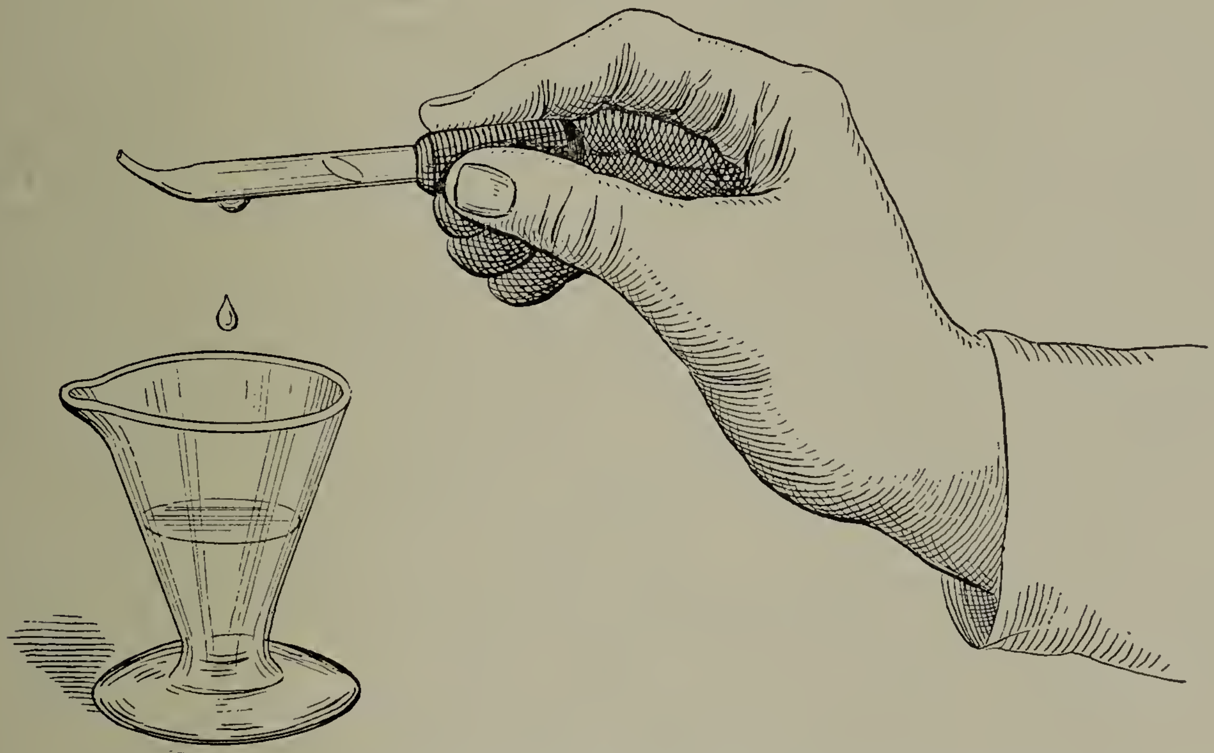

Fig. 33.—Medicine-Dropper.

Showing the correct method of dropping from the thicker portion of the tube.

Where a dose of medicine has to be administered by force, the child should be wrapped as for the examination of the throat (p. 207) and held by an attendant, and the nostrils compressed for a moment. Generally the mouth opens and the spoon may be inserted to the back of the tongue, emptied, and slowly withdrawn. There is difficulty in swallowing if the spoon is not removed from the mouth. In infants the pressing of the chin downward and backward may be sufficient to open the mouth. Often an infant who spits out the greater part of a teaspoonful of medicine will take it very nicely if it is given in divided portions. It may well be administered from a larger spoon, as there is less danger of spilling it should the child struggle.

The attendants should be told that, in cases where medicine must be given it is better to waste little time in pleading and in argument, but to use firmness and decision from the very beginning. The child must in no case be deceived regarding the character of the medicine, as its confidence is lost thereby, and the giving of the next dose will be only the harder. For the same reason a disagreeable medicine should never be mixed with the food without the previous knowledge of the child. In stuporous, delirious, or greatly exhausted conditions liquid medicine may be administered with a medicine dropper while the child is on its back. This should be inserted between the cheek and the teeth to prevent its being bitten. Nauseating remedies should not be given upon an empty stomach when this can be avoided.

In the ordering of quite small doses, or in the prescribing of powerful drugs, the physician must carefully remember the difference between the minim and the drop of many solutions, particularly of alcoholic ones, such as tinctures; and that the size of the drop also varies with the mouth of the bottle or with the dropper from which it is obtained. Consequently it is best to reckon the dose in minims rather than drops. Where the attendants are to measure the mixture a minim-glass should be used, or, if drops are ordered, the nurse should be told exactly the method of dropping to be employed. The small, sharply-pointed medicine-droppers furnish a drop scarcely more than half the size of one obtained from the edge of the bottle-mouth. A curved dropper should be used, and the drop should fall from the thicker portion (Fig. 33). It is important, too, to recognize the differences in the sizes of spoons, which make it safer to have the "teaspoonful" or "tablespoonful" prescribed measured as a dram or a half ounce in an accurately graduated medicine-glass. (See p. 246.) Mothers and nurses should be cautioned against attempting to measure half a teaspoonful with a teaspoon. The shape of the bowl of the spoon makes this impossible. To obtain half a teaspoonful, 1 teaspoonful of the medicine should be mixed with 1 teaspoonful of water, and the child given a teaspoonful of the mixture. Similarly a 5 minim glass may be made to measure 1 minim by adding to the 5 minims of the medicine 20 minims of water and taking 5 minims of the mixture.

Unless really necessary those medicaments should not be used which cause unpleasant secondary effects, sometimes worse than the disease itself. Those, too, are to be avoided, when possible, which are almost uniformly disliked by children; and the individual fancy of the patient should be studied as well. Much can be done to disguise unpleasant tastes to a certain extent. As a rule, infants and children like sweetened mixtures, and syrups, saccharin or glycerine may be used for sweetening. Among the serviceable syrups for this purpose especially for older children are syrup of ginger, syrup of raspberry (rubus idæus), syrup of chocolate, syrup of orange-flower, syrup of wild cherry, syrup of vanilla, and syrup of lemon. For infants the simple syrup of the Pharmacopoeia is to be preferred. The aromatic waters are well-liked, such as peppermint water, spearmint water, cinnamon water, and orange-flower water. For infants spearmint water (aqua menthæ viridis) is to be preferred to peppermint water (aqua menthæ piperitæ) since the sharpness of the latter is often unpleasant and occasions choking unless well-diluted. The elixir aromaticus and the elixir glycyrrhizæ (elixir adjuvans) are very serviceable for disguising an unpleasant taste in the case of older children. It must not be forgotten, however, that these contain about 25 per cent., of alcohol and are not suitable for employment in full strength in the case of young subjects. Bitter medicines are generally disliked by children, but infants often appear to find them not unpleasant, especially if an abundance of sweetening is added. In this point there is the greatest individual difference seen. In some instances the bitter taste appears to make the infant vomit promptly. Nearly all liquid medicines may be sweetened and diluted with water when given, if the child finds the dose as prescribed disagreeable. Instruction on this point should be included in the directions upon the label on the bottle. An exception exists where sugar may disagree with the digestion; in the case of oils or emulsions; or where the addition of water makes the larger bulk still more difficult of administration.

The taking of a sip of sweetened water, milk, sweetened lemon-juice or orange juice, or the sucking a mint-drop or chocolate immediately before medicine is taken dulls the sense of taste to some extent. Another sip immediately after aids in covering the unpleasant taste. The mother should be instructed that the employment of sweets in this way is to be confined to the occasions of giving medicine. Quinine may be disguised to a certain extent by licorice, or still better by syrup of yerba santa or syrup of chocolate. A nearly tasteless preparation such as quinine ethyl carbonate (aristochin) may replace it to a considerable extent. Castor oil should be given in emulsion with an aromatic water; floated on ice water, lemon juice, sarsaparilla or whiskey and water, or stirred in hot milk. By the latter method it is rendered nearly tasteless. The child must, of course, not be told that it is milk. In the case of infants it may be administered warmed in the spoon to make it less thick. Many infants always vomit it, and to these it should not be given at all. Others do not appear to dislike it in the least. There are some serviceable tasteless preparations on the market. To older children it is generally distasteful unless disguised. Cod liver oil, if disguised, is generally liked by children. It is best given as an emulsion flavored with mint, or combined with a syrupy malt-extract.

Drugs in powdered form, if comparatively tasteless and small in amount, may be placed directly upon the tongue, and washed down with a sip of water or milk. It is still better to have them combined with sugar when prepared. Powders of larger size may be mixed with a teaspoonful of jam, preserves, scraped apple, or the like, if there is no contra-indication. Entirely tasteless powders may conveniently be given on milk-toast or bread-and-milk and not be perceived at all.

Little children cannot take pills. Those of 3 or 4 years of age, if well trained, will swallow them readily if they are small. If the substance is bitter it should be sugar-coated, or the drug enclosed in capsules. The pill may be conveniently offered in a small portion of jam or of preserved or fresh fruit. The rule already expressed still holds good; that no deception is allowable, and that the child should always be told that it is "medicine."

The passage of drugs to the infant through the mother's milk has already been alluded to (p. 105). For practical purposes in infantile medication this need not be considered, although the method is sometimes useful for the treatment of the fetus, as in syphilis.

2. Dosage.—Various posometric tables have been constructed for use in childhood, and various methods proposed for the calculation of the proportionate doses as compared with those of adults. No fixed rule, however, can be entirely accurate. This is both because the relative susceptibility to different drugs varies greatly at this time of life, and because one cannot be guided either by age or by size alone. The rule of Clarke makes the weight of the child the numerator and 150 pounds the denominator, the resulting fraction being the portion of the adult dose to be employed. However, a child of 6 months weighing no more than a child of 1 month may need a decidedly larger dose than its weight would indicate. The rule of Young, which adds 12 to the age and divides the age by the result, ignores entirely the elements of size and weight. In practice, both age and weight must be considered. For the average child, Young's rule is a very convenient one down to the age of 2 years. Earlier than this the dosage may be based partly upon the age and partly upon the weight. The following table of proportionate dosage is the one I am in the habit of employing as a guide, but in a general way only.

Table 74.—Proportional Doses

Adult	1
18 years	3/4 +
12 years	1/2
8–10 years	2/5
6 years	1/3
4 years	1/4
3 years	1/5
2 years	1/7
1 year	1/10
9 months	1/15 or 2/3 dose for 1 year
6 months	1/20 or 1/2 dose for 1 year
Birth to 3 months	1/30 or 1/3 dose for 1 year

3. Effect of and Susceptibility to Certain Drugs.—Certain medicines are peculiarly well-tolerated and of much value in early life. Others are dangerous except in amounts smaller than the proportionate dose for the age would be. The dosage in general will be found in the table on p. 257. The following drugs require special mention.

Alcohol in the form of wines and spirits is tolerated in relatively large doses, and is exceedingly useful in many conditions. It is given far too frequently and freely by many physicians, and in absurdly small amounts by others. Judgment is, of course, required in deciding upon its employment and upon the dosage. It is indicated in many conditions where cardiac failure threatens, as in bronchopneumonia and in many cases of the infectious eruptive fevers, but especially in diphtheria. In exhaustion from any cause it is useful, as also in many cases of slight debility where a gently stimulating effect is sought. It should not be given in acute febrile conditions of short duration where the heart's action is strong and the temperature high. It is not needed in mild cases of typhoid fever or pneumonia.

Whiskey is the form oftenest serviceable. It contains about 40 to 45 per cent. by weight of alcohol. Brandy may be employed in equal dose. Port wine is excellent, and is well liked by many children. Its dose is somewhat over twice that of whiskey. Other wines are not so often of value in early life on account of their greater bulk. On occasions, however, champagne is useful for older children. It must be borne in mind that many of the liquid beef preparations on the market are quite strong in alcohol, although their nutritive value is slight, and that there is danger of giving them in excess. They are often excellent as stimulants, as their taste is pleasant. The dose may be determined by comparing their alcoholic strength (p. 180) with that of whiskey.

The amount of whiskey to be given varies with the demands of the case. An average dose every 3 or 4 hours up to the age of 3 months would be 5 to 10 minims (0.31 to 0.62). This may be readily increased during a short period in emergency. The dose at 1 year would be 10 to 20 minims (0.62 to 1.23).

Alcoholic stimulants should be given well-diluted. They may sometimes be mixed with the milk-food of the infant; but, if it is important to insure that the full amount be taken, it is better to give them with a little sweetened water from a spoon, or to add them to only so much of the food as is certain of being ingested.

In view of the extensive controversy which has taken place with regard to the employment of alcohol as a therapeutic agent, it would seem to be a fair statement to say that alcohol is a *drug*, which, like all other drugs of power, is capable of doing harm. That it has been greatly abused and has done much damage in the past through the too free administration of it by physicians is beyond question. That it further is not needed in many cases where it formerly was given unhesitatingly is equally true. That it is able to do much good in certain conditions seems so well supported by the clinical experience of centuries that it is difficult to controvert this by the apparent results of physiological experiments.

Alkalies are well borne and are of particular service in early life, on account of their power to neutralize gastric acidity and restrain vomiting, and for other reasons. Liquor calcis in teaspoonful doses may be given hourly if needed in early infancy, or bicarbonate of soda in doses of 1 to 2 grains (0.065 to 0.13) in the first months of life.

Alkaline salts, such as the citrate of potash and acetate of ammonia, are much employed as febrifuges. Their value for this purpose is somewhat uncertain. 1 to 2 grains (0.065 to 0.13) of the citrate or 15 minims (0.92) of the liquor potassii citratis may be administered every 3 hours at the age of 1 year. Very much larger doses are needed when it is required to render the urine distinctly alkaline.

Antimony is not borne at all well in infancy and early childhood. Its action is often very depressing, whether used as an emetic or as an expectorant. Since other less dangerous drugs readily replace it, it should not be employed. It is mentioned here only to condemn it.

Antipyretic drugs of the coal-tar series are in my experience well tolerated by children, but not often needed to reduce fever, external measures for this purpose being preferable. They are serviceable, however, when such measures are not effective or well borne or for other reasons cannot be employed. In this connection a warning must be uttered against the effort to reduce a child's temperature merely because it is elevated. The temperature rises from very slight causes in early life, and runs correspondingly higher than in adults. Even high fever, unless prolonged, is borne remarkably well in infancy and childhood. Unless it is approaching a dangerous hyperpyrexia, or is attended by threatening symptoms, acute elevation of temperature generally need cause no alarm. When, however, very prominent nervous symptoms attend the fever, such as decided delirium, great restlessness, and the evidence of impending convulsions, antipyresis of some sort is required. It is particularly in these conditions that the coal-tar antipyretics do good by their action upon the nerve-centers. Under such circumstances, and if hydrotherapy has been ineffectual, antipyrine, acetylsalicylic acid and phenacetin are to be depended upon, and are safe in proper doses. Acetanilid is more dangerous. To a child of 1 year ½ to 1 grain (0.032 to 0.065) of antipyrine or phenacetin may be given, and repeated in an hour if necessary. An amount should never be used which will produce a rapid and very decided fall of temperature with much sweating, since the danger from antipyretics lies in this critical fall.

The coal-tar antipyretics are often very serviceable in controlling nervous symptoms of any sort even in the absence of fever; such, for instance, as spasmodic affections, chorea, asthma, and especially pertussis. In the treatment of the last-mentioned antipyrine is one of the best of remedies. For the control of this, as for the management of many apyretic nervous disorders, the dose must be larger, and can safely be made so, since there is no danger of critical fall of temperature. An infant of 3 to 6 months may receive ¼ grain (0.016) of antipyrine every 3 hours for an initial dose, and one of 1 year 1 grain (0.065). In each case the dose may be increased two- or three-fold, if it is well borne and necessity demands it.

Apomorphia is too depressing a drug to be used in infancy, and even in childhood it may well be replaced by other expectorants.

Aromatic oils, generally in the form of aromatic waters, are particularly useful, well tolerated, and agreeable. Peppermint water is sometimes rather strong for the taste in the first few months of life, and spearmint, fennel (aqua fœniculi), or cinnamon water is then to be preferred.

Arsenic is borne well by children. Infants of a year may take ¼ minim (0.015) and even up to 2 minims (0.123) of Fowler's solution, well-diluted, 3 times a day, if found to agree. In later childhood the dose is often as large as or larger than that for adult life. Arsenic is used chiefly for chorea, syphilis, and in some forms of anemia.

Belladonna is borne exceptionally well in early life. One minim (0.062) of the tincture (equivalent to about 1/3000 grain (0.000021) of atropine), given 3 to 6 times in 24 hours, is usually well tolerated in the 1st year of life and the dose may need to be increased decidedly. During childhood the drug is often borne in larger doses than in adult life. Yet the individual susceptibility varies extremely, and very small amounts often flush children decidedly. The initial dose should, therefore, always be small, and the attendant informed of the possible result. The variation in the strength among different samples of the tincture renders a fresh solution of the alkaloid preferable. The drug is particularly useful in enuresis and in some forms of bronchopneumonia.

Bromides are tolerated unusually well, and are of great service in nervous conditions, especially in convulsions. They are useful also for insomnia and colic. A child of 2 years requires 3 to 5 grains (0.194 to 0.324) at a dose. An infant up to the age of 3 months takes ½ to 1 grain (0.032 to 0.065), but in serious conditions, such as convulsions, 4 or 5 grains (0.259 to 0.324) are required, either by the mouth or by enema.

A pustular eruption is sometimes soon produced by the use of the bromides. In my own experience this is rare, but the possibility of the occurrence must not be forgotten. (See Fig. 444, Vol. II, p. 874.)

Carbolic acid is only to be mentioned on account of the caution to be used in even its local employment in infancy. Cases of poisoning have repeatedly occurred, and I have seen severe injury to the skin produced by the local application of some of the proprietary preparations containing the drug. It is better to avoid it unless specially indicated.

Chloral is generally borne well by infants, but, as it is a powerful narcotic, its action must be watched carefully. It is a valuable remedy in severe colic, although it is occasionally irritating to the stomach. Its especial place is in the treatment of convulsions. The dose is ¼ to ½ grain (0.016 to 0.032) for milder ailments up to the age of 3 months.

For convulsions at this age 1 to 2 grains (0.065 to 0.13) may be given by the rectum, and repeated as required.

Cocaine is a dangerous remedy in early life, and can well be dispensed with. It is mentioned only to emphasize this fact.

Cod-liver oil constitutes one of the very best of the tonic remedies and is especially indicated in rickets. Children generally take and digest t well. From ¼ to ½ fluidram (0.92 to 1.85) in emulsion or in a syrupy malt-extract should be given to children in the 1st year, and double this after this age. It should not be administered where there is gastric ndigestion especially of fat, and occasionally it is not well tolerated when there is diarrhea. During very hot weather it is not always well borne, and it may be found necessary to intermit its administration at this time. Its use by inunction, while possibly occasionally of benefit, is unpleasant and generally unsatisfactory.

Digitalis is, in my experience, a useful drug in infancy and childhood, and generally well borne. In the first 6 months the dose of the tincture should be ¼ to 1 minim (0.015 to 0.062) or more repeated as needed. The effect upon the pulse must always be watched with care, and the drug stopped if irregularity is produced. (See Vol. II, p. 412.) The tendency of digitalis to produce vomiting is also to be remembered. The relative value of the newer preparations of digitalis is still under discussion. Digalen, digipuratum, digitalone, digitan, digitol, digitalin, and others are well spoken of. For the present my preference is still for the tincture, except for hypodermic administration.

Iron is a very useful tonic in many cases of anemia. It is not needed in other conditions with debility. To infants it should be given in a form as unirritating as possible, such as the lactate, citrate, malate, or pyrophosphate. For older children Blaud's pill (pilulæ ferri carbonatis) and Vallet's mass (massa ferri carbonatis) are excellent provided they are freshly made. Some of the newer organic compounds on the market are very serviceable, but appear no better than, if equal to, the older official preparations. The possibility of iron producing indigestion is not to be forgotten.

Ipecacuanha is generally remarkably well-tolerated. A child of 3 or 4 years, or even younger, can ordinarily take as much as, or more than, an adult. As an expectorant the syrup may be employed in doses of from 3 to 4 minims (0.185 to 0.246) in the first 3 months, and 6 to 8 minims (0.37 to 0.493) at 1 year, every 2 or 3 hours. Yet individual susceptibility exists, and the mother should be warned of the possibility of vomiting being produced. Ipecacuanha is one of the most serviceable and least depressing of emetics. A fluidram (3.7) of the syrup may be given at a dose, and repeated in 20 minutes if there has been no action.

Mercury in nearly all forms is well tolerated by children and seldom salivates. For its constitutional effect in infancy it is conveniently applied in the form of the ointment or the oleate smeared on the binder. Internally for the same purpose calomel may be given in doses of ¼ to ½ grain (0.016 to 0.032) or gray powder in doses of 1 grain (0.065) or even 2 grains (0.13) 3 times a day to infants under 3 months. The bichloride of mercury, formerly much used in diphtheria, was surprisingly well-tolerated. An amount equaling 1/32 grain (0.002) every 2 hours to a child of 2 years was a common dosage. As a rule, however, the milder mercurials are to be preferred. Bichloride of mercury used locally has produced toxic symptoms in infancy. The action of calomel as a laxative is referred to under Purgatives.

Nitroglycerine fills a valuable place in the therapeutics of infancy and childhood. It is tolerated in proportionately very large amounts. 1/500 to 1/200 grain (0.00013 to 0.0003) by the mouth or hypodermically may be given to a child of 6 months, repeated soon, and frequently in larger dose. It is often of great value where there is failure of the heart's force in various diseases, acting probably by the reduction of the arterial pressure.

Opiates.—In the first 2 or 3 months of life opiates must be given *with great caution,* and in very small initial dose until the susceptibility is ascertained. At any period of early life instructions should always be left with the attendants that the dose is to be lessened or the medicine stopped if the effect has been decided. In greatly debilitated conditions, and in impending coma at any period of infancy, opiates are liable to act with unusual power. Except under these circumstances, the susceptibility diminishes after the age of 3 months, although still present to a moderate extent. As the individual reaction varies greatly, after the initial tentative dose the amount can be increased until the desired effect is produced. Deodorized tincture, paregoric, Dover's powder, morphine, heroine and codeine are perhaps the most useful preparations. For infants we can well do without the stronger preparations except for hypodermic medication. The average *initial* dose, except in the extremely sensitive states mentioned, may be as follows: Up to the age of 3 months: Deodorized tincture 1/30 to 1/15 minim (0.002 to 0.004), camphorated tincture (paregoric) 1 minim (0.062), Dover's powder 1/30 to 1/20 grain (0.002 to 0.003), morphine and heroine 1/800 to 1/500 (0.00008 to 0.00013), codeine 1/300 to 1/200 grain (0.0002 to 0.0003). At 1 year the initial doses may be: Deodorized tincture 1/4 to 1/2 minim (0.015 to 0.031), camphorated tincture 4 to 10 minims (0.246 to 0.616), Dover's powder 1/8 to 1/2 grain (0.008 to 0.032), morphine and heroine 1/150 to 1/100 grain (0.0004 to 0.0006), codeine 1/100 to 1/50 grain (0.0006 to 0.0013). Nothing that has been said militates in any way against the proper employment of opiates when needed. At any period of life they constitute in many diseases one of the most valuable of remedies, and the physician who through unwarranted fear avoids their administration to infants or children is separating himself from one of the most useful articles of the pharmacopœia.

Purgatives are generally well borne by children, and in relatively larger doses than in later life. Calomel is one of those most frequently employed, although, in my opinion, not in general as serviceable as castor oil, and given too indiscriminately. The dose is proportionately much larger than for adults. Up to the age of 3 months 1/24 to 1/12 grain (0.0027 to 0.0054) may be given hourly until 1/2 grain (0.32) is taken unless an effect is produced sooner. After this age 1/10 to 1/8 grain (0.0065 to 0.008) may be given hourly until there is an effect upon the bowels or until 1 grain (0.065) is consumed.

Castor oil is a deservedly popular purgative in infancy and childhood, on account of the fact that the irritation produced is but slight. The dose is proportionately large. Up to 3 months of age 1/2 fluidram (1.85) may be given, and 1 fluidram (3.7) after this period. Should it tend to gripe, the addition of a small amount of deodorized tincture of opium or paregoric is serviceable. An old-time favorite and a useful mixture consists of equal parts of aromatic syrup of rhubarb and castor oil. Magnesia is also a serviceable purgative. Magnesium oxide (calcined magnesia) usually agrees well with the stomach, but is unpleasant to take. Children of a year may have from 5 to 20 grains (0.324 to 1.3) or more. Magma magnesiæ (milk of magnesia), which consists merely of a suspension of magnesium hydroxide in water, is almost a household remedy. Doses of from ½ to 1 fluidram (1.85 to 3.7) may be given in the bottle of milk-mixture in the case of infants artificially fed. Solution of citrate of magnesia is well tolerated and often liked by older children, particularly if it is not too acid in taste. 1 to 4 fluidrams (3.7 to 14.8) or more is the dose for an infant of 1 year; 2 to 4 fl. oz. (59 to 118) at the age of 2 to 4 years or older. The addition of orange juice makes it agreeable to those accustomed to this fruit. Senna in the form of the syrup, and rhubarb in that of the aromatic syrup, are each useful and of agreeable taste. Cascara in some of its more pleasant forms is very serviceable as a regulator of the bowels, alone or combined with phenolphthalein.

Pilocarpine is not at all well tolerated by infants, and should not be used. It is liable to produce dangerous depression.

Quinine is borne in exceptionally large amount. It may be given disguised by syrup of yerba santa or syrup of chocolate as already stated. Quinine chocolates also are often useful, although less trustworthy; and quinine ethyl carbamate (aristochin) is a nearly tasteless form and is often effective. The dose of sulphate of quinine for a child of 1 year may be ½ to 1 grain (0.032 to 0.065) 3 times a day. In cases of malaria larger amounts are generally needed. The drug may be given in the form of suppositories in double the dose mentioned, but the results are less satisfactory.

Salicylic acid and its compounds are well tolerated, except for their tendency to produce vomiting.

Strychnine is an extremely useful drug in early life. It may be given in relatively large doses, if there are no contra-indications. It is, however, in my opinion, often administered with little discrimination where other powerful stimulants are more serviceable. It is quite capable of producing or increasing in children a state of nervous excitability, and it should be avoided whenever such a condition or the possibility of the occurrence of a convulsion is present. The individual susceptibility varies greatly. At 1 year of age 1/150 to 1/100 grain (0.0004 to 0.0006) may be administered *daily* in divided doses, but sometimes larger amounts are required. Older children may well take it in the form of minute gelatin-covered pills. 1 minim (0.062) of tincture of nux vomica contains about 1/400 grain (0.00016) of the combined alkaloids, chiefly strychnine. The tincture forms one of the best of tonics in early life.

4. Drugs Grouped According to Their Action.—The following statements may be made in addition to and as a summary of what has just been said, grouping the drugs together according to their action.

(*a*) **Antipyretics.**—In cases of moderate fever the alkaline diuretics such as potassium citrate and ammonium acetate are very commonly administered, alone or combined with sweet spirits of nitre. The actual value of these drugs in reducing fever has been much disputed. In all more urgent cases, where external measures cannot be employed or are unavailing, the coal-tar derivatives are very useful, especially if nervous symptoms are present. There has been an unwarranted prejudice against them. Many of them are entirely safe if properly used.

(*b*) **Astringents.**—Bismuth subcarbonate is probably the best of these, alone or in combination with a tannic acid preparation, especially such as tannigen or tannalbin. Full doses should be given, as they are harmless. Opium should not be employed except when fever has gone and the intestinal peristalsis is too active, with a decided loss of liquid from the bowel; or in the dysenteric cases where there is excessive straining.

(*c*) **Purgatives.**—These are given under the same conditions as in adult life. Castor oil is perhaps the best in infancy, or milk of magnesia for less decided action. In older children citrate of magnesia is one of the best purgatives when taken readily. When a small dose is required, sulphate of magnesia, combined with rhubarb or senna, is efficacious; while cascara, phenolphthalein, and petrolatum liquidum are among the most serviceable regulators of the action of the bowels when a tendency to chronic constipation exists.

(*d*) **Sedatives.**—The bromides constitute one of the most useful and least harmful drugs of this class for conditions of moderate nervous excitability, sleeplessness and the like; and are frequently serviceable additions to preparations intended to relieve ordinary cough, pertussis, vesical irritability, colic, vomiting in infancy, and threatening convulsions. For the actual attack of convulsions full doses are required, and chloral had better be used as well. Chloral is serviceable, too, in cases of severe colic in infancy, and occasionally in very decided insomnia. Antipyrine and phenacetin are valuable in convulsive conditions of any sort, and in other nervous states; especially so when fever is also present, but often, too, during apyrexia. They may be employed with advantage to relieve insomnia and headache, and neuralgic pain in general if not too severe. Benzyl-benzoate has been highly recommended for various spasmodic conditions, among them pylorospasm, asthma, enuresis and colic. In our experience the drug has little value. Opium should be reserved for cases of severe pain of any nature, for the control of obstinate convulsions and to relieve harassing cough. It is of value in pneumonia when cough disturbs the rest too greatly, but is too be avoided when respiration is much interfered with, or when abdominal distention is present. It finds a useful place also as a sedative in diseases of the heart.

(*e*) **Stimulants.**—The indications and contra-indications have already been referred to (p. 250), and will be discussed later in considering the individual diseases in which they may be employed. When a rapid cardiac stimulant is required, as in threatened cardiac failure, caffeine, camphor and adrenaline given hypodermically are to be selected. Strychnine is much employed under much the same conditions, but is, I believe, of less value.

(*f*) **Tonics.**—Nux vomica is probably the best for use in childhood, especially in cases of loss of appetite and in persistent debility from any cause. It should be avoided when a condition of nervous excitability is present. Iron is useful only in cases of anemia. The action of quinine as a tonic is very variable. In some children it works well. Cod liver oil is one of the best of tonics for chronic states of debilitated health and poor nutrition, if the digestion is in good order.

5. **Approximate Average Dosage of Different Drugs.**—From what has been already stated the impossibility is manifest of constructing a table of doses for children which can be depended upon in more than a general way. Every medicine must be tried tentatively with every child needing it, and the amount to be given must vary not only with the susceptibility but with the necessity. Urgent cases need vigorous treatment. The following table of approximate dosage for a child of 1 year of age may, however, be of service as a partial guide. The doses in metric equivalents are enclosed in parentheses.

TABLE 75.—TABLE OF DOSAGE AT THE AGE OF 1 YEAR

Acetanilide		¼–½ gr. (0.016–0.032)
Aconite, tincture		⅛–¼ m. (0.008–0.016)
Adrenaline (1:1000 solution)		2–8 m. (0.123–0.493)
Alcohol	brandy	5–30 m. (0.31–1.86)
	champagne	1–3 dr. (3.7–11.1)
	gin	10–60 m. (0.62–3.7)
	port wine	10–60 m. (0.62–3.7)
	sherry	10–60 m. (0.62–3.7)
	whiskey	5–30 m. (0.31–1.85)
Ammonia	acetate, liquor	15 m. (0.92)
	aromatic spirits	2–5 m. (0.123–0.31)
	carbonate	½–1 gr. (0.032–0.065)
	chloride	1–2 gr. (0.065–0.13)
Antipyrine		¼–1 gr. (0.016–0.065)
Arsenic, Fowler's solution		¼–1 m. (0.015–0.062)
Asafetida, milk		15–60 m. (0.92–3.7)
Aspirin (acetyl salicylic acid)		¼–1 gr. (0.016–0,065)
Atropine		1/2000–1/1000 gr. (0.000032–0.000065)
Belladonna, tincture		½–2 m. (0.031–0.123)
Benzoic acid		½–1 gr. (0.032–0.065)
Bismuth	salicylate	1–2 gr. (0.065–0.13)
	subcarbonate	5–8 gr. (0.324–0.518)
	subgallate	2–4 gr. (0.13–0.259)
	subnitrate	5–8 gr. (0.324–0.518)
Brandy (See Alcohol)		
Bromides, sodium and potassium		1–4 gr. (0.065–0.259)
Bromoform		1–2 m. (0.062–0.123)
Caffeine citrate		¼–½ gr. (0.016–0.032)
Caffeine sodio-benzoate (hypodermically)		¼–½ gr. (0.016–0.032)
Calcium chloride		1–2 gr. (0.065–0.13)
Calomel (See Mercury)		
Camphor (hypodermically in oil)		⅕–½ gr. (0.013–0.032)
Cascara, fluid extract		1–4 m. (0.062–0.246)
Castor oil		30–60 m. (1.85–3.7)
Chalk-mixture		15–60 m. (0.92–3.7)
Champagne (See Alcohol)		
Chloral hydrate		½–2 gr. (0.032–0.13)
Chloroform, spirits		2 m. (0.123)
Cinchona, compound tincture		5 m. (0.31)
Cinnamon water		30–60 m. (1.85–3.7)
Codeine (See Opium)		
Cod liver oil		15–60 m. (0.92–3.7)
Corrosive sublimate (See Mercury)		
Digitalis, infusion		5–30 m. (0.31–1.85)
Digitalis, tincture		¼–2 m. (0.015–0.123)
Ergot, fluid extract		3–5 m. (0.185–0.31)
Fennel water		30–60 m. (1.85–3.7)
Gin (See Alcohol)		
Ginger, tincture		1–5 m. (0.062–0.31)
Heroin (See Opium)		
Hexamethylenamine		¼–½ gr. (0.016–0.032)
Hydrochloric acid, dilute		1–2 m. (0.062–0.123)
Iodine (See Potash)		
Ipecacuanha	powdered	⅛–¼ gr. (0.008–0.016)
	syrup	3–8 m. (0.185–0.493)
	syrup (as emetic)	60 m. (3.7)
Iron	citrate	¼–1 gr. (0.016–0.065)
	chloride, tincture	½–1 m. (0.031–0.062)
	iodide, syrup	1–5 m. (0.062–0.31)
	lactate	¼–1 gr. (0.016–0.065)
	pyrophosphate	¼–1 gr. (0.016–0.065)
	reduced	¼–½ gr. (0.016–0.032)
Kino, tincture		5 m. (0.31)
Laudanum (See Opium)		
Lime-water		½–2 dr. (1.85–7.4)
Liquorice-powder		2–10 gr. (0.13–0.65)

Table 75.—Table of Dosage at the Age of 1 Year (*Concluded*)

Magnesia	citrate, solution	1–4 dr. (3.7–14.8)
	milk	1–2 dr. (3.7–7.4)
	oxide	5–30 gr. (0.324–1.94)
	sulphate	3–10 gr. (0.194–0.65)
Manna		5–30 gr. (0.324–1.94)
Mercury	calomel	¼–1 gr. (0.016–0.065)
	calomel, divided doses (as purgative)	½–1 gr. (0.032–0.065)
	grey powder	1–2 gr. (0.065–0.13)
Morphine (See Opium)		
Nitre, sweet spirits of		2–6 m. (0.123–0.370)
Nux vomica, tincture		1–2 m. (0.062–0.123)
Opium	codeine	1/100–1/50 gr. (0.0006–0.0013)
	deodorized tincture	¼–½ m. (0.015–0.031)
	Dover's powder	⅛–½ gr. (0.008–0.032)
	heroine	1/150–1/100 gr. (0.0004–0.0006)
	laudanum	⅛–½ m. (0.008–0.031)
	morphine, sulphate	1/150–1/100 gr. (0.0004–0.0006)
	paregoric (camphorated tincture)	3–10 m. (0.185–0.62)
Pancreatin		1–3 gr. (0.065–0.194)
Peppermint-water		30–60 m. (1.85–3.7)
Pepsin		½–2 gr. (0.032–0.13)
Phenacetin (acetphenetidin)		¼–1 gr. (0.016–0.065)
Phenolphthalein		½–1 gr. (0.032–0.065)
Phosphorus		1/500 gr. (0.00013)
Potassium	citrate	1–2 gr. (0.065–0.13)
	chlorate	1 gr. (0.065)
	iodide	¼–1 gr. (0.016–0.065)
Quinine, sulphate		½–1 gr. (0.032–0.065)
Rhubarb, aromatic syrup		15–30 m. (0.92–1.85)
Salicylic acid (See Soda)		
Salol (phenol salicylate)		½–1 gr. (0.032–0.065)
Salophene		½–1 gr. (0.032–0.065)
Santonin		¼–½ gr. (0.016–0.032)
Senna		¼–4 gr. (0.016–0.259)
Senna, syrup		15–30 m. (0.92–1.85)
Silver, nitrate		1/120–1/60 gr. (0.0005–0.001)
Sodium	bicarbonate	1–2 gr. (0.065–0.13)
	phosphate	5–20 gr. (0.324–1.3)
	salicylate	½–2 gr. (0.032–0.13)
	sulphate	3–10 (0.194–0.65)
Spearmint-water		30–60 m. (1.85–3.7)
Squills, syrup		3–10 m. (0.185–0.62)
Strophanthus, tincture		¼–2 m. (0.015–0.123)
Strychnine, sulphate		1/500–1/200 gr. (0.00013–0.0003)
Sulphuric acid, aromatic		½–4 m. (0.031–0.246)
Tannic acid		¼–4 gr. (0.016–0.259)
Tannigen		1–2 gr. (0.065–0.13)
Tannalbin		1–2 gr. (0.065–0.13)
Terpene hydrate		¼–½ gr. (0.016–0.032)
Veronal (barbital)		1–3 gr. (0.065–0.194)
Whiskey (See Alcohol)		

B. TREATMENT OTHER THAN BY DRUGS BY THE MOUTH

1. Hypodermic Medication by Drugs.—This holds a minor place in the therapeutics of infancy and childhood on account of the pain and fright which attends the proceeding. It is, however, too much neglected, since under many circumstances it is invaluable; as when a rapid absorption of a medicine is required, where the rebellion against swallowing medicine is excessive, or where it is important that the stomach be spared as much as possible. In cardiac failure medication is far better given in this way, using camphor (1:10 in olive oil), caffeine sodiobenzoate, preparations of digitalis, strychnine, nitroglycerine and adrenaline. In respiratory failure hypodermic injections of atropine are largely employed. Morphine hypodermically may arrest convulsions or exhausting diarrhea. As in the case of adults a hypodermic dose should be, as a rule, considerably less than that given by the mouth.

2. Hypodermoclysis.—The injection of large amounts of sterilized normal saline solution (0.9 per cent.) under the skin, is invaluable in some states of exhaustion or where there has been great loss of fluid from the tissues as a result of diarrhea, vomiting or hemorrhage. Distilled water should be employed in making the solution. Solutions of glucose (5 per cent.) are sometimes to be preferred because they cause no retention of chlorides, and because, too, they possess a nutritive and diuretic value. The fluid to be injected is contained in a glass funnel or in a bottle or other vessel with an opening at the bottom (Fig. 34). To this a rubber tube is attached with a hypodermic needle of fairly large calibre on the other end. All should be carefully sterilized before using. The needle may be introduced into any region with abundant loose areolar tissue, as in the flank, below the scapulæ, or the lateral parts of the abdomen. The fluid should be warmed before use and either only a small amount poured into the vessel at a time, or the heat maintained by the application of warm cloths or hot water bags about the vessel (Fig. 36) since from ½ to 1 hour may be required to allow it to enter gradually beneath the skin. From 30 to 250 c.c. (1 to 8.5 fl. oz.) in the early weeks of life may be allowed to flow by gravity, the vessel being suspended about 2 feet (61 cm.) above the patient, and the amount given varying with the weight of the infant. The injection may be made once daily, or oftener

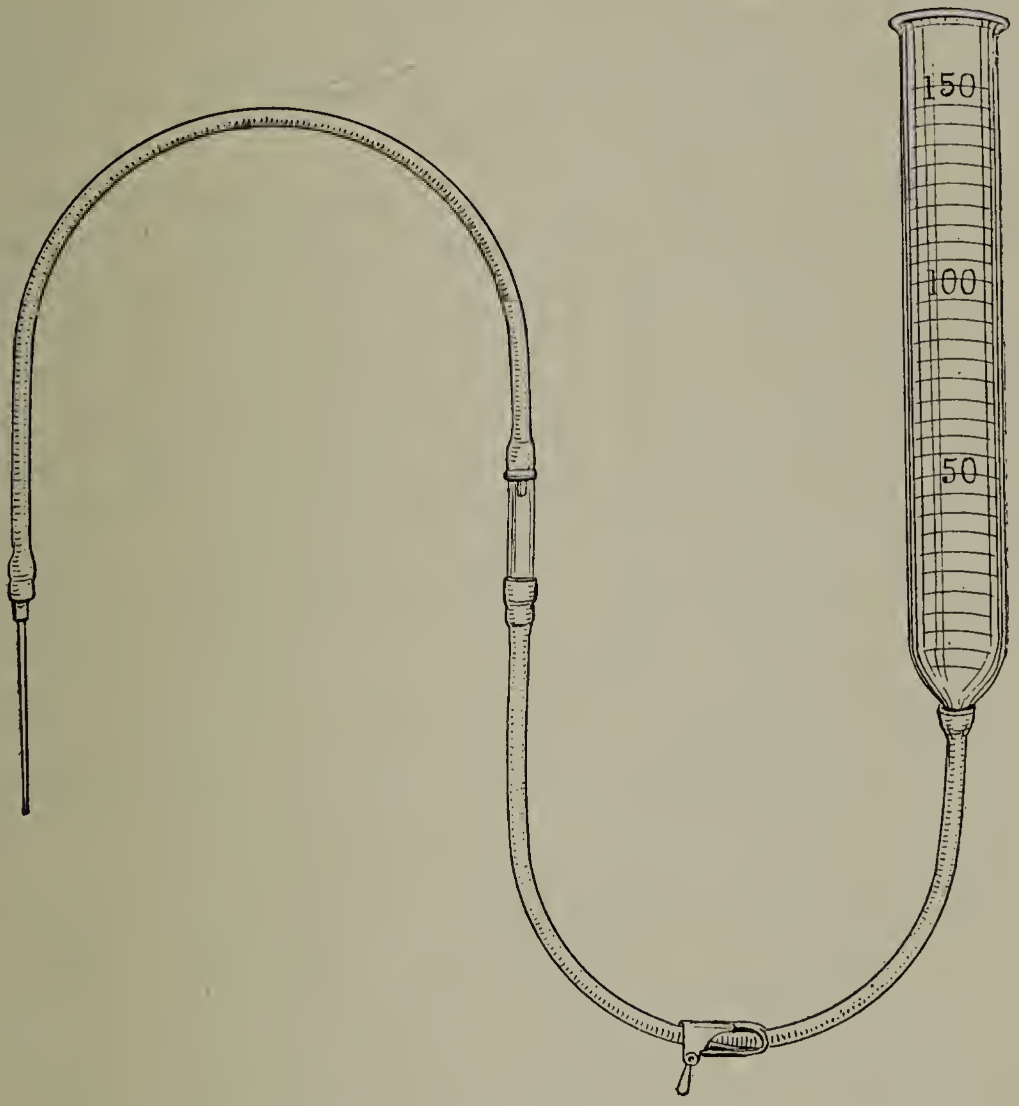

Fig. 34.—Apparatus for Hypodermoclysis.

The receptacle may be covered with hot cloths if necessary to maintain the heat.

if need be. Solutions of gelatine have been given in a similar manner in severe cases of hemorrhage. It is necessary, however, to obtain absolute sterilization of the fluid, or severe subcutaneous suppuration may occur. A simple boiling is not sufficient. It should be prepared in an autoclave. A solution of bicarbonate of soda may be given by hypodermoclysis in cases of severe acidosis. Inasmuch as it is impossible to sterilize this without transforming the bicarbonate into the irritating carbonate, the solution may be prepared as follows: Bring a litre (33⅓ fl. oz.) of distilled water to the boiling point. Remove from the flame. Add immediately 30 grains (1.9) of sodium bicarbonate (C. P.) taken directly from the center of the original container and weighed in a sterilized vessel. Cool the solution to a temperature of 100°F. (43.3°C.) and use what is required at once. Even when prepared in this way there is no certainty, however, that it will not prove very irritating, and it is better to administer the solution intravenously. (See p. 275.)

3. Intraperitoneal Injections.—As emphasized by Blackfan and Maxcy,[1] saline solutions may often be advantageously injected into the peritoneal cavity, and this procedure is widely employed in infancy. A large amount of fluid may be given in this manner; it is all retained and quickly absorbed; the injections may be frequently repeated,—as often as every 8 hours if necessary; there is little discomfort to the patient. Substances other than saline solution, such as 2–3 per cent. sodium bicarbonate (Epstein)[2] or 6 per cent. glucose (McLean and Lang)[3] have been given into the peritoneal cavity. A certain amount of reaction (sterile inflammation) in the peritoneum occurs even with saline solution (Denzer and Anderson)[4] and this is increased when more irritating solutions are employed. Consequently injections of soda or of glucose may better be administered intravenously. Intraperitoneal injections of diphtheria-antitoxin have been recommended (Platon).[5] Neoarsphenamin has been injected by Rosenberg[6] into the peritoneal cavity of the experiental animal, and in 1 human case with good results. Citrated or defibrinated blood may be administered in the same way (see p. 277).

The technic of intraperitoneal injection is as follows: After first ascertaining that the bladder is empty and that there is no very decided abdominal distention, the abdomen is painted with tincture of iodine. Physiological sodium chloride solution at a temperature of 100° is now put into a sterile glass container which is graduated. The bottom of this graduate is connected by a rubber tube with a needle of 18 gauge having a point beveled at 45 degrees. The skin of the abdomen in the midline, about ⅓ of the way from the umbilicus to the pubis, is elevated between the thumb and forefinger of the left hand. The right hand plunges the needle, with the solution flowing from it, through the abdominal wall. The needle should be pointed in an upward direction and should be inserted just through the abdominal wall, the thickness of which will differ with the individual case. An elevation of the glass container of 2 or 3 feet (61 or 91 cm.) above the abdomen will be sufficient to cause the proper rapidity of flow of the solution. When slight abdominal distention occurs the needle is withdrawn and a sterile dressing applied. The left border of the rectus may be used as the point of injection if so desired.

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[1] Amer. Jour. Dis. Child., 1918, XV, 19.

[2] Arch. of Pediat., 1920, XXXVII, 656.

[3] Amer. Jour. Dis. Child., 1920, XIX, 359.

[4] Amer. Jour. Dis. Child., 1921, XXI, 565.

[5] Arch. of Pediat., 1923, XL, 575.

[6] Jour. Amer. Med. Assoc., 1924, LXXXII, 682.

4. Suppositories and Enemata.—These constitute a useful method of giving medicines. Quinine may be conveniently administered by suppository, using at least double the dose by the mouth. Suppositories of gluten or of glycerine of small size are serviceable for the relief of constipation, as is often the simple soap-suppository or "soap-stick." Chloral and bromide of potash are to be administered by enema in cases of convulsions, and the salicylates may also be employed in this way in rheumatic conditions. Somewhat larger doses of the drugs are necessary than when given by mouth, but the size of the injection should not be large or it will not be retained. From 1 to 3 oz. (30 to 89) is sufficient, a convenient and bland vehicle being mucilage of acacia or thick starch-water. Sulphate of magnesia in a dosage of 2 ounces (56.70) in 6 to 10 ounces (177.4 to 295.8) of water may be administered by rectum for its dehydrating effect on the tissues of the body, including the brain and the subarachnoid spaces (See also Intravenous Injections, p. 274). Fischer[1] has for several years employed hypertonic salt-solution in this way. The enema is conveniently given by the infant-syringe, the fluid being warmed slightly and injected slowly. After the injection the nates must be kept pressed together, or the nurse's thumb held over the anus for a considerable time, to prevent the expulsion of the fluid. Astringent enemata were formerly much employed, as of tannic acid and of nitrate of silver. They must always be weak, especially the latter, or irritation and straining are produced. About ⅛ grain (0.008) of the silver salt or 1 to 2 grains (0.065 to 0.13) of tannic acid to 1 ounce (30) of water are sufficient, and even this strength is not always well tolerated. The injection of nitrate of silver should be followed by that of a solution of common salt. Suspensions of bismuth are frequently serviceable in cases of irritation of the lower intestinal mucous membrane, using 1 dram (3.9) of the subcarbonate to 4 or 5 oz. (118 or 148) of mucilage of acacia. When medicated enemata are to be employed the bowel should first be unloaded by an ordinary enema. For this purpose, or whenever local treatment of constipation is desired, a small injection of 1 fluidram (3.7) of glycerine, undiluted or mixed with from 1 to 2 oz. (30 to 59) of water often suffices; or a larger one of normal salt-solution or of soapy water may be given, allowing the child to receive as much as it can comfortably hold. When there are hardened fecal masses present an enema of from 1 to 2 oz. (30 to 59) or more of warm cotton-seed oil is very serviceable. This should be retained for some hours, or perhaps over night, and then followed by an injection of soapy water. Nutrient enemata are of very little service in infants, as they are seldom retained if given frequently enough to be of any possible service. They are sometimes of value in older children. The administration of a 5 per cent. solution of glucose in this way has become popular, since it can be absorbed from the large intestine. Arsenical preparations, such as neosalvarsan and arsenobenzol, may be given by rectal injection, using about 1½ times the intravenous dose of the drugs. The reports on the efficacy of this method of treatment of syphilis are conflicting. Arsenic is certainly absorbed from the lower bowel, as it has been found in the urine when given in this manner (Modigliani and Castana)[2] but the effect on clinical symptoms is variable.

5. Irrigation of the Intestine.—In some diseased conditions thorough irrigation of the entire colon is serviceable. The child should be laid on its back on a bed or table, with its hips slightly elevated and its thighs flexed on its abdomen. The bed should be protected by a large rubber sheet, so arranged that it will carry off the water into a receptacle beneath. The injection should be given from a fountain-syringe or glass funnel and tube at an elevation of about 2 feet (61 cm.) above the child. A hard-rubber nozzle may be used, a small roller-bandage being wrapped about this to act as a plug when pressed against the anus (Fig. 35). A nozzle of this sort allows of the firm pressing of the nates about it, in order to prevent the expelling of the fluid. There is no advantage offered by the employment of the so-called "high injection;" *i.e.*, the inserting of an oiled rubber-catheter for a considerable distance into the bowel and the injection of the fluid from a fountain-syringe through this. If the fluid of any injection is not expelled promptly, there is only one other direction in which it can pass, namely, toward the cecum; and it will do this whatever method of administration is followed.

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[1] Edema and Nephritis, 1921.

[2] La Pediat., 1923, XXXI, 258; 324.

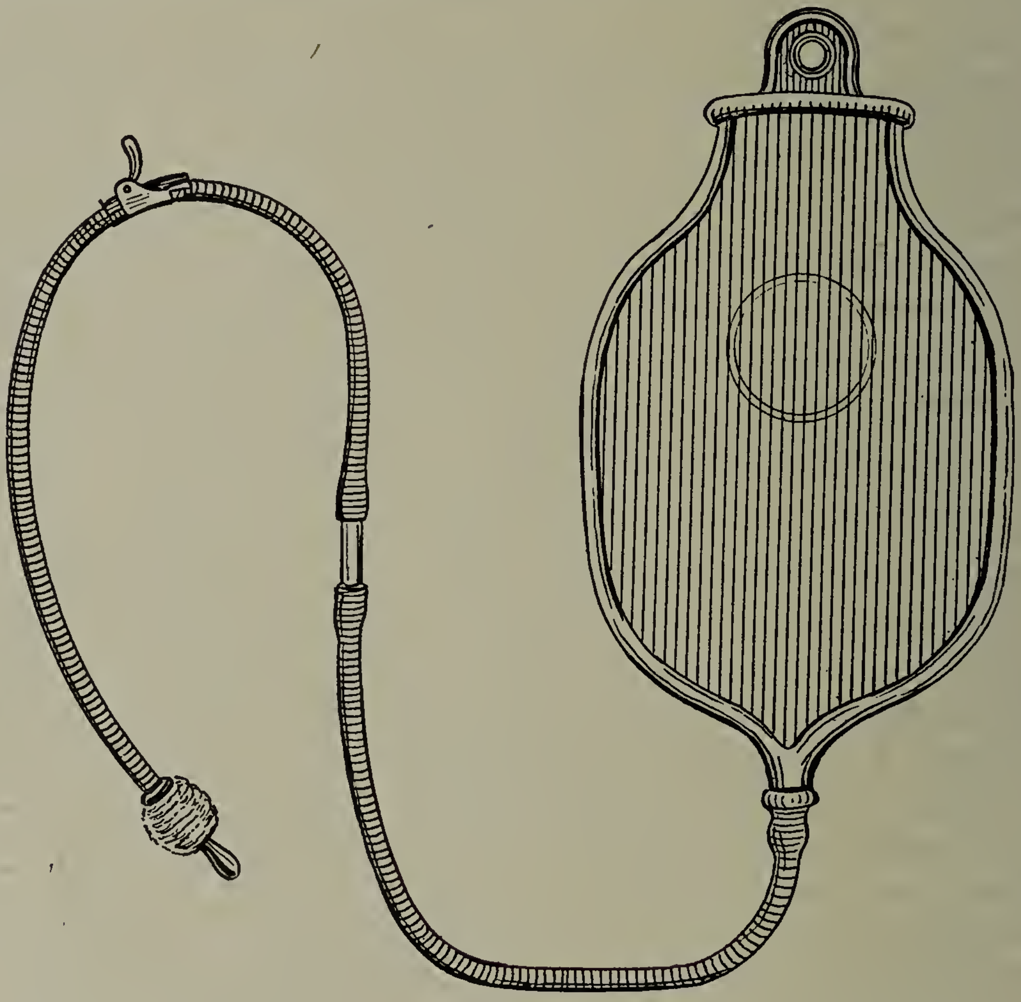

Fig. 35.—Fountain-Syringe for Intestinal Irrigation.
A roller-bandage wrapped around the nozzle checks the expulsion of the liquid.

The injection should be given slowly and with occasional cutting off of the water by squeezing or clamping the tube, in order to accustom the bowel to the pressure. There is no fear of over-distention occurring, if the bag of the syringe is not too greatly elevated. The infant will now and then expel the fluid with force at the sides of the nozzle, but the injection should be continued until a gallon (3785) or more has been used, or until the fluid expelled shows little, if any, fecal matter. The colon will hold a surprisingly large amount. A pint (473) may be introduced and retained without distention at the age of 6 months, and 1 to 2 pints (473 to 946) or more at the age of 2 years. After the injection is completed the syringe should be removed and the liquid allowed to escape. If a catheter has been employed, it may well be detached from the tube and left in position for a half hour or more until all the water has been expelled, as a portion of it is generally retained for a time.

The temperature of the fluid should generally be about 95° to 100°F. (35° to 37.8°C.) or somewhat less. In some instances slightly cooler injections of 75 to 90°F. (23.9 to 32.2°C.) are better, as where severe local inflammation exists, or where there is high fever, since the reduction of the body-temperature can be accomplished very satisfactorily in this way. Cool injections must not be used in greatly debilitated infants with low body-temperature or where collapse is impending. The fluid employed for irrigation may be simply water, or better, a normal salt-solution (0.9 per cent.) or about 1 teaspoonful to the pint. Where there is much local inflammation irrigation with starch-water (1 teaspoonful to the pint, boiled) is often very serviceable. If the starch-water is too thick or grows too cool while passing through the tube, it may jelly and cease flowing. Weak antiseptic solutions are also recommended.

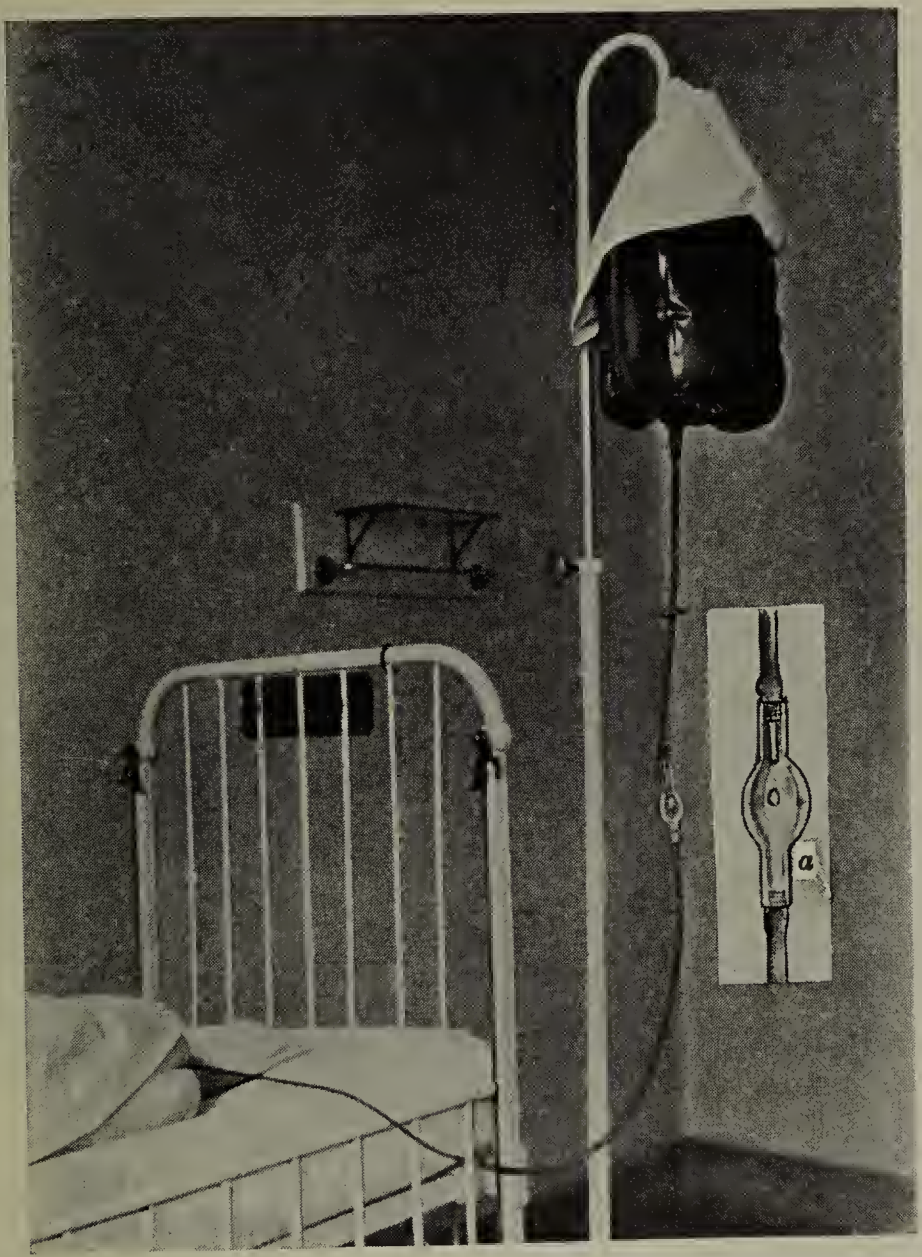

Fig. 36.—Apparatus for the Drop-Method of Enteroclysis.

The rapidity of the flow is controlled by a screw-clamp. Hot water-bags surround the vessel containing the liquid. (*a*) Larger view of the dropper-apparatus.

6. Enteroclysis.—In some cases of wasting disease or of collapse a pint (473), more or less, according to tolerance, of warm normal salt-solution may be introduced and allowed to remain (enteroclysis), the syringe being removed and the nates pressed together, the purpose now being to have as much as possible absorbed into the general circulation. A favorite plan is the "drop-method" (Fig. 36) in which the catheter is allowed to remain in place for some hours, with the fluid made to leave it in the form of drops at the rate of about 20 per minute.

7. Inhalations.—Inhalations of vapor constitute a most useful plan of treatment in early life. The inhalation may be of water-vapor alone or of chemical substances in the form of a vapor or spray. For the giving of a spray the steam-atomizer is very serviceable. The small glass vessel in front of the boiler may be filled with water or lime-water, or with solutions containing benzoin, small amounts of turpentine, or other substance as desired. The spray is best given under a croup-tent, and should not be allowed to play directly against the face.

A small room may be filled with water-vapor by repeatedly plunging red-hot iron—such as flat-irons, stove-lids and the like—or hot bricks or stones into tubs containing only small amounts of water. The slaking of lime in the room is an efficient old-time method for accomplishing the same purpose. It is impossible, however, to keep a large room satisfactorily filled with steam by these methods and the use of a croup-tent around the child is strongly to be recommended (Fig. 37). This may be improvised by placing a large opened umbrella over the patient and draping blankets over this. A better plan is to fasten poles, such as broom-handles, upright at each corner of the crib, and to connect the tops of these by cord. Blankets may now be thrown over the framework thus constructed, so that they fall down well about it. An "A" shaped opening is left near the head of the patient, and through this the vapor from a croup-kettle (Fig. 38) is conducted. The end of the spout should be close to or just within the door of the tent, but not close enough to give too great heat or to permit the child to strike it. The croup-kettle is sold provided with an alcohol lamp, and many accidents have occurred through this catching fire, or from the covers of the croup-tent coming in contact

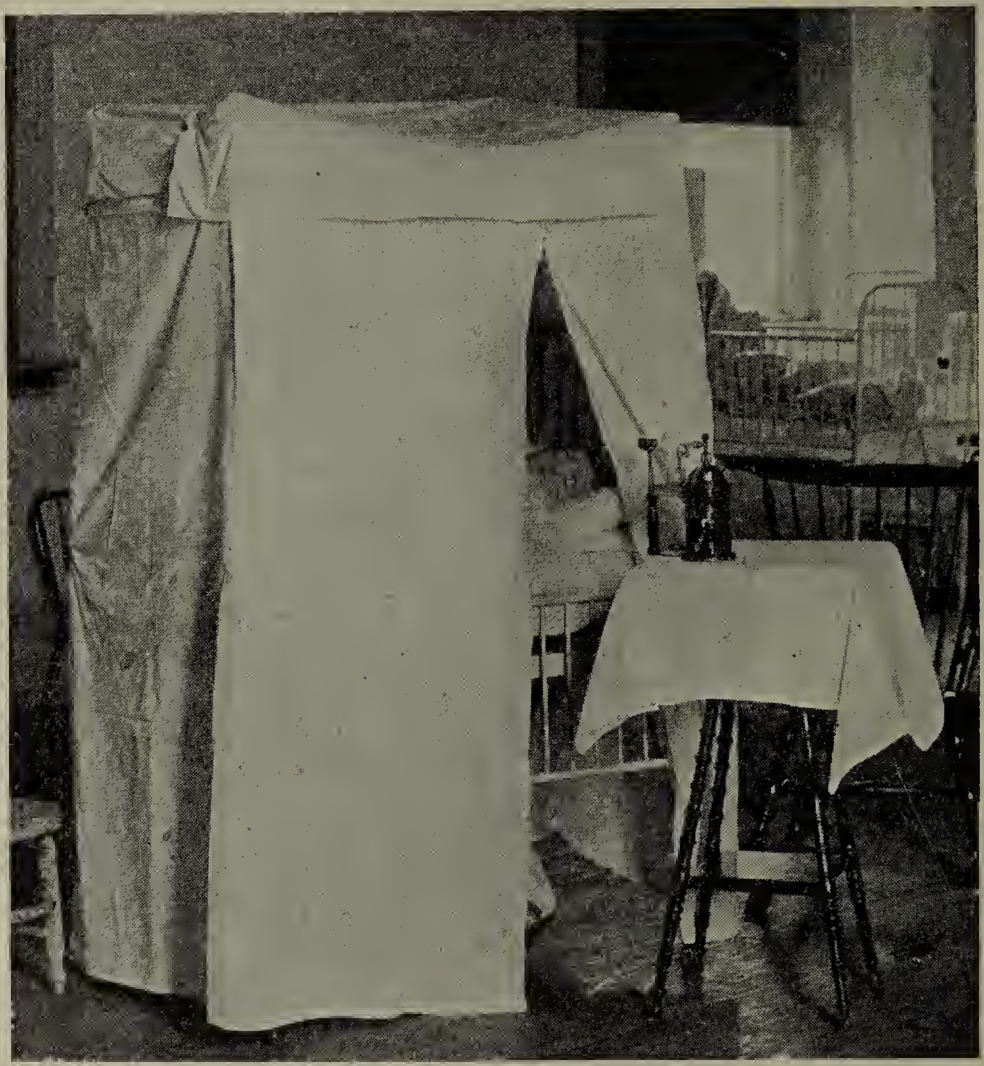

Fig. 37.—Croup-Tent.
Steam-atomizer on the table.

with it when these have been made of inflammable material. On this account sheets should not be used to form the covering, and, when possible, the croup-kettle should be heated by a small gas-stove or electric stove instead of by burning alcohol.

Volatile substances, such as benzoin, turpentine, eucalyptus, etc., may be added to the water in the kettle before the lamp is lighted; or a small sponge may be saturated with them and placed in the enlargement of the spout. Carbolic acid should be avoided, and creosote used with caution in the case of infants and young children.

Certain drugs may be given in volatile form without the use of water. Thus calomel may be volatilized by pouring it upon a piece of iron heated by a flame.

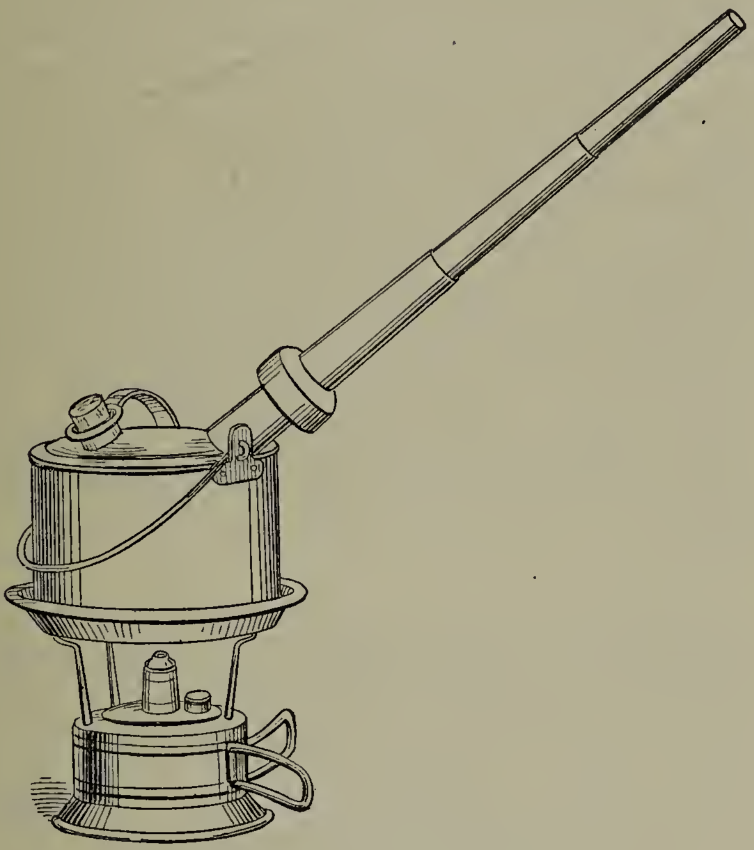

Fig. 38.—Croup-Kettle.

8. Inunctions.—Apart from the use of ointments for the local treatment of cutaneous affections, the rubbing with oily substances is sometimes employed to produce general absorption of the drug. Mercury is conveniently given in this way, a small amount of the official ointment or the oleate being rubbed into the axillæ or groins. A favorite plan in the case of infants is to apply it over the abdomen on a flannel binder. Salicylic acid, especially in the form of salicylate of methyl, is readily absorbed by the skin. There is some danger, however, of local applications of preparations of salicylic acid producing severe constitutional symptoms (Zumbroich).[1] Cod liver oil has been much used by inunction. Its odor is extremely unpleasant when employed in this manner and it is questionable whether it offers any advantage over other oils.

9. Applications to the Nose and Throat.—Gargles cannot generally be used before the age of 6 or 7 years, as the child is liable to swallow or reject them. **Painting the throat and nose** evokes resistance in nearly all cases, but is sometimes imperative, and is often easier than the use of the spray. The method of procedure has already been given in describing the examination of the throat.

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[1] Monatasschr. f. Kinderheilk. Orig., 1918, XV, 167.

Substances should be selected which do no harm if swallowed. The application should be made quickly but effectively. This may be done by means of a large camel's-hair brush mounted on a stick, or, better, cotton wrapped firmly upon a stout aluminum applicator. Painting the mucous membrane of the nose with medicated petrolatum is often very serviceable, and is usually a much easier method of treatment than the use of the atomizer unless the patient is docile. A small camel's-hair brush is required, and the procedure should be very gently carried out. In place of this in the case of infants the patient may be placed on its back upon the knees of the nurse, with its head hanging somewhat downward, and liquid petrolatum, medicated if desired, may be dropped into each nostril with a blunt-tipped medicine-dropper. In this position gravity takes the fluid to the upper part of the nostrils.

The hand-atomizer may be employed for **spraying the nose or throat.** For oily solutions an apparatus should be chosen which gives a strong spray in a short time and without too much labor. Some are very unsatisfactory. Liquid petrolatum, medicated in various ways as by camphor, menthol, etc., is much used. Aqueous sprays are serviceable for cleansing. For this purpose liquor sodii boratis compositus (Dobell's solution) or other weak alkaline-aromatic solution is serviceable. Peroxide of hydrogen is also much employed. A preparation of this should be selected which is not acid, and the dilution should be at least 1:4 for treatment of the nose.

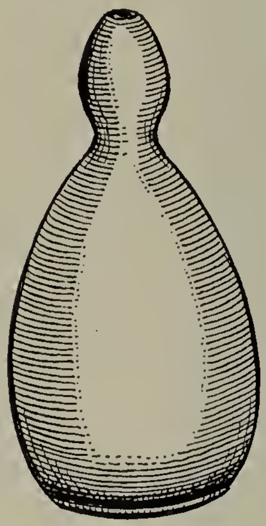

Fig. 39.—Olive-tipped Soft Rubber Syringe for Nasal Douching.

Syringing the nose is sometimes required. For this purpose the best syringe is one entirely of soft rubber (Fig. 39) holding about 1½ fl. oz. (44), or a fountain-syringe may be employed with a soft rubber nozzle. The child when debilitated should lie upon the side, the syringe being applied to the upper nostril and the injection continued until the liquid comes clear from the lower nostril. The head and body are then turned to the other side, and the syringing done through the second nostril. The danger of spraying or syringing forcing material into the Eustachean tube, or into the sinuses in older children, is a real one; and care and gentleness are required with the avoidance of much pressure. In cases where the child is strong it may be allowed to sit, having the head a little forward.

Syringing the throat is carried out in much the same way, the child sitting upright or lying upon the side. In the case of either the nose or throat, only mild, harmless solutions should be employed, such as normal salt-solution or a weak one of boric acid.

Insufflation of medicine in powdered form upon the mucous membrane of the nose and pharynx finds some place in the therapeutics of children, especially in such diseases as pertussis and diphtheria. The powder may be blown in with an insufflator. The application of drugs in this way to the larynx of infants and young children is in my experience difficult and unsatisfactory; certainly requires special training or skill, and is liable to produce severe laryngeal spasm.

10. Counter-irritants.—Treatment of this sort is particularly valuable in children. The delicacy of the skin in most cases is such that care must be taken to avoid too great irritation.

Blisters are not suitable for infants and not without danger of serious injury to the skin even in childhood.

Mustard-plasters or poultices are very serviceable in such conditions as bronchitis. They should be made of mustard with flour or flaxseed meal in proportions depending upon the age. In infancy a strength of 1 of mustard to 5 or 6 of flour is sufficient. For older children 1 of mustard to 3 or 4 of flour will answer. The mustard and flour are mixed dry and then stirred with hot water. The paste is then spread between two layers of thin muslin or linen and applied. The poultice is left on a sufficient time to redden the skin thoroughly, yet not long enough to blister. The time varies with the individual, and the skin should be inspected frequently. The plaster may be applied every day or several times a day according to the effect upon the skin and the requirements of the case. Mustard mixed with white of egg and glycerine instead of with water is less likely to irritate. The application of mustard in the form of baths or packs is considered later (p. 270).

Pepper-plasters and nutmeg plasters were formerly favorites and are sometimes useful. Lard or mutton-suet is spread upon a piece of muslin and liberally dusted with black pepper or powdered nutmeg. The plaster should be worn continuously, until sufficient irritation is produced.

Friction with turpentine and oil, oil of amber, soap-liniment, camphorated oil and the like is often of much value in bronchitis, adenitis, and other inflammations. The substance should be rubbed on with the hand or with a small piece of flannel until the skin is slightly reddened. In the case of many infants it seems impossible to produce satisfactory counter-irritation in this way, and the mustard-plaster is then to be preferred.

The spice-bag is an old-fashioned but very excellent application for colic in early infancy. Equal parts of ground ginger, cloves, cinnamon, and allspice are mixed and put into a small square flannel bag and spread evenly. The bag is then quilted to keep the powder in place. Before applying it should be wet with hot alcohol-and-water. The same bag may be used repeatedly until it begins to lose strength too greatly.

Turpentine-stupes are serviceable in abdominal pain or tympanites. A piece of flannel is wrung out in very hot water and sprinkled evenly with turpentine, about half a teaspoonful being used for each square foot of flannel, or rather less for infants. It is then applied and covered with oiled silk and a dry cloth. Its action must be watched lest too much irritation result.

Dry cups are occasionally useful in cases of severe bronchitis, broncho-pneumonia, nephritis or passive congestion of the lungs from cardiac disease. Their action is more powerful than that of friction or mustard-plasters.

11. Hydrotherapy.—The employment of water as a remedial measure, applied to the surface of the body in general or to portions of it, is one of the most important of therapeutic agents in infancy and childhood. Yet one of the greatest principles attaching to it is that hydrotherapy is not intended solely or even chiefly for the reduction of temperature. It serves many useful purposes in afebrile states; while when employed for patients with fever its favorable action is to be measured not so much by the degree of the reduction of temperature as by the other good effects produced. Hydrotherapy is often a harmful measure when used, as it too frequently is, with the apparent determination on the part of the physician to reduce the temperature of the body at any cost. Fever is only a symptom and, unless unduly high or prolonged, not one which does harm.

General baths may be divided into (*a*) sponge-baths, (*b*) tub-baths, (*c*) shower-baths, (*d*) sheet-baths, (*e*) vapor-baths, (*f*) medicated baths. Of the *local baths* may be mentioned (*g*) foot-baths, (*h*) compresses and fomentations. Baths may also be classified according to the temperature of the water. A convenient approximate classification is into: (1) Cold bath (40° to 70°F.) (4.4° to 21.1°C.); (2) Cool bath (70° to 80°F.) (21.1° to 26.7°C); (3) Tepid bath (80° to 90°F.) (26.7° to 32.2°C.); (4) Graduated bath (85° to 90°F.) (29.4 to 32.2°C.) reduced; (5) Warm bath (90° to 100°F.) (32.3 to 37.8°C.); (6) Hot bath (100° to 105°F.) (37.8° to 40.6°C.).

(*a*) The **sponge-bath** with *warm* water should be employed daily in nearly every case of illness. The child should be undressed completely and laid between blankets. The sponging should be done with care and without exposure, one part of the body being thus washed and dried before another is approached. *Cool* or *tepid* sponging is of great value for the reduction of temperature, and is often in early life as serviceable as is the whole bath in the case of adults. There is not the same need of the precaution alluded to against exposure of the body, since reduction of temperature is the object in view. The sponging should be continued for from 5 to 15 minutes, avoiding the production of decided cyanosis or long-continued depression of the pulse-strength. Some degree of pallor and of lessening of the force of the pulse is a frequent result, and considerable judgment is required to determine whether or not the bath is doing harm. Some children with fever do not bear cool sponging or even tepid sponging at all well.

(*b*) The **warm tub-bath** is an excellent measure in depressed conditions in the eruptive fevers marked by a retrocession of the eruption. Through its action the cardiac strength improves and the rash consequently reappears. It is also a useful antipyretic measure at the beginning of nearly any acute febrile disease, and is often serviceable in reducing temperature in continued fevers when a cooler bath is not well borne. It is a diaphoretic in cases of nephritis; is an excellent sedative in great nervous excitement, insomnia or convulsions; relaxes the spasm of false croup, and reduces the temperature and relieves the dyspnea of bronchopneumonia. Care must be taken to avoid exposure. The room should be warm, the child kept in the bath from 5 to 10 minutes, according to the effect, and wrapped in blankets with little drying as soon as removed from the water. In most cases of fever in infancy and early childhood it is much to be preferred to sponging.

The **hot tub-bath** is a powerful stimulant, serviceable in cases of severe exhaustion or collapse, or when the vital powers are failing and atelectasis developing, as in cases of premature birth or of bronchopneumonia in weakly infants. The temperature of the water should be 100°F. (37.8°C.) to not over 105°F., (40.6°C.) *determined by the thermometer*, and the infant should be immersed for not more than 3 minutes. Occasionally the procedure seems to do more harm than good, and to hasten a fatal ending which was inevitable. In other cases, however, the good results are surprising and justify any risk which may attend the measure.

The **tepid tub-bath** and the **graduated tub-bath** are useful antipyretic measures where sponging or the warm bath fails. In the graduated bath the water is 90°F. (32.2°C.) at the start and is gradually cooled down to 80° (26.7°C.) or sometimes less. Most older children bear bathing of this sort well and the measure is very serviceable in continued fevers, and often fatigues the patient less than sponging. Many children, however, are not benefited, and some do not tolerate baths of this temperature. It is well to bear this in mind and not to persist with bathing merely because fever continues. The child should be in the water from 5 to 10 minutes, being vigorously rubbed meanwhile, and carefully watched against too great depression. In very hot weather, when an infant appears to be exhausted by the continued heat, it is sometimes of advantage to give several tepid tub-baths daily for their bracing and cooling effect.

The **cool and cold tub-baths** are not often needed in childhood except in cases of very great hyperpyrexia, such as develops in sunstroke and sometimes in pneumonia and the eruptive fevers. In giving cold baths under these conditions the temperature of the child must be constantly watched and the bath stopped before apyrexia is reached.

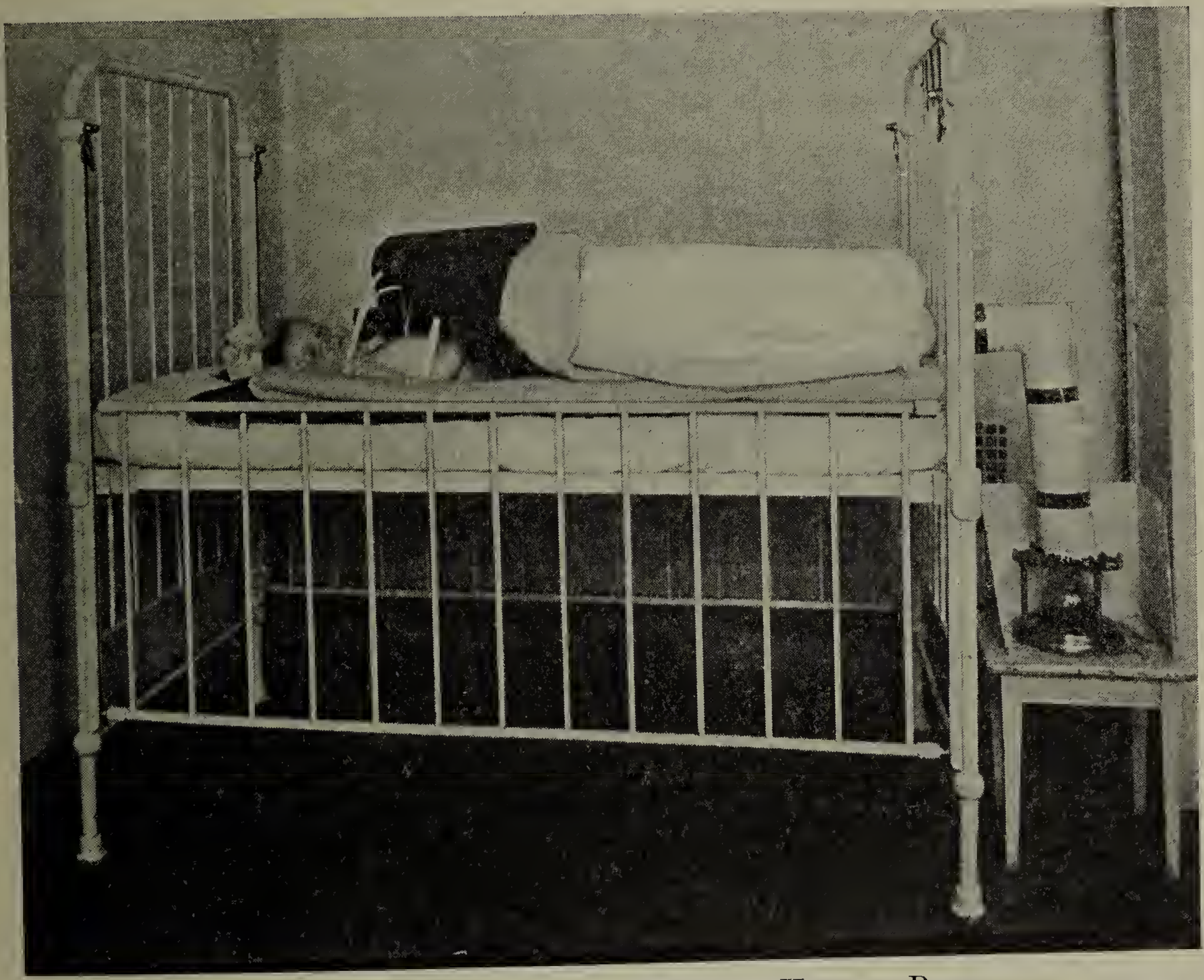

Fig. 40.—Apparatus for Giving a Hot-air Bath.

Rubber sheet and outer blanket turned back to show the supporting hoops above the child. Covered metal pipe conducts the hot air beneath the coverings at the foot of the bed.

(*c*) The **shower-bath** or **affusion**, is an excellent tonic and stimulant for delicate children. The child should stand in a tub of warm water in a warm room. The cooler water may then be applied from an ordinary shower-apparatus, or may be poured over the head and trunk from a pitcher or squeezed from a large sponge. The duration of the affusion should be brief, a minute or less being sufficient, followed by brisk rubbing with a Turkish towel. The water may be tepid, cool, or cold, according to the strength of the child and the degree or reaction which follows. If this latter is not satisfactory, warmer water should be used or the shower-bath not employed at all.

(*d*) The **sheet-bath, or wet pack,** may be either hot or cold. To apply a *cold* pack a rubber cloth is put over the bed and a sheet, previously wrung out in cold water, laid upon it. The child is now stripped, placed upon and enveloped, except the head, in the sheet, and, outside of this, in a blanket. It is often advisable to leave the feet out and to put a hot-water bag to them. The child may receive a second pack in 15 or 20 minutes, or may be left in the first for an hour or more if it has fallen asleep. When removed from the pack it should be wrapped in a warm, dry blanket.

The cold pack applied in this way is useful for quieting nervousness and often for reducing moderate fever. When a more decided antipyretic action is desired it is necessary to renew the pack every 5 minutes several times in succession, sometimes using ice water, but only if the temperature of the body is excessively high. It is often more convenient to substitute cloths dipped in ice water for the sheet, since these may more easily be removed, remoistened and replaced. The wrapping in the blanket may be omitted in these cases. A cold cloth must always be kept to the head.

The *hot pack, or blanket-bath,* is given by covering the child with towels wrung out in hot water, or wrapping it in a blanket similarly treated; and then enveloping in several dry blankets. The pack may be renewed in half an hour. The hot pack is often serviceable for producing free perspiration in cases of nephritis.

(*e*) The **vapor-bath** is employed to cause profuse perspiration in nephritis. The bed is well covered by a blanket and the child is stripped and laid upon this. Other blankets are then thrown over the child, reaching nearly or quite to the floor, but kept away from the body by half barrel-hoops, a chair in the bed, or some other support (Fig. 40) except where they are tucked in closely about the neck. Into the air-space thus formed about the child vapor is now conducted from a croup-kettle. The process is continued from 15 to 30 minutes if the child tolerates it well. Care must be taken that the vapor does not play directly against the body, since unconscious children have repeatedly been severely burned in this way. In the absence of a croup-kettle, vapor may be produced beneath the bed by slaking lime, or by hot iron dropped into water, the vapor being given an opening through which it can rise and surround the child.

The *hot-air bath* is applied in a similar manner and for the same purpose, dry hot air from an alcohol lamp or small gas-stove being conducted under the covers by a metal pipe (Fig. 40). With either procedure every precaution must be taken against the igniting of the bed-clothing.

(*f*) **Medicated baths** are of much service in childhood. The *warm* or *hot mustard tub-bath* is a powerful stimulant in cases of cardiac failure or where for any reason it is desired to bring the blood to the surface of the body. Often the good results obtained are surprising. Mustard is added to the water in the proportion of 1 oz. (28) (6 level tablespoonfuls) to 1 gallon (3785) of water. The duration of immersion should be 10 minutes, or less if the skin has become well reddened or if the bath is not well borne. The development of a sensation of tingling produced by the mustard-water on the nurse's arms is also an indication to remove the child. The *mustard-pack* is a convenient and useful application in cases of prostration or collapse where the physician fears the greater disturbance of the tub-bath. The child is stripped as for the ordinary pack, laid upon a blanket, and covered, except the head, with a cloth dipped in hot mustard-water, slightly stronger than that used for the tub-bath. The blanket is then wrapped around the body. The pack may continue for 10 minutes or more according to the degree of redness of the skin which it produces. A very efficient method of applying a powerful mustard-pack is recommended by Heubner.[1] I have seen it occasionally produce surprising results in patients apparently almost moribund. Heubner advises the mixing of "2 handfuls of mustard-flour to a liter of warm water, and stirring thoroughly until the odor of the mustard has become distinctly irritating to the nose and eyes. A cloth is now dipped in the mustard-water, wrung out slightly, and wrapped completely about the patient up to the neck, and a blanket wrapped outside of this. The child remains in the pack 10 minutes, and is then washed quickly and thoroughly with warm water, and reenveloped in a fresh pack of simple warm water, where it remains 2 to 3 hours."

The *starch-bath* may be made of the strength of ½ cupful of boiled starch to every 4 gallons (15,142) of water. If the starch has already jellied it may be reheated, or pressed through moistened cheese-cloth. This bath is useful in some affections of the skin. Starch-water for washing the skin may be made in the same way.

The *soda-bath* consists of a solution of 1 tablespoonful of carbonate of soda (washing soda) to every 4 gallons (15,142) of water. It is used for the same purposes as the starch-bath, and is often combined with it by dissolving the soda in the starch-water.

Salt-baths have been much used as a tonic treatment for debilitated children, particularly those with rickets. Rock-salt, coarse table-salt, or preferably dried sea-salt may be dissolved in water in the proportion of 4 oz. (113) (10 level tablespoonfuls) to 1 gallon (3785) of water. The child may be washed with or immersed in this after having had the soap-suds of the ordinary washing removed with plain water. The duration of immersion depends on the temperature of the water and the condition of the case in general. Doubtless salt-baths, if sufficiently cool, do good, but whether this is in any way due to the presence of the salt is questionable.

The *bran-bath* is made by putting 1 lb. (454) (about 3 pints) or more of bran into a thin muslin bag and boiling this in water for a quarter of an hour. This water is then added to that of the bath until the whole is slightly milky in appearance. Bran-baths have been employed in many irritated conditions of the skin in infants and children.

The *sulphur-bath*, used sometimes in chronic rheumatic disorders and in some affections of the skin, is made by dissolving 20 grains (1.3) of potassium sulphide in each gallon (3785) of water employed. It cannot be given in a metal tub.

Disinfecting baths are employed after recovery from infectious diseases. They may consist of a 2 per cent. solution of carbolic acid for older children, or of liquor sodæ chlorinatæ, diluted to the strength of 6 fl. oz. (177) to 1 gallon (3785) of water. A solution of corrosive sublimate of the strength of 1:10,000 may be used instead.

Various *mineral springs* furnish whole medicated baths useful in many affections, notably those of Nauheim for the treatment of cardiac diseases, and various hot sulphur springs for rheumatic conditions. Modifications of the Nauheim baths may be given at home.

(*g*) Of the local baths the *foot-bath* is one of the most serviceable for older children. It is generally given in the form of the hot mustard foot-bath, of the strength of 1 oz. (28) (5 level tablespoonfuls) of mustard to 1 gallon (3785) of water. Care must be observed that the room is warm and the bed-clothes also. The child, dressed in its night-clothes, sits on the edge of the bed, well wrapped with blankets, including the thighs, while the feet and legs are in the tub of mustard-water. After 5 or 10 minutes the feet are rapidly dried and wrapped in a warmed blanket, and the child put to bed. Exposure is avoided even more completely if the tub is in the bed under the covers, while the child lies with the knees drawn up and the feet in the mustard-water.

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[1] Lehrb. d. Kinderheilk., 1911, II, 269.

(*h*) **Compresses** and **fomentations** constitute what may be called local forms of baths. *Cold compresses* consist of thin cloth, folded into several layers, dipped in ice water, wrung out and laid on the affected part. They must be light, not wet enough to drip, and changed every few minutes. They are serviceable in inflammation of the eyes, sprains, etc. *Hot compresses*, or *fomentations*, are made of flannel in several layers, which has been wrung out in water as hot as can be borne. This wringing is conveniently done by dropping the wet flannel into a dry towel and then thoroughly twisting this. The nurse then tests the flannel against her cheek, applies it quickly to the part, and covers it with oil-silk and then with dry flannel or a dry towel. It should be renewed in an hour, or less if it is desired to maintain decided heat.

(*i*) **Poultices.**—The poultice is intended to furnish a wet dressing which will retain heat longer than a fomentation. It is commonly composed of flaxseed-meal, but other substances may be used instead, such as cornmeal, bread, starch, slippery-elm, etc., according to circumstances. The *flaxseed-poultice* is made by stirring ground flaxseed into a small quantity of water nearly or quite boiling, until it is of the consistency of hot mush, too thick to flow. This is spread with a case-knife upon a thick piece of cotton or linen cloth, the edges folded over slightly, and the whole covered with cheese-cloth, gauze or a thin old pocket handkerchief. The nurse should test it against her cheek to see that it is not too hot, apply it, cover it with oil-silk or paraffin-paper, and enclose with a bandage. It should be renewed every few hours if the heat is to be maintained. The *slippery-elm poultice* and the *cornmeal-poultice* are prepared in the same way, from ground elm-bark or from cornmeal. They have no special advantages over the flaxseed-poultice. The *bread-and-milk poultice* is popular and easily prepared. Stale bread-crumb is stirred into hot milk until the proper consistency is attained. It is then spread as described. Any of these poultices are applicable to many cases of local inflammation when there is no open wound. In the latter case an antiseptic fomentation is generally preferred.

In many cases of pain and tenderness, as in some abdominal affections, where a flaxseed-poultice would be too heavy, a *bran-poultice* may be substituted. A flannel bag is partly filled with bran, thoroughly wet with boiling water, wrung out in a towel, and applied. In place of this a *hop-poultice* may be prepared in the same way. Neither of these are as popular as formerly. The *mustard-poultice* has been described in considering counter-irritants. The *jacket-poultice* and the *cotton jacket*, the latter if properly made acting somewhat like a poultice, are apparently going more and more out of vogue; it seems to me with good reason.

12. Dry Cold.—This may be applied by coils of small lead or rubber tubing, fitted to the affected part, and through which cold water is conducted (Leiter's coils). A more convenient method, however, is the employment of ice-bags. Applied to the head an ice-bag is often of benefit for reducing temperature in febrile diseases; applied over the abdomen it is even more effective for this purpose. It often gives relief from the pain of pleurisy or cardiac disease. Ice is also useful applied below the angle of the jaw in cases of tonsilitis. The bags employed should, however, be of thin rubber. The long sausage-shaped bag of thick rubber generally sold for application to the neck is not serviceable. The ice-bag used in any region of the body should never be quite filled or it cannot adapt itself well. The local employment of ice must be watched very carefully, and is more suitable for older children than for young infants. In the latter it may cause dangerous depression. It is often best to insert one or more layers of thin cloth between the bag and the skin to prevent harmful action upon the latter. The experience of Zondeck[1] upon rabbits showed that cooling of the surface of the head produced a prompt and very decided fall of the temperature within the brain, even as much as 2.4°C. (4.4°F.). This should make us cautious in the employment of the treatment.

13. Dry Heat.—This is useful particularly for the relief of pain and for the treatment of chronic articular affections. In acute painful conditions, such as otitis; for the relief of shock or collapse; and for low temperature in marantic or premature infants the employment of hot water-bags or bottles constitutes an excellent therapeutic procedure. For chronic articular inflammation dry heat may be applied by baking in a special apparatus made for the purpose, or by the use of radiant heat from a group of electric lamps. With all infants and in unconscious states at any age precautions must be carefully taken against burning the patient.

14. Blood-letting.—Venesection is not suitable for infancy and early childhood and only rarely indicated in older children. There are exceptional cases of uremia or of acute distention of the right side of the heart where it does good. Local blood-letting by wet cups should rarely be employed except in strong subjects in later childhood. Leeching may occasionally be serviceable in dilatation of the right side of the heart, uremia, otitis, meningitis, and pneumonia in strong children.

15. Vaccine, Serum and Protein-therapy.—This method of treatment has come of recent years into great prominence, but, like all new methods, has been overrated, while possessing undoubted value in some conditions. Vaccine-treatment consists in the hypodermic administration of a suspension of dead bacteria. As a prophylactic measure for typhoid fever it appears certainly to have established its position, and for the treatment of the attack it seems to be of value. In most of the streptococcic infections the usefulness of vaccines is doubtful. Sometimes they seem to do good in erysipelas, and the treatment is well worth trying. Staphylococcic infections offer a better field, especially in suppurative processes in the skin. The value of vaccine in pertussis is still under discussion, and it must be stated that the efficacy of the treatment has at least not been proven. The same may be said regarding the employment of gonococcus-vaccine in vulvo-vaginitis, and of that of the colon bacillus in pyelitis. In all these disorders instances are on record of decided improvement following their use; while other investigators have failed to accomplish any good whatever.

The employment of sera, and of the extractive matter from bacteria, is of somewhat older date, but occupies a very limited field of usefulness.

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[1] Zeitschr. f. phys. u. diät. Therap., 1922, XXV, 84.

The serviceableness of the diphtheria-antitoxin is beyond question, as appears to be that of the toxin-antitoxin preventive treatment of this disease and of tetanus antitoxin as a prophylactic measure. The same is true also of the serum-treatment for cerebrospinal fever; and that for some forms of pneumonia is encouraging. Tuberculin has not maintained the position once hoped for it, and is a remedy by no means without danger. The serum-treatment of scarlet fever and of measles, especially in the way of prophylaxis, seems to be becoming established. Of considerable interest is the use of foreign protein-substances given by mouth, subcutaneously, or intravenously for their nonspecific effect. In certain hemorrhagic conditions the injection of a foreign protein, or even of human serum or of blood itself, appears to be distinctly beneficial in controlling the bleeding. It has been claimed by some writers that much of the value of any vaccine or serum is due to nonspecific reaction, but experience and theory do not permit the employment of nonspecific measures when specific measures are available. Especially is this true in the case of such diseases as diphtheria and meningococcic meningitis. A great variety of substances have been used to produce nonspecific reaction, among them bacteria such as colon bacilli, typhoid bacilli and Coley's fluid; horse-serum and antidiphtheritic serum; boiled milk, and secondary proteoses and peptone. Usually these are given subcutaneously or, in smaller doses, intravenously; but a few, such as peptone, have been tried by mouth. It is to be noted that the conditions which are treated by nonspecific therapy are, as a rule, those which are difficult to cure and for which specific measures are not available or are of little value. Good results have been claimed and undoubtedly obtained at times in asthma, eczema, arthritis, ocular infection, anemia, malnutrition, gastro-intestinal disturbances, influenza, pneumonia, scarlet fever, streptococcus infections, etc. It is necessary, in order to obtain results, that a certain amount of reaction should occur after the injections. This reaction is shown by chill, fever, increased pulse and malaise. The mechanism by which nonspecific therapy acts is a matter of discussion. By some investigators it is supposed to be closely allied to anaphylaxis. In some manner the body-defences are stimulated and antibody-formation is increased; leucocytosis occurs, and there is an increase of the fibrinogen in the plasma and of globulin in the blood. The theories have been reviewed by Petersen.[1]

16. Intravenous Injections.—The employment of this procedure is considerably interfered with in infancy owing to the difficulty in finding a vein. The longitudinal sinus at the position of the fontanelle may be used instead. Marfan[2] in 1898 was the first to employ the longitudinal sinus to inject saline-solution. Tobler[3] studied the anatomy of the sinus and found that it is larger at the posterior angle of the fontanelle; at which point it is 2 to 5 mm. (.08 to .20 in.) under the scalp and has a depth of 4 to 7 mm. (.16 to .28 in.). In puncturing the sinus an 18 gauge needle which has a bevel of 45 degrees is used. The posterior angle of the fontanelle is entered in the mid-line, with the needle at an angle with the head of about 35 degrees and pointed backward. The procedure seems a safe one also for the withdrawal of blood, because transfixing the sinus does not cause internal bleeding (Brown and Smith.)[4]

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[1] Protein-Therapy and Nonspecific Resistance, 1922.

[2] Quoted by Blechmann, Le Nourrisson, 1914, II, 150.

[3] Monatsschr. f. Kinderheilk., 1915, XIII, 384

[4] Amer. Jour. Dis. Child., 1917, XIII, 501.

It is a favorite way of obtaining blood for Wassermann reaction or for blood-culture. In older children intravenous injection is useful in cases of collapse, as after profuse hemorrhage or diarrhea, using a sterilized warm normal salt-solution (0.9 per cent.). The treatment of syphilis by arsphenamin is preferably given intravenously. Sodium bicarbonate may be administered intravenously in cases of acidosis (see p. 260), or citrated blood or a 5 per cent. solution of glucose in severe malnutrition in gastrointestinal disorders. About 20 c.c. (0.68 fl. oz.) of the glucose-solution may be given for each kilogram (0.31 fl. oz. per pound) of the body-weight. Diphtheria-antitoxin has been used in this manner.

The employment of intravenous injections of sodium chloride, magnesium sulphate or glucose has proved of value in certain conditions in which there is increase in intracranial pressure (Fischer,[1] Weed and McKibbon;[2] Haden;[3] Fay[4]). The action is a dehydrating one on the tissues of the body including the brain and the ventricles, and the injections would be indicated in edema of the brain, uremia and intracranial injuries. They have been of use in our hands in serous meningitis, the increase in intracranial pressure occurring in acute infections, uremia, the acute stages of encephalitis, and in purulent meningitis. Their effect is usually manifest within a few hours, and may be maintained over a long period by daily repetition of the injections. They are contraindicated in the presence of shock and such symptoms as subnormal temperature and rapid and increasing pulse-rate. Sodium chloride may be used in 15 per cent. solution, glucose in 25 to 40 per cent. solution, and magnesium sulphate in 2 per cent. solution. They should be administered slowly, at body-temperature; taking about 10 minutes to give 15 or 20 c.c. (0.507 or 0.676 fl. oz.) of the solution. The amount will depend upon the condition and the age of the patient as well as the results obtained, but in general will vary from 50 to 150 c.c. (1.69 to 5.07 fl. oz.).

Substances cannot be injected into the longitudinal sinus with entire impunity. It is easy to transfix its wall if the patient moves, and the injected material may enter the subarachnoid space or be forced into the brain-tissue. We have seen 3 instances of this when arsenical preparations were employed. Unilateral convulsions resulted in all, and 2 of the infants died. At necropsy obtained in 1 of these cases there was distinct necrosis of one cerebral hemisphere. In order to prevent movement of the needle various apparatus have been designed by Helmholz,[5] Goldbloom[6] and Ratner.[7] The latter's consists of a small block perforated so that a needle of any caliber may be used, and bevelled so that the sinus may be entered at an acute angle. Before the injection of any fluid into the sinus, one should be sure that the needle is within it, as shown by the successful aspiration of blood into the syringe.

17. Intraspinal and Intracranial Therapy.—Various sera are used intraspinally, such as antimeningococcic, antipneumococcic, antistreptococcic, antitetanic and influenzal. A serum has also been prepared against poliomyelitis. In some diseases, such as chorea,

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[1] Edema, 1910, 129.

[2] Amer. Jour. Physiol., 1919, XLVIII, 512.

[3] Jour. Amer. Med. Assoc., 1919, LXXIII, 983.

[4] Jour. Amer. Med. Assoc., 1923, LXXX, 1445; 1924, LXXXII, 766; 1925, LXXXIV, 1261.

[5] Amer. Jour. Dis. Child., 1915, X, 194.

[6] Amer. Jour. Dis. Child., 1920, XIX, 229.

[7] Amer. Jour. Dis. Child., 1921, XXI. 199.

(Goodman)[1] so-called auto-sera have been given, the blood-serum obtained by bleeding the patient being injected into the spinal canal. These matters will be discussed more fully under their respective headings. In meningitis, especially of the tuberculous variety, numerous antiseptics have been employed for intraspinal injection but without avail. For spinal anæsthesia a 25 per cent. sterile solution of magnesium sulphate in the dose of 1 c.c. (0.03 fl. oz.) for every 25 pounds (11340), (.09 c.c. (0.003 fl. oz.) for every kilogram) of body-weight may be used; or cocain (0.5 to 1.0 per cent.) and its derivatives may be given. The employment of either substance is best in the hands of an expert. Besides the treatment of meningitis by the intraspinal or intracranial route the method may be employed for other purposes. In the management of syphilis of the nervous system, when the spinal fluid shows a positive Wassermann reaction or the less suggestive signs of increased cell-count, albumin and globulin, intraspinous injections of arsphenamized serum may be resorted to; although it is usual to depend upon vigorous general treatment by inunctions of mercury, iodides by the mouth, and intramuscular and intravenous injection of arsenical preparations.

The technique for the performance of lumbar puncture, ventricular puncture and cisterna-puncturec has been described elsewhere (p. 216, 217). Whether these routes of approach to the cerebrospinal system are used for diagnosis or treatment, the method of entering the subarachnoid space is the same. If, after drainage, serum is to be injected it should always be given after it has been warmed to about 100°F. (37.78°C.), and an amount smaller than the amount of fluid removed should be introduced. The serum should be given slowly, about 10 minutes being consumed in giving 15 c.c. (0.51 fl. oz.), and the patient's condition closely observed during the process; symptoms such as great restlessness and feeble pulse, with slowing of the rate, being indications of too great an increase in cerebrospinal pressure and necessitating the cessation of introduction of the serum. In certain cases where the fluid is very thick it is of advantage to wash out the subarachnoid space either by introducing 5 or 10 c.c. (0.17 or 0.33 fl. oz.) of normal saline solution and allowing it to flow out again, or by inserting one needle into the cistern and another into the lumbar region and washing from above downward.

18. Transfusion of Blood.—The introduction of blood from a human subject into the veins of a patient has been found useful in cases of hemorrhage or of severe anemia from other causes. It has also been employed successfully in hemorrhagic diseases in the new-born, and is to be highly recommended in severe malnutrition, in which condition repeated small injections are given. The transfusion may be accomplished directly into the vein from the circulation of the individual furnishing the blood, or the blood may be aspirated into a glass-syringe and then promptly injected into one of the large veins of the child, such as the median cephalic, median basilic, external jugular, popliteal, or femoral, or the longitudinal sinus. Either procedure requires technical skill, the first especially so. The simplest method is the employment of a citrated blood, using 1 part of a sterilized 2 per cent. solution of sodium citrate and 10 parts of blood. This prevents coagulation. With any method it is essential to test first the agglutinative action of the blood of the recipient and of the donor upon each other, or dangerous hemolysis may take place (Minot).[2] This is true even in new-born infants (see

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[1] Arch. of Pediat., 1916, XXXIII, 649.

[2] Bost. Med. and Surg. Jour., 1916, CLXXIV, 667.

p. 54). From 1 to 2½ fl. oz. (30 to 75) of blood may be injected at the age of 6 months or less; 3 to 4 fl. oz. (89 to 118) at 1 year, and 4 to 6 fl. oz. (118 to 177) at 2 or 3 years (Zingher).[1]

Dunievitz and Bruckman[2] report the intracardiac injection of whole (uncitrated) blood in a new-born infant, giving 40 and later 85 c.c. (1.35 and 2.89 fl. oz.) through a No. 19 gauge needle inserted in the 4th interspace about 2½ cm. (1 in.). to the left of the sternal border. Sidbury[3] employed the umbilical vein for transfusion in the new-born, this vessel being patent up to the 4th day of life. A small amount of saline solution was first introduced to determine that the vein was still open.

19. Intraperitoneal Transfusion.—Citrated blood may be injected into the peritoneal cavity (Siperstein;[4] Sansby and Siperstein[5]), the blood acting as a true transfusion with entrance of the cells into the circulation (Ruh and McClelland).[6] The recipient and the donor are typed, and the citrated blood must be fresh and kept at body-temperature. Opitz and Metis[7] prefer defibrinated blood. The technique is the same as for intraperitoneal injections of any solution (see p. 260), and the amount of blood given varies from 50 to 500 c.c. (1.69 to 16.9 fl. oz.), depending upon the age and the urgency of the indications. We have found this procedure valuable and have used it without preliminary typing of blood. Hill, Smith and Cross[8] have successfully combined intraperitoneal transfusion with intraperitoneal saline injections, simply introducing the required amount of blood during the saline injection and through the originally inserted needle.

20. Lavage.—Very frequently obstinate acute or chronic vomiting will be made to cease by lavage, after the administration of drugs has failed entirely. A stomach-tube is employed, composed of a soft rubber catheter, No. 13 or 14 American scale, connected by a piece of thin glass-tube to a section of soft rubber tubing, the other end of which is attached to a funnel of hard rubber or glass. The infant sits in the lap of the mother while the nurse introduces the end of the catheter moistened with water or glycerin (Fig. 41) into the mouth and gently but quickly passes it backward into the pharynx and downward into the stomach. A slight resistance is felt as the tube reaches the beginning of the esophagus. The finger of the left hand, placed upon the tongue, may be used as a depressor and a guide. There is very little danger of the tube entering the larynx, but should it do so violent coughing and cessation of breathing will inform us of the fact. About 10 inches (25 cm.) of the catheter should be inserted, measuring from the gums. The funnel is then raised slightly to permit gas to escape and the solution is then poured into it and allowed to find its way into the stomach. Pinching the tube between the finger and thumb and sliding these along it toward the mouth is sometimes needed to start the flow.

After from 4 to 8 fl. oz. (118 to 237) have been given the funnel is lowered as far as possible, in order to syphon out the gastric contents into a basin. The tube is then pinched to prevent air from entering, the funnel raised and more fluid poured in. This process is repeated several times until from a pint to a quart (473 to 946) of fluid has been employed and the washings come away clear of curds or mucus.

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[1] Med. Rec., 1915, LXXXVII, 440.
[2] Arch. of Pediat., 1925, XLIII, 139.
[3] Amer. Jour. Dis. Child., 1923, XXV, 290.
[4] Amer. Jour. Dis. Child., 1923, XXV, 107; 202.
[5] Jour. Amer. Med. Assoc., 1923, LXXX, 1763.
[6] Ohio State Jour. Med., 1923, XIX, 780.
[7] Jahrb. f. Kinderheilk., 1924, CVII, 269.
[8] Arch. of Pediat., 1926, XLIII, 186.

The solution ordinarily used is a warm (100° to 110°F.) (37.8° to 43.3°C.) normal saline solution (0.9 per cent.), simple boiled water, or, where there is much mucus, an alkaline solution containing 1 per cent. of bicarbonate of soda. The procedure is entirely without danger, very easy to carry out during infancy, and causes, as a rule, little or no exhaustion. It may be performed daily until no longer needed.

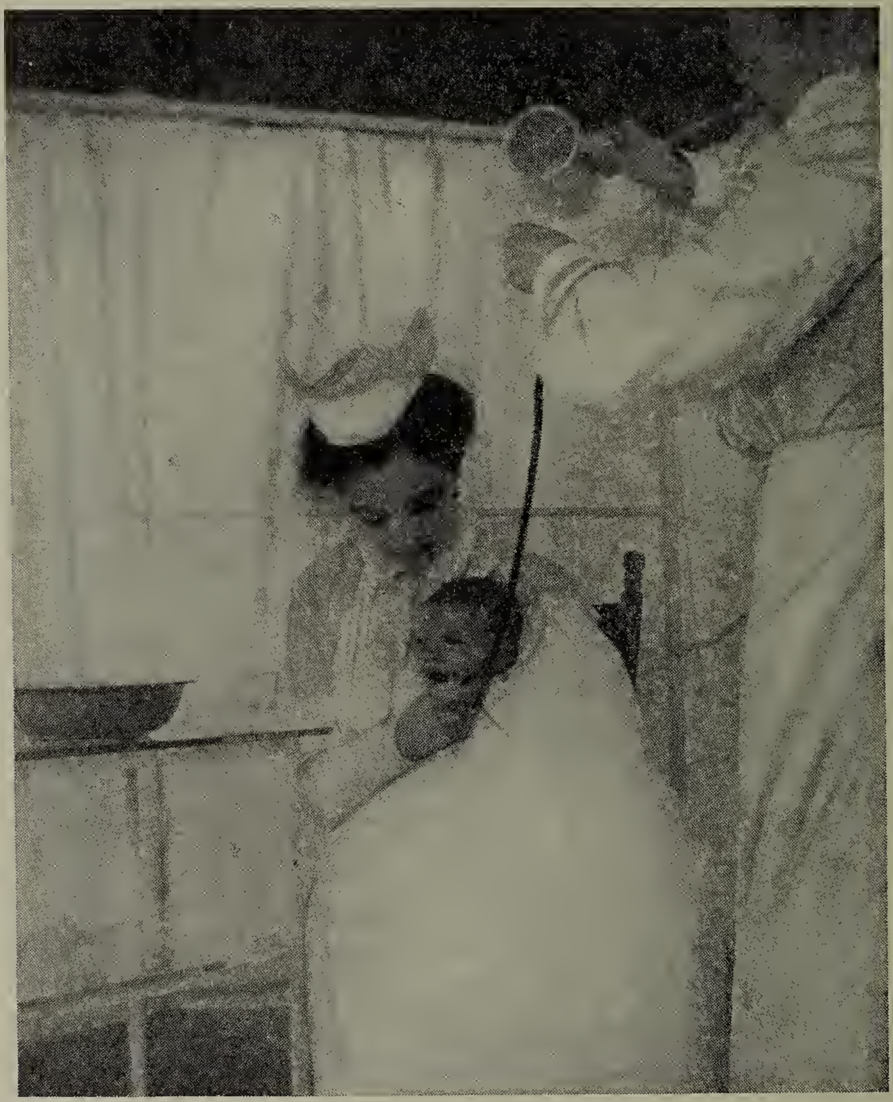

Fig. 41.—The Performance of Lavage of the Stomach.

21. Gavage.—The forced feeding of infants is so closely connected with lavage that it may well be described in this connection. After the stomach has been washed, the tube may be left in position and the child fed through it. The funnel should be held elevated for a moment before the food is poured into it, in order to permit gases to escape from the stomach. Instead of sitting the child should be flat upon its back, as it is less likely to regurgitate the food when in this position. For the same reason the child must be kept entirely quiet after the gavage is over. In older subjects it may be necessary to use a mouth-gag to prevent the tube from being bitten, or to protect the finger from the child's teeth. When the tube is about to be withdrawn it should be pinched tightly to prevent food from dropping from it into the pharynx, as this is liable to induce vomiting. The last part of the withdrawal should also be done very quickly. Should vomiting occur immediately a second gavage may sometimes be given at once with advantage.

Gavage is very easy to perform and is extremely useful in the case of infants who for any reason are unwilling or unable to take sufficient nourishment, or in those where vomiting is troublesome. In the latter case lavage should precede each gavage; in the former lavage is not necessary. It is constantly observed that food given by gavage will often be retained much better than when swallowed. Cases where extreme anorexia threatens life may often be fed with success by gavage. The food may sometimes be thoroughly peptonized with advantage. Sometimes it must be weak on account of an impaired digestive power; in other cases, where the trouble is the refusal to take nourishment in the ordinary way, it may often be given rather stronger and in larger amount than usual, since the intervals of feeding are longer. Children may be fed in this way 3 or 4 times a day, or occasionally more frequently. In some cases where vomiting or lack of power to swallow is not a factor, but where unwillingness to take food is the cause, it is not necessary for the tube actually to enter the esophagus, but a short piece of tubing may be passed through the mouth to the pharynx and the liquid introduced from a funnel or syringe through this.

22. Nasal Feeding.—In older children where the jaws are tightly shut and a mouth-gag would be required to permit of inserting a tube into the esophagus, it is better to feed through the nose. This is also a serviceable method of feeding after intubation. The procedure is similar to that described for gavage, except that a smaller tube is required, No. 8 or 10 American scale (Fig. 42). In some instances it is better to use the nasal tube for the performing of lavage.

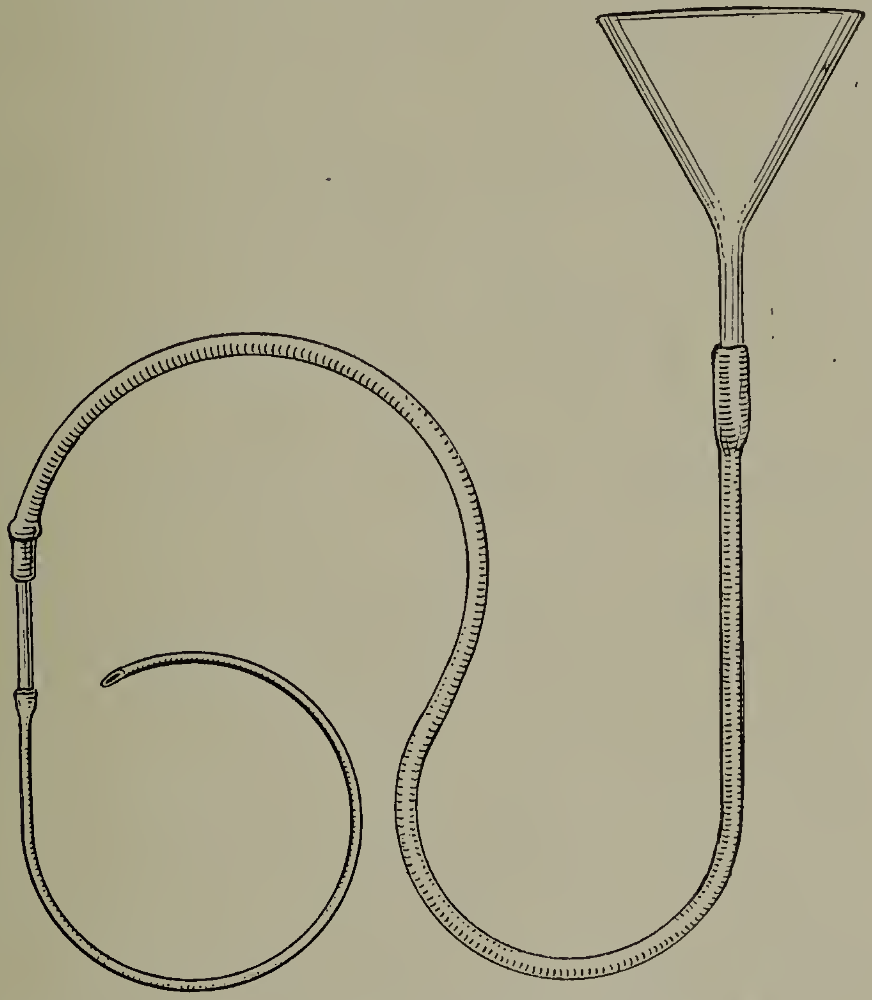

Fig. 42.—Funnel and Tube for Nasal Feeding.

23. Anæsthesia.—Little can be said in this connection which does not apply to adults as well. As a rule anæsthetics are well borne in early life. There is a possible danger of sudden death in cases of lymphatism, but this may occur in those of older age as well. Ether is by all odds the safest anæsthetic and the one usually to be preferred. The excitement and resistance which it occasions may be avoided by beginning the anæsthetization with ethyl chloride, which, however, is a more dangerous anæsthetic if depended on solely, particularly in infancy. Nitrous oxide may be used in the same way, or alone for short operations in older children, but is not as safe for infants. Ether is generally to be avoided in diseases of the lungs and in nephritis. Whether ether or chloroform is selected, the vapor should be administered slowly and with the admixture of plenty of air; and this applies with especial force to chloroform.

24. Psychotherapy.—The enormous importance of this form of therapeutics in early life is too frequently overlooked. Even in early infancy it is seen, for instance, in the good effect of mental quietude or the influence of a calm and somewhat phlegmatic nurse in the controlling of troublesome vomiting; as also in the relief of insomnia by such measures as the avoidance of excitement before the hour for sleep, the removal of noise or light from the room, and the like. In older children the insistence by the physician that the mother shall not speak of nervous symptoms in the presence of the patient, and that these shall be in every way ignored, is something we cannot afford to forget if good results are to be obtained. The remarkable influence of complete change of surroundings and of removal from association with over-anxious parents is shown by the frequent, prompt disappearance of spasmodic symptoms, nervous anorexia or dysphasia, or other nervous or psychic manifestations when a child is placed under hospital-influences or sent from home in the care of a properly selected nurse. The control of the exhibition of anxiety by the parents and of the bad methods of training shown by them is often the principal and the most difficult object of treatment. The subject is too large for further discussion in this connection. It is referred to briefly to some extent in the chapter upon Diseases of the Nervous System, and I[1] have discussed it also elsewhere.

25. Mechanotherapy.—**Massage**, even when carried out in a very simple and unscientific manner, and in a way which any mother can do, occupies a valuable place in the therapeutics of infancy. The rubbing of the body with the flat of the hand, lubricated with sweet oil or cocoa butter, is an excellent procedure in feeble circulation, malnutrition, anemia, and paralytic conditions. Still better is it to encircle the arm or leg of the infant with the well-greased thumb and forefinger, and to push up and down the limb with the exercise of considerable pressure. More effective is a gentle kneading of the muscles done by a trained masseuse. The procedure usually soon becomes very soothing and agreeable to the patient. Care must always be taken to avoid undue exposure of the body lest chilling result. In older children a more systematic massage, given by one experienced in the method, is often useful, especially so in paralytic or pseudo-paralytic states, faulty habits of sitting or walking, lateral curvature of the spine, and the like. Abdominal massage is frequently one of the best remedies for obstinate chronic constipation. In the case of active children massage does not fill the place which it does with adults, since the child takes relatively far more exercise than the average man undergoes.

Resisted movements may be used either alone or combined with massage. They are serviceable, for instance, in some cases of paralysis and of diseases of the heart and for the training of any especially weak muscles.

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[1] New York Med. Jour., 1914, XCIX, 1113.

For children sufficiently old **gymnastic exercises** are of great value, conducted by a trained observer and selected to meet the needs of the individual case. Even without such an advantage, a series of exercises can be chosen by the physician and supervised by an intelligent mother or nurse in the patient's home. These may be used to overcome the results of faulty habits of standing, sitting or walking; distortions of the chest following rachitis or pleurisy, or deformities acquired in other ways. It is necessary, of course, to know the action of the different muscles and to outline special exercises to bring these into play. The reader may be referred to treatises upon exercise and upon orthopedic surgery, and especially to the interesting contribution by Keating and Young[1] upon Physical Development.

In the class of mechanical therapeutics is, of course, to be placed the application of **mechanical apparatus** of different sorts, as in the treatment of the paralysis of poliomyelitis, hip-joint disease, and the like. This is eminently in the domain of orthopedic surgery. Merely to be mentioned here is the use of mechanical support in combination with muscle-training for lordosis and faulty position of other sorts, with consequent enteroptosis, constipation, poor digestion and similar conditions. The subject has been instructively discussed by Talbot and Brown.[2]

Rest, with or without isolation, is often most important. Thus in chorea, many cases of cardiac disease, epilepsy, and hysteria it is by far the most important part of the treatment. The fact that nervous, active children are constantly liable to take by far too much exercise is always to be remembered. In such cases systematic enforced rest during a portion of the day is to be enjoined. Many functional nervous disturbances may be aided by the prescribing of recumbent rest for an hour in the middle of the day. That the child sleep at this time is an advantage, but need not be insisted upon. If he is wakeful or restless, he may be allowed to look at a picture-book or to have some not too exciting story read to him.

Electricity is a useful form of mechanotherapy in paralytic conditions and in some other affections. The fright which the application may cause often interferes largely with the benefit which might be expected. Only the weakest current should be employed at first, and never one of sufficient strength to cause pain. Indeed, the first application may well be that of the wet sponges alone, the battery being in action but unconnected. The current to be chosen depends upon the nature of the disease. As a mere matter of muscle-exercise the faradic is indicated.

26. Radiotherapy.—The value of radiotherapy as a therapeutic method has been constantly coming into increasing prominence. Although its field is as yet limited, it has established its value in such conditions as some forms of splenic hypertrophy, notably leukemia; enlargement of the thyroid and thymus glands; cervical and tracheo-bronchial adenitis; and in eczema, tinea tonsurans, nevi, and other affections of the skin. The subject has become so specialized and the treatment requires so much judgment and experience that it cannot be given further consideration here.

27. Heliotherapy.—There is much value in the sun's rays, of which advantage may be taken in the treatment of disease. In rickets, tuberculosis of glands, bones and peritoneum, and in local cutaneous affections the effect of exposure to sunlight is often curative. Heliotherapy is particularly valuable in rickets, and in this condition it is the short ultraviolet ray which is effective. This ray is filtered out by ordinary window-glass, and consequently unobstructed exposure is necessary. Heavy clothing, especially that which is dark in color, also cuts out the ray. In glandular tuberculosis the use of sunlight seems to be more beneficial when given at a considerable altitude, but in surgical tuberculosis the treatment is better combined with sea-air. In pulmonary tuberculosis heliotherapy has less effect than in the forms just mentioned, but in subjects with a tuberculous tendency, from constitutional character or inheritance, it is widely employed. In rickets the mercury vapor-lamp can be used successfully to obtain the same results as exposure to sunlight (see Rickets, p. 706). The lamp likewise produces cure of tetany, perhaps more rapidly than does any other means of treatment.

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[1] Keating, Cyclopedia of the Diseases of Children, 1890, IV, 301.

[2] Amer. Jour. Dis. Child., 1920, XX, 168; 1921, XXI, 347.

Children must be exposed only gradually to the rays of the sun, in order to avoid burns of the skin and the nervous irritation which occasionally occurs. Individuals whose skin does not pigment on exposure seem to derive less value, as a rule, from heliotherapy than those who become characteristically bronzed. Lucas,[1] who has given an excellent summary of the use of heliotherapy in pediatrics, has described the program as follows: On the first day the exposure is made of the feet, and consists of from 1 to 3 periods at intervals of at least 1 hour, each lasting from 3 to 5 minutes. On the 2nd day there are from 1 to 3 periods at hour intervals of 5 to 10 minutes applied to the feet and of 3 to 5 minutes to the legs. On the 3rd day there are 1 to 3 periods at hour intervals, of 10 to 15 minutes to the feet, 5 to 10 minutes to the legs, and 3 to 5 minutes to the thighs. In this manner the exposures are increased so that by the end of the 1st month the patient can remain in the sunlight, practically totally exposed, from 3 to 5 hours a day, and by the 3rd month he can remain from 5 to 8 hours both winter and summer.

The action of the sun's rays is in part understood. In addition to the specific effect of certain rays there is also the action of heat and drying. In rickets heliotherapy produces a retention of calcium; in local infections there is a bactericidal action. The blood shows an increase of hemoglobin, red blood-cells and eosinophiles. At first there is an elevation of temperature and an increase in the rate of both respiration and pulse, both of which soon strike a constant equilibrium. Continued irregularity of temperature and of pulse is an indication of an intolerance of the treatment.

28. Climatotherapy.—It is a matter of everyday experience that the cure or relief of many diseased conditions will be accomplished more surely and promptly by change of climate than in any other way. Children with subacute bronchitis often react quickly to such a procedure. Asthma, pertussis, chronic or recurring rheumatism, chronic nephritis, tuberculosis, anemia, a debilitated state of the general or the nervous system, and slow convalescence from many acute diseases, are all aided by it. The choice of locality is to be made carefully. A fuller discussion is out of place in this connection, and reference may be made to works upon Climatology. Here it may be said only that many of the good effects of change of climate can be obtained by a more careful regulation of the hygiene at home, particularly in the matter of obtaining sufficient fresh air in the living rooms, both by day and by night, a life largely in the open air, and the guarding against overheating from too great warmth of the clothing.

[1] Arch. of Pediat., 1920, XXXVII, 193.

DIVISION II

DISEASES

SECTION I

DISEASES OF THE NEW-BORN

Monsters and similar malformations arise during *embryonal* life, that is, through the first 2 lunar months. They are the products of defective development rather than of disease and are properly considered in works on teratology rather than on pediatrics. Some of them will be mentioned briefly in other sections. Certain of the diseases of the *fetus; i.e.* developing during the last 8 lunar months of intra-uterine life, continue as diseases of the new-born, since the pathology is the same for both periods of life. Others, however, seem incompatible with the continuance of life outside the uterus and cannot properly be considered at any length in works treating of the diseases of infants and children. Consequently any grouping of Diseases of the New-born is at best artificial and incomplete, and only to be excused by the greater convenience for study which it offers. In the following pages, therefore, are discussed chiefly (1) some of those diseases of fetal life which are capable of continuing, at least at times, in the living child at birth, and (2) certain affections which are peculiarly liable to be acquired by the new-born child or are witnessed only at this time. There are excluded from this section or mentioned only briefly (1) numerous diseases of the new-born which are more conveniently treated of later, (2) diseases limited to fetal life and not seen in the living infant; and (3) most instances of defective embryonal development; *i.e.* matters pertaining to malformations and to teratology in general.

CHAPTER I

PREMATURE INFANTS

Etiology.—The frequency of premature birth varies to some extent with the locality and the influence of attendant circumstances. There is a range of from 5 to 25 per cent. according to the statistics of institutions, as published by Rommel.[1] An average might be fairly assumed as 9 per cent. of the total births (Schauta).[2] After the 38th week of intra-uterine life the infant can no longer be called premature, inasmuch as it is then perfectly developed to all intents. In the absence of actual knowledge of the duration of the pregnancy, the existence of prematurity must be determined according to somewhat arbitrary standards. In general it is assumed that a birth-weight of less than 2500 grams (5.51 lb.) or a length of less than 45 cm. (17.72 inches) is an indication that the child is probably premature. Yet there is a great variation in the weight of premature infants, similar to that seen in those born at full term; and the criterion of length is decidedly more reliable, although even this is not entirely to be depended upon.

A distinction is to be made between cases of congenital asthenia and those of premature birth. The latter, it is true, generally exhibit debility, but not necessarily so; whereas the child with congenital debility born at full term may present many of the evidences of imperfect development characteristic of the prematurely born. The distinction, therefore, cannot always be made clinically with clearness.

Among the causes of premature birth are especially conditions affecting the mother. Syphilis is probably chief of these, 10 to 12 per cent. of the premature births being due to this cause (Baumm)[3] but acute infectious disorders, pneumonia, nephritis, tuberculosis and diseases of the heart occupy a prominent etiological position, as do violent exercise, trauma, placenta prævia and premature separation of the placenta. Twin births are very likely to be premature, Miller[4] finding prematurity in 60.6 per cent. of 3380 twin births. These estimates were, however, based upon the size and weight of the infants, which are less applicable to twins than to single births.

Characteristics at Different Periods of Intra-uterine Life.—A brief description may be given of the characteristics of the infant born prematurely after different periods of intra-uterine life which are compatible with at least temporary viability. The appearances at earlier periods than these have no bearing upon pediatrics. The lengths and weights of the body are those given by Ballantyne.[5] The months are calendar months:

At 24 weeks (5½ months), the fetus measures from 28 to 34 cm. (11 to 13.4 inches) in length and weighs 676 grams (1.49 lbs.). There is a large amount of lanugo, the skin is wrinkled and the vernix caseosa is present. The eyebrows and lashes are evident. The deposit of subcutaneous fat has only just commenced. The testicles have descended to the internal inguinal ring. The eyelids have become separated.

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[1] Pfaundler und Schlossmann, Handb. der Kinderheilk., 1906, I, 2, 492.

[2] Eulenburg's Real Encyclopädie, 1895, VIII, 120.

[3] Zentralbl. f. Gynäk., 1923, XLVII, 1624.

[4] Jahrb. f. Kinderheilk., 1886, XXV, 181.

[5] Antenatal Pathol. and Hygiene, 1902, 77.

At the close of 28 weeks (6½ months), the fetus measures about 38 cm. (15 inches) in length and averages a weight of 1170 grams (2.58 lbs.). The whole body, except the palms and soles, is covered with lanugo and the vernix caseosa is present. The hair on the head is about 0.5 cm. (0.2 inch) in length. The skin is dull-reddish and somewhat wrinkled. There is little subcutaneous fat. The pupillary membrane which had previously covered the pupil has commenced to disappear. Meconium is found in the intestine. The testicles have nearly or quite descended. (Figs. 43; 44.)

At the age of 32 weeks (7½ months) of intra-uterine life, the prematurely-born child measures from 39 to 41 cm. (15.4 to 16.1 inches) and weighs 1571 grams (3.46 lbs.). The lanugo has diminished on the body and the hair increased on the scalp. The skin is still dark-red and wrinkled, although there is more subcutaneous fat than before. The nails are harder and horizontal, but do not project beyond the ends of the fingers. The pupillary membrane has disappeared. The testicles have fully descended. The infant is very feeble, of low body-temperature, hardly opens the eyes, and cannot suck.

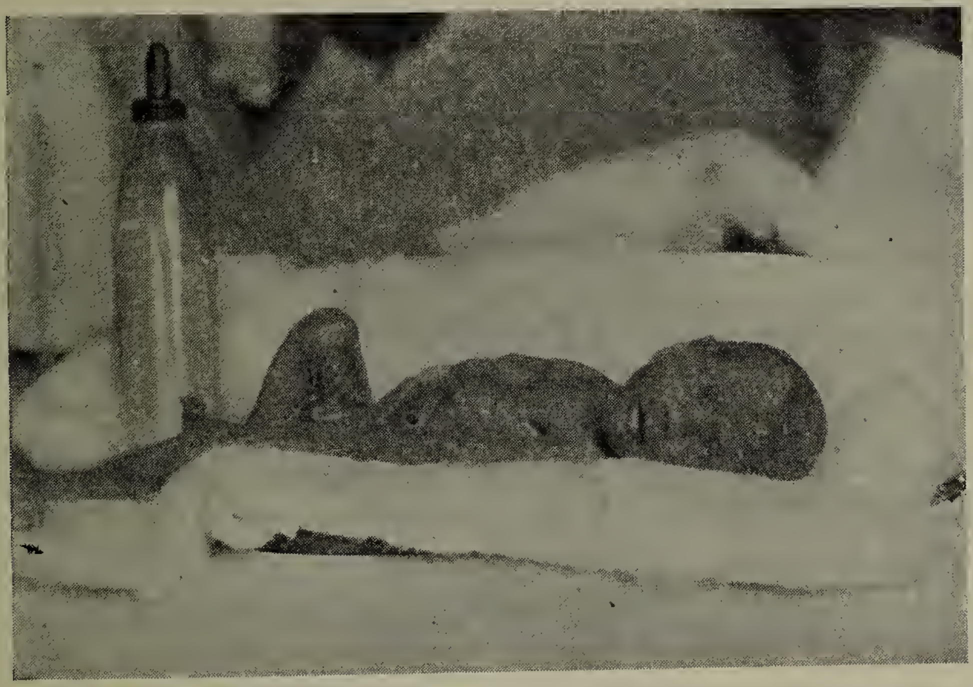

Fig. 43.—Premature Infant.

Born at 6½ to 7 months; weight on admission to the Children's Hospital of Philadelphia when 10 days old 2 pounds, 8 ounces (227). The size of the infant can be estimated by comparison with the 8-ounce nursing-bottle.

At the age of 36 weeks (8½ months), the fetus measures 42 to 44 cm. (16.5 to 17.3 inches) and weighs 1942 grams (4.28 lbs.). The subcutaneous fat has very decidedly increased and the wrinkling of the skin is much less, while the face is more rounded. The lanugo has largely disappeared. The deep-red color of the skin is now found only on the genitals. The hair on the scalp is over 1 cm. (0.39 inch) in length. The nails do not reach the finger-tips. Children born at this period should live under careful attention.

General Symptoms.—The general symptoms characteristic of prematurity vary with the duration of the period of intra-uterine life. In typical cases the cry is feeble and infrequent; the body-temperature is low and maintained at the normal only with difficulty; the infant moves its limbs but little and lies most of the time in an inactive, torpic state; there is a great tendency to atelectasis and consequent cyanosis which recurs readily after temporary successful treatment; the skin is often icteric and red, or pale, or cyanotic; the respiration is markedly irregular, feeble and intermittent; the power to suck is often absent and swallowing is slow. Inasmuch as the infant was born before its natural time, all the organs are in an imperfectly developed condition It should be stated, however, that Ylppö,[1] as a result of extensive autopsical experience, does not believe that mortality at this period depends upon the imperfect development so much as upon distinct pathologica lesions largely due to trauma incident to birth. This produces hemorrhage in various parts of the body, but is especially frequently subarachnoid or intrapial; at least 30 per cent. of the deaths occurring shortly after birth depending upon intracranial hemorrhage.

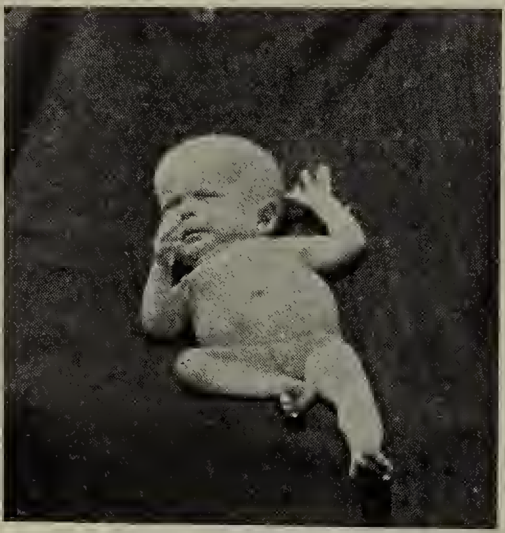

Fig. 44.—Premature Infant. Same case as in Fig. 43. Now aged 6 months. Weight 7 pounds, 10 ounces.

Course and Prognosis (See also Mortality, p. 235).—Even with successful treatment the progress is slow; repeated relapses into atelectasis are prone to occur; convulsions and pneumonia are liable to develop the danger of infection is decided; the digestion is often feeble; a toxic condition often develops; there is constant difficulty in maintaining the body-temperature, and the occurrence of any chilling may be followed by fatal results; the gain in weight is slow; and internal hemorrhage takes place readily, sometimes in the suprarenal bodies and especially, as stated, within the cranium, generally from the meninges. As a result of this intracranial hemorrhage, or depending upon the imperfect development of the brain-cells, imbecility, epilepsy, or cerebral diplegia is liable to be present in later life. The initial relative loss of weight in premature infants as compared with their birth-weight is greater than it is in the normal new-born (see p. 5), and the gain in weight and length is liable to be slower. Other things being equal, the chance of living depends upon how early the special care required is commenced, and the skill with which this is conducted. In addition to this, and very largely, the prognosis for final recovery is determined by the intra-uterine age. A child born at the end of 24 weeks usually dies in a few hours. At the close of 28 weeks, recovery occurs in about half of the cases with sufficient care. At the end of the 32d week the majority of the infants will live with proper attention. These figures show the inaccuracy of the quite wide-spread popular belief that the 7-months infant is more likely to survive than one born at 8 months. The statistics of Potel[2] are interesting in this connection. They are shown in the following table:

Table 76.—Viability of Premature Infants

			Per cent.
56 infants born at 6½ fetal months;	45 died	=	80.4
131 infants born at 7 fetal months;	76 died	=	58.1
53 infants born at 7½ fetal months;	17 died	=	30.1
110 infants born at 8 fetal months;	39 died	=	35.5

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[1] Zeitschr. f. Kinderheilk. 1919, XX, 212.
[2] Thèse de Paris, 1895.

Only 1 case survived out of 26 born at the end of the 6th fetal month. A few cases are reported in medical literature of survival at even earlier periods, as, for instance, those recorded by Smyth (21 weeks) and Barker[1] (23 weeks). The statistics in the table correspond quite closely with those of others, which may consequently be omitted.

As already stated, the weight alone is not a safe prognostic guide, since it by no means always corresponds with the actual intra-uterine age of the child. Some remarkable instances of recovery are on record in infants with unusually small birth-weights; as, for example, the case reported by Mansell[2] in which the baby weighed 18 oz. (510) at birth and survived, and that by Bonnaire[3] of an infant weighing 820 grams (28.8 oz.). The length of the infant is a better prognostic indication, especially as the intra-uterine age is often difficult to determine with accuracy. Ostrčil[4] gives the following serviceable table:

Table 77.—Viability of Premature Infants

40 cm.	(15.8 inches)	Chances of living 21	per cent.
41 cm.	(16.1 inches)	Chances of living 20	“
42 cm.	(16.5 inches)	Chances of living 25	“
43 cm.	(16.9 inches)	Chances of living 28	“
44 cm.	(17.3 inches)	Chances of living 51	“
45 cm.	(17.7 inches)	Chances of living 50	“
46 cm.	(18.1 inches)	Chances of living 55	“
47 cm.	(18.5 inches)	Chances of living 58	“

According to Cook[5] if a premature infant survive the first 24 hours its chances are doubled, and after 3 days the mortality drops to 15 per cent. Whatever handicap there is in size, gestation-period, or infantile or maternal complications will operate largely during the first 3 days. After a few months anemia and rickets and perhaps spasmophilic manifestations are very prone to develop. Bunge[6] showed that in the full-term new-born animal at least 5 times as much iron is stored in the liver as occurs in the case of the adult. This prevented the development of the anemia which would otherwise result from the low iron-content of the milk. In the premature infant, however, it is claimed that there is but a small amount of iron stored in the liver, since the deposition of most of the iron takes place in the latter months or weeks of fetal life. Lichtenstein,[7] in a series of careful experiments, found the iron-balance always negative in premature infants, and even abundant nourishment and the administration of large amounts of iron as medicine gave only partial relief. This indicated that the cause is a functional insufficiency of the hemapoietic apparatus. There is also a deficiency in the calcium deposited in infants born before term, which partly accounts for the frequent and early development in them of rickets and spasmophilia. The studies of Givens and Macy[8] and Schmitz[9] show very definitely that the calcium-content of the fetus increases greatly in the last few months of pregnancy. Further, there is a low calcium-retention (Hamilton),[1] and the impossibility of exposure to the direct rays of the sun furnishes another factor in producing the condition attributable to calcium-impoverishment. The premature infant who escapes these consequences of prematurity, or who overcomes them through proper treatment, will develop as well in later years as the one born at full term. Extensive study by Ylppö,[2] however, showed that up to about the age of 5 years there are in most cases still evidences of retarded corporal development.

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[1] Ref. Kleinwachter in Eulenburg's Real Encyclopädie, VIII, 60.

[2] Brit. Med. Jour., 1902, I, 773.

[3] Bull. soc. de méd. légale de France, 1912. Ref. Arch. f. Kinderheilk., 1913, LIX, 213.

[4] Monatsschr. f. Geburtsh. und Gynäk., 1905, XXII, 45.

[5] Arch. of Pediat., 1921, XXXVIII, 201.

[6] Lehr. d. phys. u. path. Chemie.; 1894, 99.

[7] Acta Pædiat., 1921, I, 194.

[8] Jour. Biol. Chem., 1922, L, 34.

[9] Arch. f. Gynäk., 1923, CXXI, 1

Treatment.—Among the principal difficulties in the treatment of premature infants are the maintenance of the body-temperature; feeding; the control of imperfect respiration; and the avoidance of infection. For many years the accomplishing of the first of these was attempted by the employment of an *incubator*, and numerous forms have been devised. In all the effort is made to maintain the temperature automatically at a fixed degree, and to supply abundant warm, fresh, moist air. The difficulties attending this successful ventilation are so great that, on the whole, incubators have been found unsatisfactory even in practise in institutions. One of the best is the Hess[3] bed, which is electrically heated and water-jacketed. A much better plan is to have a room set apart for the purpose, in which the temperature is kept at from 85° to 90°F. (29.4° to 32.2°C.) and fresh warm air constantly supplied.

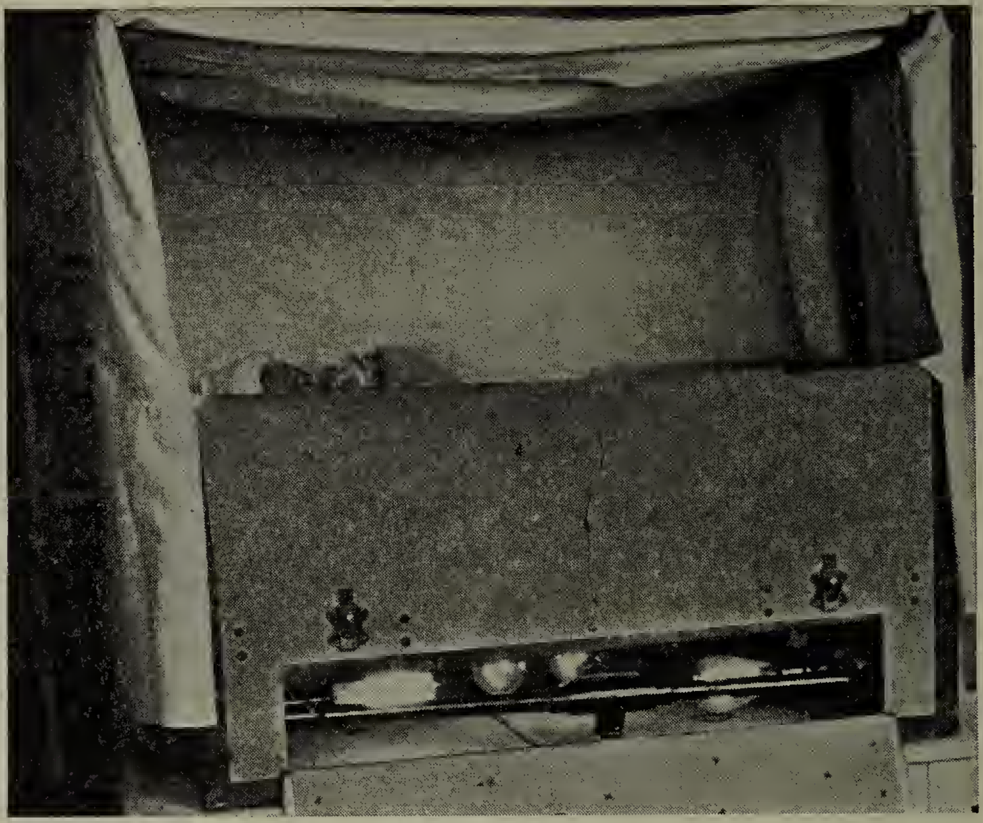

FIG. 45.—BED FOR PREMATURE INFANT.

Iron framework covered with asbestos-board. Cover thrown back. Door open at bottom showing electric lamps.

In institutions where no such room is available, as well as in private practice, very good results may be obtained in other ways, if sufficient intelligent attention is given. A lined clothes-basket or an infant's metal bath-tub may be partially filled with absorbent cotton and the child placed in this after having been oiled and wrapped in warmed cotton and then in blankets. Hot water-bottles are concealed in the cotton in the tub, close to but not touching the infant. A thermometer in the cotton shows the temperature attained here. It should range from 85° to 95°F. (29.4° to 35°C.), and must be carefully watched, as otherwise the infant's temperature may readily become too high. The aim should be to keep the baby's temperature steady. Irregular rises and falls are an indication that the infant is not doing well. The bottles should be refilled with hot water as needed, but not all of these at the same time, in order to keep the temperature of the bed as uniform as possible. Electric warming pads are theoretically excellent but practically not without danger, as a short circuit may develop and the bed-clothing be burned. I have, however, used them with decided satisfaction. A very serviceable device consists of a box-like crib of asbestos card-board or of metal, with an upper and a lower compartment. In the lower a series of electric lamps are installed, and any number desired turned on to produce the required heat in the bed. In the upper compartment is placed a thin mattress or a blanket, and over this cotton, and the infant enveloped as already described. Perforations in the partition between the compartments allows the heated air to rise. A sheet or blanket should be suspended on a support over the upper end of the bed to cut off excessive light and any draughts. I have used a device of this nature with success at the Children's Hospital of Philadelphia and in the Maternity Ward of the Hospital of the University of Pennsylvania (Fig. 45). The room in which the infant is kept should receive an abundance of fresh, clean, moistened, warm air, and the room-temperature maintained at 80°F. (26.7°C.) or sometimes over this. The infant should have no clothing whatever, and the cotton be arranged to cover the whole body except the face. A diaper, or an extra layer of absorbent cotton, should be laid under the nates. In changing this, and, in fact, under all circumstances, the child must be handled or exposed as little as possible. Unless strong enough to nurse from the breast, it is not necessary to remove it from the receptacle even for feeding, but a frequent change of position in bed is important. Bathing should not be employed at all, unless it be hot baths as needed for the relief of atelectasis. Once a day the child may be rubbed all over with warm sweet oil. After an evacuation of the bowels or bladder the nates may be cleansed with cotton and warm oil. The infant's rectal temperature should be taken every few hours, and an effort made to keep it at over 98°F. (36.7°C.). When it is found that the temperature is maintained without artificial heating, this may be gradually abandoned, and the child dressed in the ordinary manner.

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[1] Amer. Jour. Dis. Child., 1920, XX, 316.

[2] Zeitschr. f. Kinderheilk., 1919, XXIV, 111.

[3] Feeding and the Nutritional Disorders in Infancy and Childhood, 1925, 517.

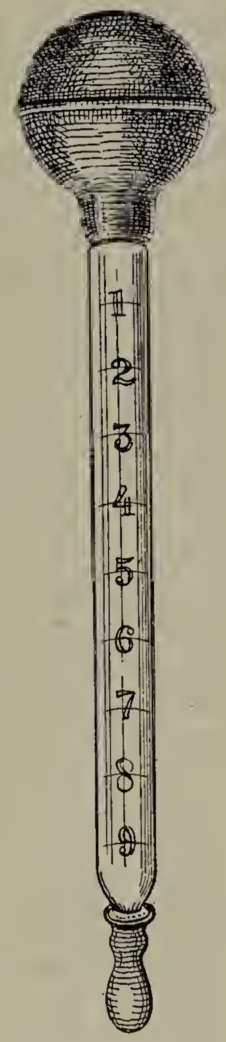

Fig. 46.—Breck Feeder.

The best food by all odds is a suitable breast-milk. In fact this is usually the only food with which satisfactory results can be obtained. The infant may nurse from the breast when it has the power to suck. If it has not, the milk may be pumped from the breast and given in a medicine-dropper or the Breck feeder (Fig. 46), the infant not being removed from the bed. It is sometimes best to dilute the milk ½ with water. Carbohydrate is especially well tolerated by these infants, and in many cases, where an insufficient amount of breast-milk is taken, this may be reinforced by the addition of 10, 15 or more per cent. of sugar (see

Concentrated Feeding, p. 149). When there appears to be difficulty in swallowing gavage may be employed. Hoffa[1] advises keeping the stomach-tube constantly in place. As soon as the infant is able to suck feeding should be directly from the breast, if possible.

In the cases where substitute-feeding is absolutely unavoidable, is necessary to determine what artificial food will answer best. Its strength in all the elements must be very weak at first, since the power of digestion is but feeble. The direction in which an increase in the strength of the food shall be made can be determined only by observing the results upon digestion, as shown by the character of the stools, the frequency of vomiting, and the like. According to the individual case we may employ whey-mixtures, buttermilk, food consisting chiefly of dextrin-maltose, or peptonized milk. It would ordinarily seem best to have the percentage of fat low at first and to increase it with great caution. Yet in some cases, where the amount of food ingested is small, concentrated foods are indicated and are often remarkably well tolerated. For this purpose we may employ the butter-flour mixture of Czerny-Kleinschmidt or of Moro, lactic acid milk with corn-syrup, or the concentrated mixture employed by Schick (see Concentrated Feeding, p. 149). As soon as needed the strength of the food in general and the amount given may be increased; but with care against over-feeding. The infant should be weighed daily, guarding against chilling by exposure, since it is principally by the weight and the general satisfactory appearance that we can determine that it is thriving. Failure to gain properly may mean that more food is required. Sometimes, if there is indigestion, it may be an indication to reduce the amount.

The basal metabolism of premature infants has been studied by Talbot, Sisson, Moriarity and Dalrymple[2] and by Murlin and Marsh,[3] and has been found to be strikingly low, because there is a very small amount of active heat-forming tissue. Regarding the quantity of food needed and the frequency of administration there can be no absolute rule. A large excess in caloric intake over basal metabolism is necessary, because of the relatively greater amount of growth essential in these babies. The caloric needs of the premature infant are probably from 110 to 130 calories per kilogram (50 to 59 per lb.) daily, exceeding those of the full-term child. Some investigators place them as low as 100 calories per kilogram (45 per lb.) daily and others as high as 170 per kilogram (77 per lb.). The relatively high caloric need is perhaps the result of the greater body-surface as compared with the body-weight. The increased need is not, however, found in the 1st week, and perhaps not until 1 or 2 weeks have passed. Indeed in the first 2 or 3 days we may well be content with a sugar-solution. Benedict and Talbot[4] estimate that the energy requirements of the full-term infant during the first 6 days are only 62 calories per kilogram (28 per lb.). It is probable that the premature infant requires no more or even less than this during this period. The amount of nourishment taken in the early weeks is very small, varying with the weight of the premature infant (125 to 345 c.c.) (4.23 to 11.67 fl. oz.) in the first 10 days, Délestre).[5] The best plan is to calculate the number of calories probably required, and to give a daily amount of food

[1] Arch. f. Kinderheilk., 1922, LXXII, 6.
[2] Amer. Jour. Dis. Child., 1922, XXIV, 95.
[3] Proc. Soc. Exper. Biol. and Med., 1922, XIX, 431.
[4] Carnegie Instit. Wash., 1915, Publication 233.
[5] Thèse de Paris, 1901.

which will supply these; using the calculation, however, only as a guide, and allowing especially the progress of the infant in weight and the condition of its digestion to determine the increase. The frequency of feeding is to be based upon the amount which the infant can take and retain at a time. If it refuses to swallow more than 2 or 3 drams (7 or 11), or vomits if more is forced upon it, feeding every 1 to 2 hours may be necessary. If it can ingest a proper amount at one time, intervals of 2½ to 3 hours will be chosen. In general we may aim for a quantity of from ½ to 2 ounces (15 to 57) every 2½ to 3 hours in the early weeks, depending upon the weight, length, and age of the infant. Vomiting resulting from the administration of too much food is to be avoided as far as possible, on account of the danger of the regurgitated milk entering the respiratory passages and producing asphyxia or aspiration-pneumonia.

The treatment of the premature infant is in other respects symptomatic. The relief of atelectasis and cyanosis is to be obtained by reflex stimulation, as by plunging into a hot bath of 100 to 105°F. (37.8° to 40.6°C.), preferably containing mustard, in order to produce crying. The inhalation of oxygen is often of service, and a retort of the gas should be close at hand for immediate use when required. Infection is to be very carefully avoided, especially by great attention to the care of the navel, and the employment of sterile water for any washing necessary. In the after-care of premature infants it is well to remember the tendency to anemia and rickets, and both of these conditions should be treated prophylactically. Iron may be given as ferric citrate, ¼ grain (.016) a day dissolved in orange-juice, or as syrup of ferrous iodide, 5 to 10 minims (.31 to .62) a day. As the infant's age and condition may not permit sufficient sunlight, 5 to 10 minims (.31 to .62) or more of cod liver oil must be administered daily. To supply the deficiency in mineral matter Neubauer[1] employs whey in addition to breast-milk.

CHAPTER II

SEPSIS IN THE NEW-BORN

The septic infections of the new-born include a number of conditions which clearly are due to the entrance of pyogenic germs into the organism. Many of these exhibit also such special peculiarities or localization that they may be described as distinct diseases. Still others are possibly septic, yet are of an origin not clearly understood. General septic infection as it occurs in the new-born is the topic now under consideration.

Etiology and Pathological Anatomy.—Sepsis is still too common, but its incidence is trifling as compared with the former extreme prevalence of the affection. The opportunity for infection is frequent. Hymanson and Hertz[2] claim that 100 per cent. of infants exhibit bacteria in their mouths immediately after birth. In rare instances septic infection may take place before birth. In this event the fetus generally is born dead. The cause in such cases may be a penetration of bacteria or of toxic substances from the mother through the placenta, the mother

[1] Monatsschr. f. Kinderheilk., 1921, XXI, 21.

[2] Amer. Jour. Obstet., 1917, LXXV, 662.

herself being ill, often with sepsis. In some instances the infection is through the amniotic fluid, in not a small number of cases depending upon a maternal perityphilitic inflammation (Hellendall)[1]. In sepsis acquired in this way the primary lesion is a pneumonia. More often sepsis develops through injuries received during or after birth, as through abrasions of the skin or by the entrance of septic matter derived from the discharges of the mother into the mouth, stomach, rectum, vagina or lungs of the infant. The water used for washing the infant may be the source of the infection. Hemolytic streptococci are almost constantly present on the nipple of the nursing mother (Clauss).[2] Operations performed on the new-born, such, for instance, as circumcision, the opening of a cephalhematoma or the incision of the frænulum linguæ for tongue-tie may serve as portals of entry. Although the possibility of the development of sepsis through such sources is to be recognized, in the great majority of instances it starts at the umbilicus, beginning oftenest before complete separation of the cord, and is first manifested by an umbilical arteritis. In 340 autopsies in new-born infants Runge[3] found 36 cases of general sepsis. In 30 of these infection could be traced to the navel, and in all of these umbilical arteritis was present. The fact that the umbilical wound is healed is no proof that infection of the umbilical vessels does not exist.

Beginning at the portal of entry, wherever this may be, the infection may either remain local, or may become more general, there occurring in the new-born but little protecting lymphadenitis to prevent this; or general septic infection may occasionally take place without the portal of entry showing anything abnormal or even being discoverable. If the sepsis is local only, there may be produced phlegmonous inflammation of the injured skin of various parts of the body, mammitis, omphalitis, thrombosis of the umbilical arteries or vein, stomatitis, rhinitis, etc.; or in cases of aspiration of an infectious fluid through the mouth, septic pneumonia. If the process extends from one of these portals of entry the sepsis may involve almost any of the more distant parts of the body.

In infants still-born or dying very shortly after birth, the subjects of intra-uterine infection, there is found macerated skin, petechiæ on the surface of the body and on the serous membranes, bloody or serous effusion into the serous cavities, and smaller or larger extravasations of blood into and fatty degeneration of the internal organs. In those acquiring the disease after birth there is the same tendency to fatty degeneration of and hemorrhages into the internal organs, with petechiæ of the serous membranes, and bloody, serous, or purulent effusion into the serous cavities. There are also various lesions of different organs, depending upon the localization of the septic process. Septic thrombi are frequent, especially of the umbilical arteries. Inflammation or hemorrhage of the brain or meninges is also common, as are cutaneous or subcutaneous abscesses, peritonitis, pericarditis, pneumonia and pleurisy. Müller[4] regards peritonitis as one of the frequently observed localizations in general septic infection. Bednar[5] found croupous pneumonia 15 times and pleurisy 10 times in the autopsies on 87 cases of sepsis. Runge[6] recorded pneumonia in 22 out of 55 cases of umbilical arteritis. Involvement of the digestive apparatus is frequent. Osteomyelitis, arthritis, nephritis, pyelitis, and otitis are often observed.

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[1] Beiträge z. Geburtsh. u. Gynäk., 1906, X, 320.
[2] Zeitschr. f. Geburtsh. u. Gynäk., 1921, LXXXIV, 385.
[3] Krankh. d. ersten Lebenstage, 1893, 136.
[4] Gerhardt's Handb. d. Kinderkr., 1877, II, 177.
[5] Krankheit. d. Neugeb. u. Säugling, 1850, IV, 245.
[6] *Loc. cit.*, 94.

The ultimate cause of the pyogenic infection appears to be chiefly varieties of the staphylococcus and streptococcus. The pneumococcus, colon bacillus, bacillus pyocyaneus, gonococcus, bacillus proteus, and others are also factors in some instances. The germs, as stated, are acquired in many ways. The vaginal discharges are a fruitful starting point for the cases developed during birth. Later there is no dearth of sources from which infection can arise, all depending upon lack of aseptic cleanliness of some form. All new-born infants are prone to the development of the disease, but it is particularly common among those born in institutions, since the dangers of infection are greater there. It is disputed whether infection may take place through the milk of the mother.

Symptoms.—The symptoms usually appear either immediately after birth or at some time within the first 10 days. They vary greatly, depending upon the seat of the lesions. As a rule, however, in general septic infection there is great and characteristic depression of strength with rapid loss of weight and entire anorexia. Fever is irregular. It is generally present at the onset and frequently high, but may be absent throughout and the temperature is often subnormal toward the end. Severe diarrhea is a common symptom; vomiting may occur; icterus is frequent and often intense, or the skin may be of a pale-grey tint, and septic erythemata are frequently observed. The pulse is rapid and weak, the respiration irregular; sometimes deep and rapid. The occurrence of small or larger hemorrhages in different parts of the body is a common and characteristic symptom. The child looks ill, is usually apathetic or somnolent, has a feeble cry and sometimes develops the evidences of collapse. Various nervous symptoms may be seen, among them tossing, rolling of the head, hypertonic states, twitching of the muscles, tremor, and sometimes convulsions. The urine generally contains albumin. Enlargement of the spleen may be discovered in some cases. The coagulation-time of the blood is increased.

When the disease is prolonged the symptoms may be masked, the principal one being a rapid, continuous loss of weight, until finally the development of some local septic process makes the condition clear. Naturally in many instances the character of the symptoms is modified by the development of those of localized involvement as well.

Course and Prognosis.—Some cases run a rapid course, ending fatally in from 1 to 3 days. Others last for weeks; but in all the prognosis is very unfavorable. The mildest and more slowly progressing cases may occasionally recover, but this is certainly unusual, and all those with well-marked symptoms of a general infection terminate fatally. The earlier the infection occurs and the more wide-spread the lesions, the worse the prognosis.

Diagnosis.—The diagnosis offers no difficulty where the symptoms are well-developed, and a source of infection can be recognized. It is often impossible when no portal of entry can be found which shows evidence of septic infection, or unless some local metastatic manifestation of sepsis develops. The discovery of enlargement of the spleen is corroborative but not positive evidence. The absence of fever does not exclude sepsis, and the failure of a leucocytosis to appear is not conclusive. The only positive evidence may be the discovery of septic germs in the blood.

Treatment.—This consists principally in prophylaxis. Every possible source of infection is to be removed. Should the mother be suffering from puerperal fever, the child must be separated absolutely from her. Any discoverable wound upon the skin or mucous membrane of the child must receive as careful antiseptic treatment as possible. The umbilicus (see p. 68) and the mouth should have special attention. For the disease when once developed little can be done, except the employment of vigorous stimulating and supporting measures, the prevention of dehydration by the employment of hypodermoclysis, and the adoption of such surgical treatment for local conditions as seems indicated.

CHAPTER III

ACUTE FATTY DEGENERATION OF THE NEW-BORN

(Buhl's Disease)

This condition was first described by Buhl[1] in 1861. Cases have been reported by a few others, notably Hecker[2] and Runge.[3]

Etiology.—The disease is a very uncommon one, or at least seldom recognized in human beings, although a similar condition in the new-born of animals has been seen more frequently. The cause is entirely unknown. Although it is very probable that it is of a septic nature, this has not been proven, and has, indeed, been strongly contested. In a typical case recorded by Lucksch,[4] colon bacilli were found in the blood. Elizalde and Puente,[5] who describe the occurrence of acute fatty degeneration in a full-term infant born after a prolonged labor, believe that the ether given to the mother and the deficient oxygenation of the fetus play a part in the etiology. The disease occurs in isolated cases only, and has been seen especially in well-developed infants who have been born much asphyxiated without discoverable cause, and have continued so to some extent in spite of treatment.

Pathological Anatomy.—The body is cyanotic and frequently icteric also. The skin is often edematous and may exhibit hemorrhages. Effusions of blood, either ecchymotic in character or in large amounts, are found in nearly all the internal organs; especially from or in the serous membranes of the brain, the pleura and pericardium, the endocardium, the mediastinal connective tissue and the peritoneum. They occur also in the muscles, the thymus gland, and most of the mucous membranes. The brain is soft and congested; the lungs often contain infarcts and the bronchi bloody mucus. The heart-muscle is firm and dark-red in cases dying promptly; pale and soft in those of longer duration. The spleen is generally enlarged and very soft. The liver is at first dark-red; later pale, icteric and perhaps slightly enlarged. The stomach and intestines often contain effused blood and there are hemorrhages in the mucous membrane, and the walls are thickened and edematous. The kidneys are swollen and dark-red in the early stages, but later pale and

[1] Klinik d. Geburtsk., 1861, I, 296. Ref. Runge, 162.
[2] Arch. f. Gynäk., 1876, X, 537.
[3] Krankh. d. ersten Lebenst., 1893, 162.
[4] Prag. med. Wochenschr., 1913, XXXVIII, 167.
[5] Rev. di la Assoc. Med. Argentina, 1921, XXXIV, 10.

yellowish. They exhibit numerous hemorrhages into their substance, and often a complete choking of many of the tubules with granular fatty matter. The umbilicus and its vessels are normal, except for the evidences of the presence of hemorrhage which has occurred from it and in the tissues about it.

The change most characteristic of the disease is the marked fatty degeneration of the liver, heart, kidneys and epithelium of the pulmonary alveoli This does not necessarily involve in each case all of the organs mentioned, but may in some of them be entirely absent or be replaced by a parenchymatous inflammation. The degenerative lesions are similar to those produced by phosphorus-poisoning.

Symptoms and Course.—The asphyxia with which these children are born does not yield to treatment, and the majority of the cases die promptly. If life is maintained longer there develops diarrhea often with bloody or blackish stools (melena), frequent vomiting of blood, and hemorrhage from the navel after the separation of the cord, this being generally in small amount, but sometimes large enough to be fatal. The hemorrhagic symptoms appear about the 5th day of life. The cyanotic color of the skin persists, becomes combined with or gives place to icterus, and edema is seen in some instances. Bleeding may occur into the skin or from the mucous membrane of the mouth or nose, conjunctiva or ear. There is anemia, great prostration, rapid loss of weight, and collapse. Fever is absent or inconsiderable. Not always are all the symptoms present. The asphyxia may be followed by sudden death, or may in some cases be slight at first and then may increase rapidly, accompanied by the characteristic symptoms. In some instances no external hemorrhage whatever is seen. The disease may last but a few hours before the fatal ending occurs. It rarely continues as long as 2 weeks. The **prognosis** is entirely unfavorable. Whether lighter forms recover cannot be known, since in these the determining diagnostic feature, the fatty degeneration, is necessarily undiscoverable.

Diagnosis.—An absolute diagnosis is impossible except by microscopic examination of the tissues. Even a probable diagnosis is difficult unless all the symptoms are present, especially the combination of marked asphyxia and wide-spread hemorrhage, and the absence of any umbilical disease. The disorder resembles closely and probably is identical with some forms of sepsis of the new-born. Infectious hemoglobinemia is like it in many particulars, but is distinguished by the characteristic condition of the urine present in that affection. In the case of infants who die asphyxiated soon after birth it is impossible to determine without autopsy whether acute fatty degeneration was not the disease present; and this applies, too, to those born little or not at all asphyxiated, who later suddenly develop this symptom and die promptly. The importance of the microscopic examination in all doubtful cases is, therefore, evident.

The **treatment** can be only symptomatic, efforts being directed to the sustaining of strength, the relief of the asphyxia, and the control of the hemorrhage.

CHAPTER IV

ACUTE INFECTIOUS HEMOGLOBINEMIA OF THE NEW-BORN

(Winckel's Disease; Hæmoglobinuria neonatorum; Cyanosis afebrilis icterica perniciosa cum hæmoglobinuria; etc.)

Although Winckel[1] recognized the existence of hemoglobinuria as a symptom in an epidemic affecting 23 children in the Lying-In Hospital of Dresden, yet an earlier epidemic in which 10 children were attacked was well described by Bigelow[2] in 1875. A condition probably the same had already been reported by Parrot[3] and by Charrin[4] in 1873, and still earlier by Pollak[5] in 1871.

Etiology.—The affection is a rare one. It occurs generally in institutions and in an epidemic form, although isolated cases have occasionally been reported, and it attacks well-developed children equally with others. It appears closely allied both to the septic infection and to the hemorrhagic disease of the new-born, and is also very closely related to acute fatty degeneration of the new-born (p. 294). That it is an infection seems certain, yet the nature of this is unknown; for although both streptococci and colon bacilli have been reported, these are germs commonly found in sepsis, which is without any such characteristic group of symptoms. That it is not produced by the ingestion of a poison was proven by Winckel's investigations.

Pathological Anatomy.—The lesions as based upon Winckel's description show the internal organs as well as the skin cyanotic and icteric in hue. The spleen is much enlarged and hard, of a blackish-red color, and contains a large amount of brown coloring matter, partly free, partly in the cells of the pulp. The cortical layer of the kidney is thicker than normal, of a brownish color, and exhibits small hemorrhages. The pyramids are blackish-red and show narrow black streaks converging toward the papillæ and due to the deposit of hemoglobin in the canals. The bladder contains greenish-brown urine. The liver is enlarged; the mesenteric glands and Peyer's patches swollen. Punctate hemorrhages are seen in most of the organs of the body, as in the membranes of the brain and spinal cord, under the serous covering of the liver, and especially in the pleura, endocardium and pericardium, and in the mucous membrane of the stomach and intestines. There is sometimes a fatty degeneration of the liver and heart-muscle. The umbilical vessels are nearly always normal. Bacteria of any sort have usually not been discovered.

Symptoms.—The disease begins usually on from the 4th to the 8th day of life, occasionally earlier or later. The earliest symptoms are restlessness, loss of appetite, prostration, and intense cyanotic discoloration of the entire surface. Well-developed icterus is promptly combined with this, and rapidly increases in intensity until the surface is of a bronze color. The temperature is normal or below, rarely slightly elevated. The respiration is generally accelerated, the pulse little if at all altered. The urine is pale-brown in color and voided frequently in small amounts with straining. Examination shows the presence of hemoglobin, granular casts and renal epithelium, urate of ammonium, micrococci, and a small amount of albumin. No bile or biliary acids are present in it. The stools vary in color from dark-green to yellowish or brown. Vomiting and diarrhea occasionally occur. On incising or scratching the skin over the most cyanotic regions a thick, syrupy fluid of a blackish-brown color exudes, but only on firm pressure. The blood shows an increase in the number of leukocytes, numerous granules, and a great diminution in the number and increase in the size of the red blood-cells, many of which appear to have lost their coloring matter. Collapse develops with great rapidity; there is somnolence, and convulsions are liable to terminate the case.

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[1] Deutsch. med. Wochenschr., 1879, V, 303.
[2] Bost. Med. and Surg. Jour., 1875, XV, 277.
[3] Arch. de phys. norm. et path., 1873, 512.
[4] Thèse de Paris, 1873.
[5] Wien. med. Presse, 1871, 457.

Prognosis.—This is most unfavorable, as all severe cases die. Only 2 of Bigelow's and 2 of Pollak's cases recovered; while 19 of Winckel's 23 cases are known to have ended fatally, and only 1 certainly to have recovered. Ljwow[1] reported better results, 4 out of 7 cases recovering. Death usually occurs in a few hours to 4 days, generally in 2 days.

Diagnosis.—The disease is so characteristic that mistakes in diagnosis can hardly be made. Although it resembles strongly in some respects acute fatty degeneration of the new-born, it is to be distinguished by the presence of hemoglobinuria and the occurrence usually in epidemics.

The **treatment** can be only symptomatic, efforts being made to maintain life by stimulants and food.

CHAPTER V

HEMORRHAGE IN THE NEW-BORN

Hemorrhages at this early period of life, although not common, are still frequent as compared with their presence later in childhood. They may occur in different parts of the body and from different causes. Those not mentioned here will be found in other chapters.

Etiology.—The previous general health seems to have no constant influence, for while some of the infants affected have previously been in poor condition others appear strong and hearty. In some instances the hemorrhages, usually single, seem to be the result of accident, as in hematoma of the scalp (p. 303) or of the sternocleideomastoid (p. 306), and in some cases of hemorrhage from the umbilicus (p. 329) or from some other part of the body as a result of trauma received. After difficult and prolonged labor various visceral hemorrhages may occur, particularly those within the cranium. Spencer[2] concludes from 130 autopsies that the use of the forceps is a common producer of intracranial hemorrhage

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[1] Medicinskoje Obosrenje, 1893, No. 14. Ref. Jahrb. f. Kinderheilk., 1894, XXXVIII, 497.

[2] Trans. Obst. Soc., Lond., 1891, XXXIII, 203.

Premature birth is another cause of hemorrhage, especially within the cranium. Difficult breech-presentations may produce intra-cranial bleeding or occasion hemorrhages of the abdominal viscera. In very many instances the bleeding depends upon septic infection, as already pointed out, and bacteria of various sorts have been found in the blood. (See p. 292.) In these cases the hemorrhages are liable to be wide-spread and of small size. In others special hemorrhage-producing bacteria have been found (Schloss and Commiskey).[1] Congenital syphilis appears to be the active agent in some instances. The influence of this factor has been reviewed by Hess[2] and by Pontoppidan.[3] Wilson[4] reported hemorrhage occurring in 45 of 3364 new-born infants, and in 10 of these it appeared to be due to congenital syphilis. In many others we are ignorant of the exact etiology and pathology, although the presence of some infection or toxemia seems probable. Either the resistance of the blood-vessel walls is weakened in some way, or the blood itself is altered. In regard to the first factor Ylppö[5] observed that in new-born infants, and especially in those who were premature, negative pressure by a suction cup easily produced subcutaneous hemorrhage. Studies made by Schloss and Commiskey[6] upon the coagulability of the blood in 10 cases of hemorrhagic disease found this diminished in some instances, but unaltered in others. Many of the forms of hemorrhage will be referred to later in discussing the diseases of the organs in which the bleeding occurs.

Bleeding which takes place without any evidence of trauma may be called "spontaneous." Townsend[7] has applied the term **"The hemorrhagic disease of the new-born"** to the condition in which small or large effusions of blood occur simultaneously in many different parts of the body, independent of any discoverable cause. It was earlier described by Minot[8] in 1852. A great deal has been written recently about the disorder. Statistics indicate its frequency and it must especially be considered as one of the causes of intracranial hemorrhage (see Vol. II pp. 602, 658) and of gastro-intestinal hemorrhage (see p. 301) in early life In the 1st week of life there is normally a prolonged coagulation-time and bleeding-time, and in the hemorrhagic disease the prolongation is somewhat greater (see p. 53). The delay in coagulation is claimed to be due to diminution in the prothrombin element (Lucas et al).[9] Although this prolonged coagulation-time is often found in hemorrhages of different localization, it may be present without evidence of hemorrhage. Certainly, however, when trauma is added to the factor of undue delay in coagulation, bleeding may be expected to occur rather readily. This early spontaneous hemorrhage is quite distinct from hemophilia (Vol. II, p. 784), among other respects in that the tendency of the new-born to bleed does not persist if the child survive. Hemorrhage in the new-born occurs much most frequently in institutions. Ritter[10] found 190 such in 13,000 infants in the Prague Foundling Hospital; *i.e.* 1.46 per cent., and Townsend[11] 32 cases in 7225 births; *i.e.* 0.44 per cent.

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[1] Amer. Jour. Dis. Child., 1911, I, 276.
[2] Arch. of Pediat., 1904, XXI, 598.
[3] Hospitalstidende, 1916, LIX, 626. Ref. Brit. Jour. Child. Dis., 1916, XIII, 308.
[4] Arch. of Pediat., 1905, XXII, 43.
[5] Zeitschr. f. Kinderheilk., 1924, XXXVIII, 32.
[6] Amer. Jour. Dis. Child., 1912, III, 216.
[7] Bost. Med. and Surg. Jour., 1891, CXXV, 218; Arch. of Pediat., 1894, XI, 559.
[8] Amer. Jour. Med. Sci., 1852, XXIV, 310.
[9] Amer. Jour. Dis. Child., 1921, XXII, 525.
[10] Oesterreich. Jahrb. f. Padiatrik, 1871, I, 127.
[11] *Loc. cit.*, 218.

The accident occurs much the most frequently in the 1st or the 2d week of life. Abt[1] estimates that from 1 in 500 to 1 in 700 institution-infants suffer from hemorrhage. Cruickshank[2] found hemorrhage of some degree in 77 per cent. of 200 full-term infants. If intracranial hemorrhage is included in the hemorrhagic disease, the percentage-incidence is greatly increased (see Vol. II, p. 601).

Locality of the Lesions.—The regions of the body affected are various and generally are multiple. One of the commonest seats is the gastroenteric tract. In Ritter's[3] 190 cases hemorrhage occurred from the intestine in 39, the mouth in 28, and the stomach in 20. (See Melena, p. 301.) The flow of blood is generally small, but is often in larger amount and discharged from the bowels as a black, tarry substance. Bleeding may take place into other of the abdominal organs, due generally to difficult labor when the hemorrhage is single, but oftener the result of the hemorrhagic disease, sepsis, or other causes when the hemorrhage is multiple. Large effusion into the suprarenal capsule is one of the most frequent forms (Vol. II, p. 850). Small hemorrhages under the peritoneum are seen not infrequently. Larger ones also may occur here, being commonly traumatic in origin. These and especially those into the suprarenal bodies may burst into the peritoneal cavity and produce sudden death from collapse. In some such instances it may be impossible to discover the original source. Occasionally bleeding may take place from the liver (Bonnaire and Durante).[4] Intracranial hemorrhage is one of the commoner varieties. (See Vol. II, p. 601.) The numerous instances of cerebral birth-palsy are generally dependent upon intracranial bleeding. (See Cerebral Palsy, Vol. II, p. 657.)

Small hemorrhages may occur upon the serous covering of the lungs. More rarely larger ones take place into the pulmonary tissue, the bronchi, or the pleural cavity. Epistaxis is uncommon. Small effusions of blood may be found in the thymus gland. Hemorrhage, generally in small amount, may occasionally arise from the female genitals. Schukowski[5] collected only 35 cases of metrorrhagia in 10,000 new-born female infants. (See also Vol. II, p. 503.) Bleeding from the bladder or kidneys may exceptionally occur. This may be the result of a general hemorrhagic disease or may be purely traumatic in origin, dependent upon the presence of uric-acid infarcts. Hemorrhage may take place from the ears or the eyelids. Hemorrhage into the spinal cord, probably traumatic in origin, has been reported by Spencer,[6] Burr[7] and Kohlbry.[8] We have seen it in at least 3 instances.

Bleeding from the umbilicus is the most common variety, 132 (69.47 per cent.) of Ritter's[9] 190 cases being of this nature, and in 97 the hemorrhage being limited to this region. It may occur as a result of accident or as a manifestation of a general hemorrhagic condition (see Umbilical Hemorrhage, p. 329), and is very often combined with bleeding from other parts of the body. Wide-spread subcutaneous hemorrhages are not uncommon. They develop oftenest in parts most pressed upon, although other regions are not spared. Bleeding from the skin following a slight wound, as for an examination of the blood, is not infrequent in hemorrhagic cases.

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[1] Jour. Amer. Med., Assoc., 1903, XL, 284.

[2] Lancet, 1923, I, 836.

[3] *Loc. cit.*, 159.

[4] L'Obstétrique, 1911, IV, 825.

[5] Spareda Vop., 1902, H. 3., Ref. Jahrb. f. Kinderheilk., 1903, LVII, 105.

[6] *Loc. cit.*

[7] Amer. Jour. Dis. Child., 1920, XIX, 473.

[8] Amer. Jour. Dis. Child., 1923, XXVI, 242.

[9] *Loc. cit.*, 190.

Symptoms.—The *traumatic* hemorrhages, due as they generally are to injury at birth, as a rule reveal themselves promptly, except those of the abdominal and thoracic viscera, where no symptoms at all may appear until, perhaps, a sudden collapse occurs followed promptly by death. Those of a *spontaneous* nature manifest themselves generally by visible bleeding in several regions of the body. Inasmuch as infants tolerate loss of blood very badly, a comparatively small hemorrhage may readily produce weakness of pulse, great depression of strength and loss of weight. Anemia is a natural result. The temperature may be elevated, but is often subnormal. Icterus is a common symptom. It was seen in 21 per cent. of Ritter's cases. As a rule the amount of blood lost at one time is small. It is the repeated losses, and especially losses in many parts of the body, which occasion the general symptoms in most instances.

Prognosis and Course.—These depend much on the cause, on the amount of blood lost, and on the duration of the process. The mortality was formerly high; 75.79 per cent. in Ritter's cases of spontaneous hemorrhage and 62 per cent. in Townsend's 50 cases; while in 609 published reports of hemorrhage collected by the latter writer the mortality was 79 per cent. Of recent years improved methods of treatment have decidedly diminished the death-rate. Death in the fatal cases generally occurs within a week. It may be very sudden from collapse when the hemorrhage is large.

Diagnosis.—Where the bleeding is evident to the eye the diagnosis is easy. Where it is concealed it is difficult and often impossible. The determining of the nature of the cause is important. As a general rule large single hemorrhages are the result of local processes, while wide-spread multiple hemorrhages depend upon sepsis, syphilis, or the general hemorrhagic condition referred to. Of course, the discovery of blood effused in or from only one part of the body does not prove that concealed visceral bleeding may not be taking place as well.

Treatment.—Local measures to arrest bleeding are indicated where applicable. Among these are the use of astringents, especially the application of a caustic, such as nitrate of silver in solid form, liquor ferri subsulphatis or chromic acid. The exhibition of gelatine in 10 per cent. solution administered internally freely has been largely employed, and appears to be of value. From 10 to 50 c.c. (0.34 to 1.69 fl.oz.) may be given subcutaneously once or twice a day with the greatest caution to obtain complete sterilization and to follow aseptic precautions, lest septic infection or even tetanus result. Internally gelatine may be administered either by the mouth or by the rectum. Adrenaline chloride has been recommended, giving 1 to 4 minims (0.062 to 0.246) of a 1:1000 solution hypodermically. It may also be used locally. Calcium chloride or lactate in amounts of 20 to 40 grains (1.3 to 2.6) in 24 hours in divided doses has also been employed, although theoretically it can be expected to do little good, since the calcium-content of the blood of the new-born is usually high (Lucas, et al).[1] One of the best of remedies is the administration of a blood-serum. Human blood from a parent may be given by direct transfusion (see p. 276), or intraperitoneally (see p. 277), or this or the serum be injected subcutaneously, as recommended by J. E. Welch[1] in quantities of from 10 to 30 c.c. (0.34 to 1.01 fl. oz.) every 4 to 8 hours. The umbilical vein is patent up to the 4th day of life, and when the stump of the cord is cut proximal to the place at which it has been tied off, it may be used as the vessel for transfusion. The needle of the injecting syringe is inserted into the vein (the largest of the 3 vessels in the cord) and is held in place by tying a ligature above the cord (Sidbury).[2] To prevent clotting the blood before injection may be mixed with a 2 per cent. sterilized sodium-citrate solution in the proportions of 1:10. (See p. 276.) Should human blood not be available, horse-serum or rabbit-serum may be given subcutaneously in the same amount, diphtheria-antitoxin being selected for this purpose if nothing else can be obtained. In the way of general treatment the loss of blood must be made up by subcutaneous, intraperitoneal or rectal injections of normal salt-solution, the temperature maintained, and the strength supported. (See also Vol. II, p. 601, for Intracranial Hemorrhage.)

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[1] Amer. Jour. Dis. Child., 1921, XXII, 525

MELÆNA NEONATORUM

In this form of hemorrhage in the new-born are grouped cases which are quite dissimilar in origin and nature, but which show the characteristic symptom, *i.e.*, the discharge of black, altered blood (μέλαινα: black) from the intestines or, by vomiting, from the stomach. The condition was first described by Ebart[3] in 1723. The title should be reserved to describe the symptom merely; not the cause.

Etiology.—The affection is sometimes divided into *Melœna spuria* and *Melœna vera.* In the former the blood enters the infant's stomach or intestines from outside sources, as from a hemorrhage from the mouth, nose or lungs, a wound in the nipple of the nurse, or from the swallowing of maternal blood during labor. In melæna vera, which is the form about to be considered, it comes from the effusion of blood into the stomach or intestines. Even the cases of the latter group may be divided into the *symptomatic,* in which other manifestations, often hemorrhagic, of a constitutional disease are present, and the *idiopathic,* in whcih the melena is the only symptom of a hemorrhagic condition observed.

Melena is a comparatively uncommon disorder, occurring about once in every 1000 or 2000 births, according to different estimations (Gerhardt-Seifert).[4] The etiology is not clearly understood, and certainly varies with the case. Lesions of the mucous membrane or of the deeper vessels of the gastroenteric canal are prominent causes, but the method of production of these lesions is far from clear. Often they are the result of congestion, which may itself be brought about in various ways incident to birth, such as prolonged asphyxia, violent efforts at extracting the child or at its resuscitation, compression of the umbilical cord, congenital diseases of the heart, congenital syphilitic hepatitis, and the like. In other instances some constitutional condition has produced a weakness of the vessels or an alteration of the blood itself; among these causes being syphilis and sepsis, the melena being then only one of the evidences of the hemorrhagic tendency existing. A delay in the coagulation of the blood is said by Lövegren[5] to be present, but Bernheim-Karrer[6] observed normal coagulation-time as well as normal platelet-count. Various bacteria have been reported as found from time to time but their relationship to melena has never been satisfactorily proven. It is probably very close in some instances but of no bearing in others. The influence of the development of ulcers in the gastroenteric tract, especially the duodenum, in giving rise to gastrointestinal hemorrhage is easy to appreciate, but the method of production of such ulcers is not well understood; whether embolic, or dependent on erosion after hemorrhage into the mucous membrane. (See Ulcer of the Duodenum, Vol. II, p. 170.) Müller[1] in 5 fatal cases of melena found 1 with ulcer of the esophagus and was able to collect 4 others from the literature. Ulceration is not a common post-mortem finding. Kennedy,[2] however, considers minute duodenal ulcers, due to bacterial invasion, a common cause. Vorpahl[3] reported an instance of melena dependent upon rupture of dilated veins in the esophagus. Often it would appear as though the blood makes its way from the vessels through the mucous membrane of the intestine without any discoverable lesion.

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[1] Amer. Jour. Med. Sci., 1910, CXXXIX, 800.

[2] Amer. Jour. Dis. Child., 1923, XXV, 290.

[3] Widerhofer, Gerhardt's Handb. d. Kinderkr., IV, 2, 408.

[4] Lehrb. d. Kinderkr., 1897, 71.

[5] Jahrb. f. Kinderheilk., 1914, LXXIX, 708.

[6] Zeitschr. f. Kinderheilk., 1921, XXVIII, 335.

Pathological Anatomy.—The surface of the body and the internal organs are anemic, and the gastrointestinal canal filled with blackish fluid. Beyond these there is no uniformity in the conditions present. Occasionally ulcers are found in the stomach or intestine, especially the duodenum. They may be very minute or larger, and sometimes exhibit the characteristic appearance of the ordinary peptic ulcer. In some cases only minute erosions and in others only minute extravasations of blood are found. In the large majority nothing whatever of moment is discovered. Careful study of other parts of the body may reveal the evidences of sepsis or of syphilis, or the characteristic lesions of acute fatty degeneration.

Symptoms.—Although in a certain proportion of cases there is some asphyxia at birth, in the large majority the child is of healthy appearance. The discharge of blood begins usually on the 2d day of life and seldom later than the 4th day. In most cases the blackish or blackish-red matter is vomited and also evacuated by the bowel, but in many instances it is passed from the bowel only. In an analysis by Vassmer[4] of 67 published cases blood was found in the stools alone in 20, in the vomitus alone in 6, and in both in 37. The amount lost may be slight, but it is oftener so large that the infant rapidly becomes very anemic, apathetic, prostrated, with feeble cry, and finally collapsed. There may be slight transitory fever, or normal or subnormal temperature. As a rule there is no distention or tenderness of the abdomen. The disease seldom lasts longer than 2 to 3 days. In the cases which survive recovery is rapid, except from the anemia, unless some constitutional disorder, such as syphilis, is accountable for the hemorrhage and keeps the child ill in other ways. The disappearance of the anemia, of course, requires a longer time.

Prognosis.—The death-rate is high, probably between 50 per cent. and 60 per cent. in untreated cases, (56 per cent. Silbermann).[5] In the cases of idiopathic melaena vera; *i.e.* those in which the hemorrhage is limited to the gastrointestinal tract and is not a symptom of a general hemorrhagic diathesis, the mortality is distinctly less than the figures given. The mortality is less also in the cases where the hemorrhage is

[1] Zeitschr. f. Kinderheilk., 1921, XXX, 234.
[2] Amer. Jour. Dis. Child., 1926, XXXI, 631.
[3] Arch. f. Gynäk., 1912, XCVI, 377.
[4] Arch. f. Gynäk., 1909, LXXXIX, 275.
[5] Jahrb. f. Kinderheilk., 1877, XI, 378.

from the bowel only than when there is hematemesis. Thus in the 20 cases of Vassmer's series in which the hemorrhage was from the bowel alone the mortality was 10 per cent. The liability of death depends largely on the cause of the bleeding and the amount of it, and upon the promptness of treatment.

Diagnosis.—Melaena vera is to be sharply distinguished from the spurious melena referred to. In the latter the hemorrhage is seldom large; it does not always begin so early in life; and the child does not, as a rule, appear ill. Examination of the nipple of the mother and of the nose and mouth of the child may discover the source. When melena is limited to discharge of blood from the intestines it may be readily confounded with the passage of meconium, especially in mild cases, and be easily overlooked. The material is, however, evacuated more frequently in cases of melena, and the slightly reddish color of the blackish masses or of the diaper serves to distinguish it. The use of the microscope, especially the combined chemical and microscopical examination for blood-corpuscles and hemin-crystals, makes the diagnosis positive. Finally, the presence of hemorrhages from other parts of the body or of symptoms other than those described will remove the case at least from the category of idiopathic melaena vera. Occasionally the gastrointestinal hemorrhage is concealed, and the child dies without any external bleeding having taken place. In such instances an antemortem diagnosis is not possible.

Treatment.—This consists in supporting the strength and checking the hemorrhage. Stimulants, digitalis, the maintaining the temperature, and the administration of food are required. Some physicians recommend starvation until the hemorrhage is arrested, but this seems dangerously depressant. Cold to the abdomen may check the hemorrhage, but is generally poorly tolerated by infants. Tincture of the chloride of iron (1 minim) (0.062) or fluidextract of ergot (2 to 5 minims) (0.123 to 0.308) has been advised. Calcium lactate, 20 to 40 grains (1.3 to 2.6) in 24 hours, has been recommended. Gelatine seems to be one of the best of remedies. It may be given by the mouth in a 5 to 10 per cent. solution, as for any form of hemorrhage in the new-born. (See p. 300.) If hemorrhage is not quickly arrested, human blood or serum, or that of the horse or rabbit, should be used in one of the various ways already described (p. 300).

CEPHALHEMATOMA

By this title is designated a form of hemorrhage in the new-born producing a fluctuating swelling situated between the bones of the skull and the overlying tissues (cephalhæmatoma externum). Sometimes the blood is effused within the skull between the bone and the dura mater (cephalhæmatoma internum). The description which follows applies almost entirely to the external variety.

Etiology.—The disease is not very common. Hennig[1] found it recorded 230 times in 53,506 new-born infants, *i.e.* 0.43 per cent., and Hofmokl[2] 371 times in 59,885 cases; *i.e.* 0.6 per cent. Almost always the condition develops in the first few days of life, but very rarely cases have been recorded occurring in the first weeks or months, or even later. (Friedemann.)[3] Males are most frequently affected.

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[1] Gerhardt's Handb. d. Kinderkr., II, 49.

[2] Arch. f. Kinderheilk., 1880, I, 313.

[3] Münch. med. Wochenschr., 1904, LI, 387.

The direct cause of cephalhematoma is often some injury received during birth. Thus the incidence is much the greatest in the children of primiparæ (34 out of 40 cases, Meyer),[1] and in vertex-presentations when pressure has been long-continued. The circular pressure exercised by the uterus and the absence of this over the central presenting part probably tend to make the vessels yield and break. Yet in very many instances no evidence of direct trauma can be discovered, and the lesion appears to be due entirely to other causes. The condition may even develop in premature infants or after very easy labor, or sometimes in breech-presentations. The presence of asphyxia tends decidedly to produce it, and it may also be one of the evidences of a general hemorrhagic disorder in the new-born. The looseness with which the periosteum is attached to the underlying bone in the new-born, and the delicacy of the blood-vessel walls also predispose to its development.

Pathological Anatomy.—The lesion is generally unilateral, yet it may be bilateral, and rarely even appears over 3 different bones. The seat of the swelling is much more frequently over the right parietal bone. Hennig[2] reported it here in 57, and over the left parietal bone in 37, out of 127 cases. The hemorrhage takes place below the periosteum, lifting this from the bone possibly as far as the sutures, where it adheres too firmly to permit of this. The blood remains fluid sometimes even for some weeks. It averages from 0.5 to 1.5 fl. oz. (15 to 44) in amount, but it may be much more than this. In recent cases it is discharged with a spurt when incision is made. The bone is pale, rough, and often covered with fibrinous clots. An exudate of new osseous tissue forms a peripheral boundary-ridge upon the bone about the tumor. This is soft and velvety in recent cases, but later hard and projecting distinctly above the surface of the skull.

The periosteum over the swelling is bluish and often covered with small effusions of blood. In advanced cases small plates of new bone are deposited here and there in the periosteum. Section of the scalp over the swelling shows numerous scattered punctiform hemorrhages.

Quite exceptionally the bleeding instead of taking place under the periosteum occurs under the aponeurosis (*cephalhœmatoma subaponeurotica*). In such cases, of course, no bony ridge exists and the tumor is not necessarily limited by the sutures. Both varieties may occur together. Infrequently examination within the skull reveals also a hemorrhage between the dura mater and the bone, corresponding in position to the hematoma outside and perhaps connecting with it. McKee[3] found this internal cephalhematoma reported in but 16 instances in medical literature, in 11 of these associated with the external variety. When there has been decided trauma during labor fissures or fractures of the bones of the skull may be present. In the cases depending upon constitutional conditions hemorrhages occur in other parts of the body as well.

Symptoms and Course—Although generally discovered 2 to 3 days after birth the condition begins earlier than this but is not noticed. Sometimes it is at first concealed by a caput succedaneum, and only becomes visible as this disappears. It is at first small, but grows rapidly. Its maximum size is attained in from 6 to 8 days, the bleeding then ceasing, and the tumor varying in size up to that of a large hen's egg (Fig. 47). It is at first rather flat and soft, but soon becomes rounded and tense although fluctuating. It is not hot or tender to the touch and rarely pulsates. It cannot be reduced by pressure, and is not affected by crying. The skin over it is sometimes darker in color than normal but otherwise unaltered in appearance. The change in color is observed especially when the hemorrhage is subaponeurotic. The tumor covers all or a part of one of the bones of the skull, but if subperiosteal never passes beyond the sutures or over the fontanelle. Where the swelling appears to cover the adjoining bone also it is really a double cephalhematoma, and a distinct groove can be felt beneath the two at the position of the suture.

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[1] Hospitalstidende, 1877, IV, Nos. 37–38, 585–616.

[2] *Loc. cit.*

[3] Cincin. Lancet-Clinic, 1883, XI, 317.

After the tumor has existed from 2 to 3 days, a soft ridge can be felt forming around its periphery. This later becomes harder and thicker as osseous tissue develops in it, and gives very much the sensation of a soft mass protruding through a large hole in the skull. The tumor maintains its size unabated until its 2d week and then begins slowly to grow smaller and less tense. As this occurs the bony periphery broadens toward the centre. A parchment-like sense of crepitation can now often be developed on palpation, dependent upon the new osseous growth in the periosteum.

Although recovery is exceptionally more rapid, as a rule absorption of the effused blood goes on very slowly and the tumor does not disappear for 2 or 3 months from the onset. The bony ridge may still be felt more or less distinctly for months more, or the whole region of the tumor may be left somwehat thickened. General symptoms are entirely absent.

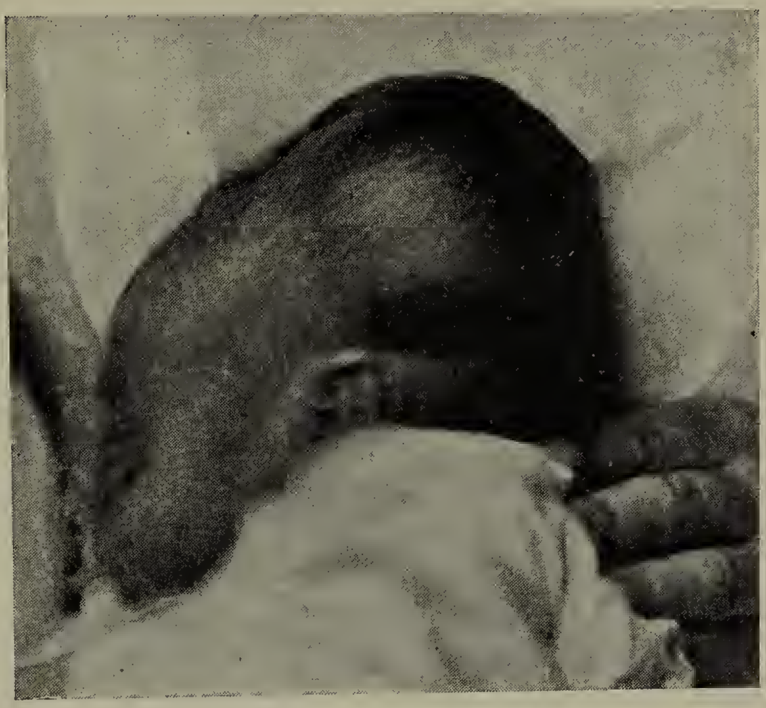

Fig. 47.—Cephalhematoma. (*From a photograph.*)

Occasionally the course is not so favorable. Suppuration may take place within the tumor, especially if there are external wounds of the scalp present. In this event the swelling becomes red and tender, and the infant is evidently ill with the general symptoms of suppuration. The abscess which forms may discharge externally and be followed by healing, or it may give rise to a diffuse inflammation of the tissues of the scalp with involvement of the bone and secondary meningitis. In other cases the prolonged suppuration produces fatal exhaustion or general sepsis.

Sometimes the disease is complicated by cerebral symptoms from the beginning. In such instances it is probable that there is an internal cephalhematoma or a meningeal hemorrhage also present.

Prognosis.—This is entirely favorable in every uncomplicated case, and where the hematoma is not the result of a general disorder. The development of suppuration in the tumor is a serious complication, but a rare one, and makes the prognosis doubtful. Still more serious is the presence of a complicating hematoma within the cranium. These cases are fortunately exceptional, since they nearly always die.

Diagnosis.—Between the subperiosteal and the subaponeurotic cephalhematomata the distinction may be made by the presence in the former of a distinct, hard, marginal ring, the limitation by the sutures, and the absence of decided discoloration of the skin. Hematoma may be readily confounded with *caput succedaneum* during the first few days of life. This condition consists of an edematous swelling of the connective tissue of that portion of the scalp which has been presenting through the patulous os uteri, and is the result of the obstruction to the circulation at the periphery and the absence of pressure over the swollen region. The fact that it disappears in a few days, and the absence of fluctuation and of the development of any marginal wall also serve to distinguish it from cephalhematoma. *Meningocele* and *encephalocele* resemble cephalhematoma to some extent. They may exhibit pulsation, are influenced by respiration and by crying, are to some extent reducible often with the production of convulsions, and correspond in position with a suture or a fontanelle. (See Meningocele, Vol. II, p. 597.)

Abscess of the scalp is attended by heat, tenderness, and discoloration of the skin, with constitutional symptoms. A telangioma resembles a hematoma but slightly. There is no bony wall or fluctuation, while crying makes it larger and pressure somewhat smaller and the skin is discolored.

Treatment.—The best treatment, as a rule, appears to be a wholly expectant one. The child should be so placed that no injury to the hematoma can occur, and recovery entrusted to time. No applications are required. Many writers have urged early incision in order to drain away the fluid blood and hasten recovery. This, however, adds an unnecessary element of danger, in that there is the possibility of producing sepsis through the open wound, beside the chance of having a secondary hemorrhage occur. The expectant treatment although slow is safe. On the slightest evidence of suppuration beginning the hematoma should be incised and drained antiseptically. If there are indications of intracranial pressure, and an internal cephalhematoma communicating with an external one is suspected, the question of aspiration by puncture with antiseptic precautions is to be considered.

HEMATOMA OF THE STERNOCLEIDOMASTOID MUSCLE

This consists of a hemorrhage into the body of the sternocleidomastoid muscle. It is of rare occurrence and is the result of injury received during birth. In about 75 per cent. of the cases this is produced by traction upon the legs in breech-presentations, as a result of which the blood-vessels in the neck are torn and a blood-tumor results. It may be brought about also by the pressure of forceps in vertex-presentations or may occur without any cause for the lesion being discoverable.

The tumor produced is quite small, not over 1½ inches (3.8 cm.) in length. It is tender on pressure due to the attending myositis, and at first soft. The skin over it is not discolored. Soon it becomes hard and the tenderness disappears as cicatricial changes take place in it. It may occupy any position in the muscle but is oftenest in the middle or upper part, and is most frequently situated upon the right side, only exceptionally occurring upon both sides. In 38 cases reported by Henoch[1] the right side was involved in 31. Although produced at birth it generally does not become apparent until the age of 2 weeks or later, especially if the neck is fat. The rigid inclination of the head of the infant toward the affected side with the chin turned toward the other is a characteristic symptom which leads to the examination of the neck and the discovery of the swelling. This alteration of position may, however, be absent.

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[1] Vorlesungen über Kinderkrankheiten, 1895, 35.

The immediate **prognosis** is nearly always good. The swelling slowly grows smaller, although it may not entirely disappear for a few months. It is probable, however, that the rigidity of the neck may in rare instances remain throughout life. The cases of so-called congenital torticollis are probably produced in this way. (See Torticollis, Vol. II, p. 709.)

No **treatment** except rest is required in the acute stage. Later gentle massage and passive movements of the head may be of benefit. Should persistent torticollis remain surgical treatment may be demanded later, in the form of orthopedic apparatus or of operative interference.

CHAPTER VI

ICTERUS IN THE NEW-BORN

Icterus is of very frequent occurrence in the new-born, and may be due to various causes. It may conveniently be divided into (1) symptomatic, and (2) idiopathic icterus.

1. *Symptomatic icterus* depends upon such general diseases as infectious hemoglobinemia, acute fatty degeneration of the new-born, sepsis, syphilitic hepatitis, congenital cirrhosis of the liver and congenital obliteration of the bile-ducts. These are all of rare occurrence. The ordinary gastroduodenal catarrh with closure of the common bile-duct, which occasions icterus so frequently in adult life, is often a cause in the new-born also and is liable to be more severe than in later life. Most of the forms of symptomatic icterus are discussed elsewhere in course, with the exception of that depending upon the congenital obliteration of the ducts to which brief special consideration may be given.

In symptomatic icterus the discoloration of the skin is generally very decided, and there are other symptoms of the causative disease evident. The urine often shows the presence of bile, and, in obstructive jaundice, the feces often the absence of it.

2. In *idiopathic icterus* in the new-born there is present no discoverable congenital or acquired anatomical defect which can produce jaundice by obstruction. In this class are to be placed the cases of congenital hemolytic icterus; and here, too, are some very severe instances of jaundice in the new-born of a familial nature. Both of these conditions will be discussed later. (See Vol. I, p. 216.) The great majority of cases, however, constitute a form of jaundice of very common occurrence, to which the application of the title icterus neonatorum is usually limited.

CONGENITAL OBLITERATION OF THE BILE-DUCTS

(Congenital Biliary Cirrhosis)

This is a rare condition of which, however, according to Thomson[1] more than 100 cases had been reported at the date of his writing.

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[1] Allbutt and Rolleston, System of Med., 1908, IV, 1, 103.

Holmes[1] in a very careful review of the subject published in 1916 estimates that nearly 120 cases have been reported and other cases have since been published. I[2] have observed 2 instances of it verified by autopsy, and others probably of this nature but without verification. Congenital biliary cirrhosis, although described as a distinct affection, would seem probably, as claimed by Skormin[3] and by Rolleston and Hayne,[4] to be of the same nature as obliteration of the bile-ducts, both in pathogenesis and in symptomatology.

Etiology and Pathology—The cause is little understood. The disease is liable to occur in several members of one family, this supporting the view that there exists some congenital failure in development. Some cases indicate the existence of a prenatal obliterative inflammation of the bile-ducts, which either starts as a biliary cirrhosis and descends to the larger ducts, or in which the two regions are affected at the same time. Still another view makes the cirrhosis secondary to complete obliteration of the main ducts and dependent upon the interference with the exit of bile. One of my cases supports this view. Thomson has shown that a biliary cirrhosis was present in nearly all of the recorded instances which he collected. Parsons and Hickmans[5] report a case of congenital biliary cirrhosis with atresia of the intrahepatic ducts but without any of the extrahepatic ones. (See also Icterus, Vol. II, p. 213.)

It does not seem likely that syphilis plays any important part in the etiology. Some of the instances of severe congenital icterus may possibly be due to a partial obliteration of the bile-ducts involving the mucous membrane of the ducts and to a certain extent the connective tissue as well, but never advancing to complete obstruction. This theory would possibly explain certain cases of jaundice reported by Arkwright,[6] Weber[7] and Pearson.[8] It is certain that the obstruction may occasionally not be complete at first, and that entire obliteration may develop only considerably later.

Pathological Anatomy.—The bile-ducts may be undiscoverable or they may be replaced by a fibrous cord; or there may be obliteration at but a single point, the ducts above this being without stenosis. The gall-bladder may be absent or distended. The liver is generally enlarged, firm, and of normal or greenish color, and in most cases exhibits the histological lesions of hypertrophic cirrhosis. The blood-vessels are seldom involved; the spleen is enlarged.

Symptoms.—The principal symptom is icterus, which is present at birth or develops within the 1st week or occasionally later. The color is intense and often of a greenish hue. The occurrence of acholic stools is also of importance. Sometimes ordinary yeollw movements are present at first, to be replaced later by the characteristic appearance. The passage of green feces does not, of course, necessarily indicate the presence of bile. The urine is intensely bile-stained; fever is not a symptom, but vomiting is liable to occur. Hemorrhages from the umbilical cord and into different parts of the body are a very characteristic symptom, yet in no way pathognomonic, as they may appear in severe icterus from other causes. Emaciation develops if the case is at all prolonged.

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[1] Amer. Jour. Dis. Child., 1916, XI, 405.

[2] Arch. of Pediat., 1905, XXII, 257. Ibid., 1908, XXV, 174.

[3] Jahrb. f. Kinderheilk., 1902, LVI, 203.

[4] Brit. Med. Jour., 1901, 1, 758.

[5] Amer. Jour. Dis. Child., 1926, XXXI, 459.

[6] Edinburgh Med. Jour., 1902, LIV, 156.

[7] Edinburgh Med. Jour., 1903, LVI, 111.

Underwood's Dis. of Child., 1842, 9th Edit., 101.

Course and Prognosis.—The outcome is necessarily fatal, if we include among the cases only those in which complete organic obliteration finally occurs. The course is seldom acute. Death takes place in convulsions or stupor, generally only after several weeks or even months, although sometimes the child dies in the 1st week of the disease. Perhaps the longest duration of life recorded was in a child dying at 7½ months reported by Nobecomb and Janet[1].

Treatment can be only palliative and symptomatic. Operative interference at this early age appears to offer little hope; both on account of the difficulties attending it, and because biliary cirrhosis is usually combined with the obliteration.

ICTERUS NEONATORUM

Etiology and Pathogenesis.—The condition is a very common one, ranging according to different statistics from 15.69 per cent. (Seux)[2] to 84.46 per cent. (Cruse)[3] of all births. It is probable that careful observation would show it present to at least a slight degree in the great majority of the new-born. Boys are oftener affected than girls; the first-born children oftener than later ones, and premature, light-weight, or atelectatic infants oftener than others (Kehrer).[4]

Regarding the exciting causes of the condition and the method of its production the years of investigation have not yet given an entirely satisfactory solution. The different theories group themselves for the most part into two classes: first, that the icterus is hepatogeneous in origin; *i.e.* arising from some disturbance in the secretion of bile in, or its exit from, the liver; second, that it is hematogeneous; *i.e.* depending upon degeneration of the blood-corpuscles in the circulation and the production in this way of biliary coloring matter. In support of the hepatogenous nature is the finding of biliary acids in the serous fluids of the body (Runge),[5] and it is admitted that these must have their origin in the liver. The method, however, in which the hepatogeneous icterus is produced in the new-born is uncertain and various theories exist. Silbermann[6] maintains that there is an abundant breaking up of red blood-cells in the circulation after birth. This produces an increase of material from which bile is constructed, and, in addition, an increase of fibrin-ferment, which, on its part, occasions great congestion in the blood-vessels of the liver, and a consequent compression of the biliary capillaries. Ylppö[7] and Hirsch[8] have shown that the blood-serum of the fetus in its last month contains much more biliary pigment than is present in that of adults, and that the amount increases still more in the first days after birth. The liver is functionally immature, and is therefore unable to dispose of the excess of pigment, if this is unusually great, which consequently enters the blood-current instead of the biliary capillaries, and produces jaundice. When the amount of pigment exceeds 125 milligrams per 100 c.c. of blood the infant becomes icteric (Ylppö). The functional inability of the liver to dispose of the very large production of bile-pigment in the new-born is confirmed by the studies of Hess.[1] Schiff and Faerber[2] and Cserna and Liebmann[3] also found bilirubin constantly present in the blood of new-born infants.

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[1] Arch. de méd. des enf., 1922, XXV, 90.
[2] Recherches sur les mal. des enfants nouveau-nés, 1855, 269.
[3] Arch. f. Kinderheilk., 1880, I, 353.
[4] Oesterreich. Jahrb. f. Pädiat., 1871, II, 71.
[5] Krankh. der ersten Lebenst., 1893, 225.
[6] Jahrb. f. Kinderheilk., 1887, XXVI, 252.
[7] Zeitschr. f. Kinderheilk., Orig., 1913, IX, 208.
[8] Zeitschr. f. Kinderheilk., Orig., 1913, IX, 198.

Among other opinions of the etiology, Birch-Hirschfeld[4] believes that icterus is due to a compression of the larger biliary ducts by an edema of Glisson's capsule, the edema being dependent upon venous congestion incident to the circulatory changes following birth. Frerichs[5] and others, on the other hand, consider the icterus caused by a diminution of venous blood present in the liver, the result of the cutting off of the blood-supply which had been received through the umbilical vein. A resorption of bile follows this. Still another view which has many adherents is that of Quincke[6] that the icterus depends upon an increased production of bile derived from the meconium, the resorption of this from the intestine, and the direct passage of it through the patulous ductus venosus into the general circulation. Yet that the jaundice and the increased content of the blood in biliary coloring-matter may be independent of the action of the liver has much in its support. Thus Schick,[7] Wagner[8] and Knopfelmacher and Kohn[9] conclude that the increase of biliary coloring-matter in the blood, and the consequent icterus, is dependent upon the maternal blood, and is therefore not hepatogenous in origin. The blood of the mother passes to some extent into the fetus and is there disintegrated, the hemoglobin being transformed into bilirubin. Deluca[10] attributes the jaundice to the disintegration of extravasated blood from the meningeal hemorrhages so common in the new-born, and Herzfeld[11] believes that the swallowing of the vaginal bacteria causes the jaundice, and that this can be prevented by aspirating the mouth of the new-born infant with a soft catheter.

No theory advanced has not met with opponents, and the question still remains an open one to a large extent. A very good review of the subject is given by Van Crevela.[12]

Pathological Anatomy.—Inasmuch as the condition is not a fatal one, autopsies can be made only on those infants who have died from other causes. The skin, subcutaneous connective tissue, lining of the arteries, and most of the serous membranes and exudates are yellow; the extent of this varying with the case. The brain, spinal cord, spleen, kidneys and liver are only slightly discolored if at all. The ductus choledochus is patulous; the kidneys are congested and exhibit uric-acid infarcts and sometimes infarcts of biliary coloring matter, and the latter have also been found in the brain.

Symptoms.—The discoloration begins oftenest on the 2d or 3d day of life; much less often earlier or later than this. The surface of the body is the region most markedly affected, but in most cases the scleræ are

[1] Amer. Jour. Dis. Child., 1912, III, 304.

[2] Jahrb. f. Kinderheilk., 1922, XCVII, 245.

[3] Klin. Wochenschr., 1923, II, 2122.

[4] Gerhardt's Handb. d. Kinderkr., IV, 2, 691.

[5] Klinik d. Leberkrank., 1858, I, 199.

[6] Arch. f. exper. Path. u. Pharmakolo., 1885, XIX, 34.

[7] Zeitschr. f. Kinderheilk., Orig., 1921, XXVII, 231.

[8] Zeitschr. f. Kinderheilk., Orig., 1921, XXVII, 251.

[9] Monatsschr. f. Kinderheilk., 1921, XXII, 1.

[10] Semana med., 1921, II, 635.

[11] Jahrb. f. Kinderheilk, 1922, XCIX, 75.

[12] Amer. Jour. Dis. Child., 1925, XXX, 240.

also involved. The mucous membranes also are icteric. The discoloration is first noticed on the face or chest, and its degree varies greatly. Sometimes it is intense, but often it is very slight and can be appreciated only in good daylight and after the red color of the skin is removed temporarily by pressure with the finger. The urine is generally free from bile-pigment, at least by ordinary tests, although the microscope may sometimes reveal masses of biliary pigment. There is an increase in the excretion of urea and uric acid (Hofmeier).[1] The stools are unaltered in character. The pulse, respiration and temperature of the child are entirely unaffected, and the liver and spleen are little if any enlarged. A study of the coagulation-time of the blood made by Greuter[2] showed that in 75 per cent. of the cases examined there was a prolongation. This disappeared by the 9th day even if icterus was still present. Regarding the influence of the disease on the general health and nutrition there is some dispute. The majority of statistics would indicate that there occurs a greater loss of weight and a slower regain of it when icterus is present.

The duration is about 3 or 4 days in the milder cases. In the severer forms it may be 2 weeks or more before all traces of the icterus have disappeared, but in these long-continued cases it is always questionable whether the condition does not depend upon some other cause.

Diagnosis.—The distinction is to be made between icterus neonatorum and the various forms of symptomatic icterus. This is to be done in part by the presence of other symptoms in the latter condition. Then, too, in symptomatic icterus the urine is more liable to be visibly discolored by the bile-pigment. In cases of decided obstructive jaundice the stools show an absence of bile. A very slight degree of discoloration of the skin speaks rather for icterus neonatorum, while a late development of it indicates some other cause. Yet an early diagnosis is often impossible. This is especially true in slight cases of catarrhal icterus due to duodenal catarrh, and still more in the instances in which icterus neonatorum happens to be combined with other diseases of the new-born; such, for instance, as sepsis. Congenital obliteration of the bile-ducts gives rise to an intense icterus developing very promptly after birth and persisting. Certain severe cases of icterus in the new-born will be discussed later. (See Icterus, Vol. II, p. 213.) Such infants are distinctly ill, and the icterus is accompanied by other signs, sometimes of a hemorrhagic nature.

Treatment.—Nothing is needed, and nothing can be done for the ordinary average case. Time will cure it. The ordinary care should be taken to keep the functions of the organism in good order.

FAMILIAL ICTERUS OF THE NEW-BORN

This form of icterus as seen in the new-born cannot be distinguished from the ordinary icterus neonatorum except by its familial nature, the severity of the symptoms, and the bad prognosis. It will be discussed later in considering Congenital and Familial Icterus (See Vol. II, p. 216).

[1] Zeitschr. f. Geburtsh. u. Gyn., 1882, VIII, 287.
[2] Zeitschr. f. Kinderheilk., 1923, XXXV, 210.

CHAPTER VII

ASPHYXIA NEONATORUM

The term Asphyxia, or Suffocation, as applied to the new-born indicates a condition in which, although the heart's action continues, the supply of oxygen to the blood ceases, and respiratory movements after birth are either absent or insufficient to overcome this lack.

Etiology.—The disease may either be (1) of *intra-uterine* or (2) of *extra-uterine* origin, the latter being much less frequent. The distinction depends on whether or not the circulation through the placenta has been interfered with.

(*A*) Intra-uterine Asphyxia.—This develops before or during birth, and is due to some interruption of the normal interchange of gases in the placenta. Among maternal causes which interfere with the placental circulation may be mentioned excessive uterine contraction, unduly prolonged labor, uterine hemorrhage, and severe complicating illness or the death of the mother. On the side of the child are such factors as detachment of the placenta, compression of the brain interfering with the action of the heart, and compression of the umbilical cord. Efforts at intra-uterine respiration occur in most cases as a result of the undue stimulation of the respiratory centres; but whether this stimulation is the result of the overloading of the blood with carbon-dioxide or of the deprivation of oxygen is not yet certainly determined. The consequent filling of the respiratory passages with fluid increases the danger to the infant after birth has occurred. It has been denied by some that intrauterine respiratory efforts are sufficient to cause aspiration of amniotic fluid, but Duvoir[1] concludes, from the study of reported pathological examinations, that the lungs of still-born infants may show fluid within the alveoli. In cases, however, where the asphyxia has developed slowly the respiratory centres have sustained a paralyzing action, and efforts at breathing have not taken place.

It is evident that the likelihood of the occurrence of intra-uterine asphyxia must increase with the duration of labor, especially of its second stage. The statistics of Veit[2] show very strikingly that the mortality from asphyxia after a second stage of 4 hours or more was over 3 times as great as when it had lasted but 1 hour.

(*B*) Extra-uterine Asphyxia (*Atelectasis pulmonum*).—The child is born without any evidence of asphyxia, but develops it soon after birth from some of the numerous causes which interfere with the gaseous interchange. Among these may be mentioned obstruction to the access of air, as by unruptured membranes, or by maternal discharges in the infant's air-passages; malformation of the diaphragm; intra-uterine pneumonia or pleural effusion interfering later with the action of the lungs; malformation of the lungs; severe injuries to the brain at the moment of birth, which afterward affect the action of the respiratory centres; malformation of the heart, which renders the carrying of oxygenated blood impossible; etc. Premature birth is a very potent cause of extra-uterine asphyxia, the active factor being the general feebleness of the child, the weakness or imperfect development of the respiratory nerve-centres, or a similar condition of the muscles and bones of the chest-wall or of the lungs preventing satisfactory pulmonary or thoracic expansion.

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[1] La Médecine, 1920, II, 534.

[2] Monatsschr. f. Geburtsk., 1850, VI, 112.

Pathological Anatomy.—The lesions found are those characteristic of suffocation. The blood is fluid and of a dark color. The heart, especially the right chambers, is overfilled with blood. The blood-vessels in general are distended, and all the internal organs congested. The liver is of a dark bluish-red color. Numerous punctiform or larger hemorrhages may be found in nearly any of the organs of the body. Bloody serous fluid is present in the serous cavities. There is often the escape of a considerable amount of blood into the intestines.

If the child has made attempts at inspiration while still in the uterus, mucus, bloody amniotic fluid or meconium may be found in the larynx, trachea and large bronchi; less often in the finer bronchi and the alveoli. The lungs are very dark-red, heavy, much congested and of uniform atelectatic appearance when there has been no entrance of air. This intense congestion is evidence that intra-uterine efforts at respiration have probably taken place. If air has entered to some extent, either through the infant's own efforts or as a result of the employment of artificial respiration, small scattered areas of distended pulmonary tissue will be visible, especially in the upper lobes.

Symptoms. (*A*) INTRA-UTERINE ASPHYXIA.—In asphyxia of intra-uterine origin certain symptoms discoverable before birth make the diagnosis very probable. There occurs in the intervals between the labor-pains a retardation of the fetal heart-sounds. Finally, if delivery does not occur, the sounds grow more irregular, rapid, and weak, and then cease altogether. A suspicious, although not entirely diagnostic, symptom in vertex presentations is the discharge from the maternal vagina of meconium which has just been passed by the infant, as a result of the increased intestinal peristalsis that asphyxia produces. The intra-uterine movements of the child may become more active. Exceptionally in breech presentations, convulsive movements of the body may be noticed. Sometimes, too, intra-uterine efforts at respiration may occasionally be detected by the finger inserted into the infant's mouth.

In the case of new-born healthy children respiration, followed by a vigorous cry, begins at once, or at the most after a very few seconds. In asphyxiated children, on the other hand, either no respiratory efforts at all are noticed after birth, or only imperfect and intermittent ones. The body is motionless and the child appears to be dead except for the continued action of the heart.

Two degrees of asphyxia of intra-uterine origin are observed: a milder form, *asphyxia livida* and a severer form, *asphyxia pallida*. The symptoms, prognosis and treatment of the two are very different.

1. **Asphyxia Livida.**—In this milder form the skin is dark bluish-red in color, the heart's action is strong although decidedly slow, and the pulse in the umbilical cord is full and strong and the tension high. The conjunctivæ are injected and the face turgid. Respiratory efforts are absent or occur only occasionally, and at first very superficially, and are attended by a contortion of the face. Coarse râles are audible in the lungs. Stimulation of the skin causes an energetic inspiratory effort, and the finger introduced into the mouth for the purpose of cleansing it pro-

duces attempts at vomiting or swallowing. A very characteristic symptom of asphyxia livida is that the muscle-tonus is preserved; *i.e.* although the child is motionless, yet it is not absolutely flaccid when it is lifted.

2. **Asphyxia Pallida.**—In this variety the skin is pale and corpse-like, and the heart's action very weak and usually rapid. The vessels in the cord appear to be empty, and the pulse there is entirely absent or extremely feeble. Generally there are no true respiratory efforts, and any occasional attempts at inspiration which may occur seem to depend solely on the action of the diaphragm, the thorax moving not at all, and no grimace of the face attending them. No râles can be heard; this showing that the efforts at inspiration have been absent or entirely futile. Stimulation of the skin is without result, and the finger in the pharynx produces no reflex movements of its muscles or of the palate. The great characteristic of this grade of asphyxia is complete loss of muscle-tonus. The child is absolutely limp, the jaws fall, the head drops completely in some direction if the child is lifted, and the anus is open.

(*B*) EXTRA-UTERINE ASPHYXIA.—In asphyxia of extra-uterine origin the symptoms vary somewhat, depending upon the cause. The skin is usually dark reddish-blue. The heart's action is strong, and the vessels of the cord are filled with blood. The pulse, however, is not at first slow, and although it becomes so later it finally, as a rule, grows rapid again. This distinguishes the condition from intra-uterine asphyxia. Respiration is wanting or occasional only, and is not attended by râles. This lack of râles shows the absence of fluid from the respiratory tract, an evidence that the condition is extra-uterine in nature. In asphyxia in premature infants respiration may continue irregular and very superficial for days, and is almost entirely diaphragmatic. The children lie for the most part with eyes closed, motionless and somnolent, and make no sound or only occasionally utter a feeble cry. The face is dark-red and somewhat swollen; edema of the extremities and scrotum develops; the temperature is subnormal; there is loss of weight. In marantic infants who have passed the age generally allotted to the new-born, as well as later in badly rachitic subjects, asphyxia is of not infrequent occurrence. It is the result of atelectasis, and its symptoms are those of the extra-uterine form developing earlier in life.

Course and Prognosis.—Unless treated there is probably little tendency for cases of asphyxia to recover. **Asphyxia livida,** when not excessive, will nearly always yield quickly to appropriate treatment. In the course of a few seconds or possibly minutes respirations become more frequent and effective, and are finally succeeded by loud crying. Many children, however, pass from the milder form into the severer degree. The result in **asphyxia pallida** is always more doubtful. The duration is variable. Some children die almost immediately, while others may continue for hours without any apparent change; or, after improvement begins, may relapse if treatment is not persisted with. If recovery ensues intermittent and occasional respirations begin and are perhaps followed by a slight cry, and finally satisfactory breathing is established. The longer the duration of the second stage of labor, the worse is the prognosis. The prognosis is graver, too, in proportion to the weakness of the pulse or of the heart-sounds. The presence of any complication, such as intracranial hemorrhage or acute fatty degeneration, likewise makes the prognosis unfavorable. Yet even in the severer grades of intra-uterine asphyxia the majority of infants will recover. Even those who have seemed hopeless, and who for hours have appeared to be dead, may still survive. Asphyxia of extra-uterine origin generally runs a rapid course if dependent upon some malformation or some disease of the respiratory apparatus. When the result of premature birth it may continue, as stated, for days.

Regarding the after-effects of the disease upon a child who has survived, it has been maintained that long-continued severe asphyxia is liable to produce idiocy or paralytic conditions. Although it seems more probable that such disorders are the result of some injury to the brain at birth, of which the respiratory condition was but a symptom, yet it is possible that the congestion accompanying the asphyxia may have produced the breaking of blood-vessels within the cranium, and thus have become the first cause of later nervous or mental disorders. It may happen, too, that aspiration-pneumonia may develop in an infant after its recovery from asphyxia.

Diagnosis.—The milder grade of asphyxia may be confounded with *compression of the brain* by hemorrhage. Meningeal hemorrhage may indeed produce symptoms resembling those of asphyxia very closely, and as the latter condition may be one of the symptoms of the former, the object of diagnosis is to determine whether or not asphyxia is actually present, and, if so, whether it depends on an intracranial compression. Compression produces slowing of the pulse and irregularity of respiration. If a child exhibits at birth the symptoms of the milder degree of asphyxia and, instead of responding quickly to treatment, does so not at all, or but temporarily or imperfectly, while the slowness of the pulse persists, the existence of a complicating meningeal hemorrhage is very probable. This is true also if the child is born without evidence of asphyxia, but soon passes into this condition, with slow pulse, slow irregular respiration, coma, and bulging fontanelle. In other cases there is marked asphyxia from the beginning and the diagnosis must for a time remain in doubt. The presence of paralysis renders the diagnosis of hemorrhage positive. The history of the case aids in the recognition of compression; as, for instance, when the labor has been unduly prolonged, the pelvis narrow, or forceps applied.

Intense anemia resulting from severe hemorrhage may simulate asphyxia pallida closely. The presence of râles, if a respiration occurs, is an indication that intra-uterine efforts at breathing have taken place and that asphyxia exists. The history is of the greatest diagnostic value, for only the tearing of the umbilical cord during birth can produce a hemorrhage which is sufficient to account for so great a degree of anemia.

Asphyxia may be complicated by, or be the only symptom of, *acute fatty degeneration of the new-born.* In such cases the issue is fatal, and only the post-mortem microscopic examination makes the diagnosis clear.

Treatment. (*A*) ASPHYXIA OF INTRA-UTERINE ORIGIN.—The condition should be prevented as far as may be by employing such measures as will obviate unduly prolonged delivery, and in every other respect maintain the labor in as normal a condition as possible. Evidences of asphyxia developing during labor must be watched for carefully. The subject is treated of in works upon obstetrics.

Whatever the degree of asphyxia present after the birth of the infant the first indication is to free the respiratory passages as far as possible. A finger should be introduced into the mouth and pharynx in order to remove any mucus or fluid present. The child may also be suspended with head downward for a moment in order to favor discharge of fluid, and to produce a congestion and stimulation of the respiratory centres, and the body be slapped smartly with the open hand. In the case of **asphyxia livida,** if well-marked, the cord may be severed at once, and ½ ounce (15) or less of blood allowed to escape. The child may then be slapped with a cold, wet towel or plunged into cold water in order to stimulate respiration by cutaneous irritation. It must then immediately be put into a hot bath. This procedure, involving the alternation of hot and cold water, may be quickly repeated as often as necessary. Efforts at respiration are generally produced by the cold water. These are attended by fresh accumulations of mucus in the throat necessitating repeated cleansing of this in the manner mentioned. Sometimes a single application of cold will suffice. In other cases we must continue perhaps a half hour before all evidences of abnormal color have disappeared, and the infant is breathing well and crying lustily.

In **asphyxia pallida**, or in those cases of asphyxia livida in which the treatment described does not quickly produce some decided efforts at respiration, valuable time is lost by employing it. In the latter form of the disease the child has passed the stage where cutaneous stimulation can produce any effect whatever, and in the former the permitting of a flow of blood from the cord can do only harm. The cord should be ligated and cut promptly, and artificial respiration commenced at once. Whatever means are employed an artificial *expiration* should be sought first, in order to remove aspirated fluid from the respiratory tract. If inspiration occurs first the fluid is drawn still further into the lungs. The method of Schultze[1] is that most in vogue. The physician grasps the child with both hands in such a way that his thumbs rest loosely on the front of the child's thorax, his first fingers pass from behind into the axilla, and the remaining fingers are spread over the back of the child. The infant's head rests against his forearms and hands. Standing with knees somewhat apart, and having the child hanging feet downward and face forward, he now swings the body forward and upward, exercising no compression with the fingers, until his arms are somewhat above the

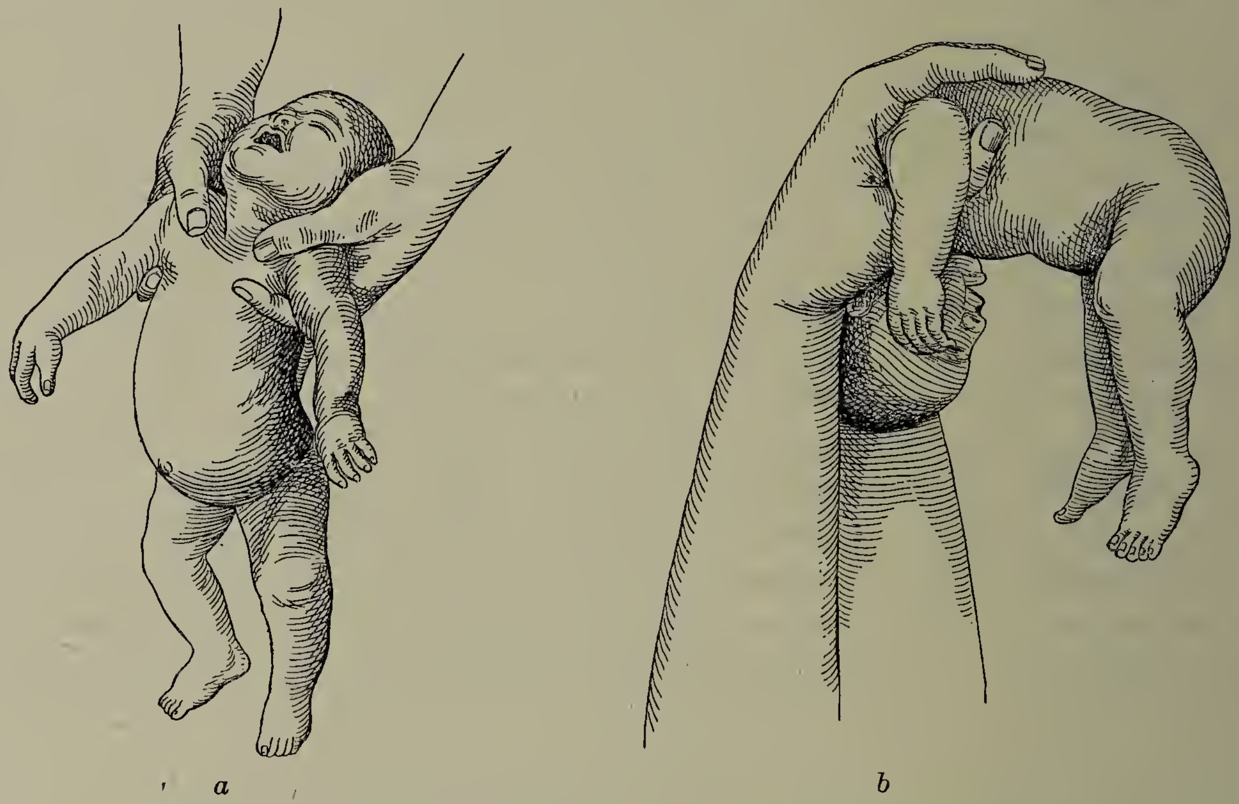

FIG. 48.—SCHULTZE'S METHOD OF ARTIFICIAL RESPIRATION.
(*a*) Inspiration; (*b*) expiration. (*B. C. Hirst, Obstetrics, 8th Ed.* 816.)

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[1] Der Scheintod d. Neugeborenen, 1871.

horizontal position. The swing now ceasing, momentum carries the lower part of the child slowly forward, throwing the legs up and over toward the physician, while the head hangs down. The trunk becomes in this way strongly flexed, and the abdomen and chest consequently compressed, the weight of the child resting against the physician's thumbs. This pressure causes an enforced expiration, and the aspirated fluid may be driven from the mouth and nose. After a pause of a few seconds the child is again swung downward into the original position. All pressure being thus removed from the chest, an enforced inspiration follows. In a few seconds more the movements are repeated, and so on for 6 or 8 times in the course of a minute or less, after which the child is placed in a tub of warm water for a few minutes. If no natural respiratory movements have commenced, the whole process is repeated (Fig. 48).

It is important that the inversion of the child in the production of expiration should occur slowly, in order that the flexion of the spine may take place in the lumbar region and not above this. The first part of the swing upward is quickly made, the latter part more slowly. The neck must always be kept from flexing in order to permit free entrance of air. Any compression by the hands must be carefully avoided at every stage. The movements, though performed thoroughly, must always be done gently and skilfully. The method requires some practice to be carried out properly. If the swinging movements have succeeded in starting respiration and increasing the power of the heart's action, the case has now become one of the milder degree of asphyxia, and cutaneous stimulation may be tried. If it has not succeeded artificial respiration must be used again and again. It should be persisted with as long as the heart continues to beat, even for hours if necessary.

It is, in fact, very important not to abandon too soon this or any other method, of resuscitation which is employed. A few efforts at respiration by the infant, or a single cry, do not constitute recovery, as relapse is very liable to occur. Easy respiration must be fully established, the normal color must return, the extremities move, and the child cry well and open its eyes before the physician may feel content. Very careful supervision is necessary after respiration seems established. Small doses of alcoholic and other cardiac stimulants are useful, as well as careful protection against chilling by the use of cotton wrappings and hot-water bags.

Weakly infants with great debility of the heart's action may not well tolerate the Schultze's treatment, or the chest walls may be so yielding that inspiration does not occur. In other instances the presence of such injuries as fractures, especially of the arms or clavicles, interferes with the use of the method, and always there is the difficulty in avoiding chilling, In place of it the direct inflation of the lungs may be tried. Mouth-to-mouth inflation is an old method, which may be employed in emergency, a clean towel being placed between the mouth of the child and that of the physician and only the first part of the expired air from the physician blown into the child. The nostrils should not be compressed. If this does not succeed a soft rubber catheter may be passed into the larynx, and, after fluid aspirated by the child has been withdrawn by suction by the mouth or by a rubber bulb attached to the tube, air may be forced into the lungs and again drawn out, aided by pressure of the hand upon the thorax. This should be continued, using fresh air at each inflation until respiration is established. Hofmeier[1] claims that with this method he has had bettter results than with that of Schultze. The method is not without objections, for not only is it difficult to pass the tube into the larynx instead of the esophagus, but too great pressure may easily overdistend the lungs and produce emphysema or even rupture of the vesicles. A more scientific modification of this is the furnishing of a continuous supply of air, as used in animal experimentation, and the employing of a manometer to guard against too high a pressure; and very good results may be obtained with a pulmotor especially adapted for use in new-born infants.

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[1] Monatsschr. f. Geburtsh. u. Gynäk., 1916, XLIV, 289.

Numerous modifications of or substitutions for Schultze's method have been proposed, some of them antedating it. Among these may be mentioned those of Marshall Hall[1] and of Silvester[2] as used for the recovery of persons from drowning, and the method of resuscitation recommended by Laborde.[3] This latter consists in grasping the tip of the tongue, wrapped about with gauze, and making rhythmic traction on it 8 to 12 times a minute. It is a powerful awakening of respiratory efforts if there is any reflex excitability remaining in the nervous supply of the tongue. Its advantage over Schultze's method is that it can be used while the child is kept in the warm bath. According to Dew's[4] method the left hand of the physician grasps the infant by the back of the neck and the right hand the knees, the body and thighs resting upon the palms. The lower extremities and abdomen are now flexed upon the thorax and head producing expiration, and the body and head then extended into a backward curve to bring about inspiration. This seems to be practically identical with the method of Byrd,[5] which consisted in letting the child lie with its back upon the palmar surface of the physician's two hands. The body is then flexed, bringing the head and feet close together and forcing the air from the chest. A movement of over-extension in the opposite direction tends to produce inspiration. A similar procedure is recommended by Ssokolow[6] (Fig. 49), the head being allowed to flex toward the spine during the extension of the body. These methods have the advantage that the infant can be kept in the warm bath much of the time. Minkévitch[7] places the infant on its back, with the hand of the physician in each axilla, and alternately bends the trunk forward between the separated legs, and extends it again into the horizontal position, while Rosenthal[8] flexes the knees upon the breast with the infant similarly placed. Prochownick[9] advises that the child be held inverted while an assistant alternately compresses and releases the thorax; Zangemeister[10] recommends forced inhalation of oxygen through a tracheal catheter fitted with a bulb; and La Rue[11] claimed good results in re-establishing cardiac action by massage of the heart by the thumbs over the precordial area. Greenwood[12] moves the infant quickly feet-first downward for about 2 feet, the inertia of the abdominal viscera on the abrupt cessation of the movement depressing the diaphragm and thus producing inspiration. Then the process is repeated with the head downward, expiration occurring on the stopping of the movement. This procedure is repeated about 15 times in a minute.

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[1] Lancet, 1856, II, 124.
[2] Brit. Med. Jour., 1858, 576.
[3] Les tractions rhythmées de la langue, 1897.
[4] Koplik, Dis. of Inf. and Childh., 1910, 196.
[5] Baltimore Med. Jour., 1870, I, 646.
[6] Monatsschr. f. Kinderheilk., Orig., 1911, X, 457.
[7] Semaine méd., 1902, XXII, 372.
[8] Therap. Monatsh., 1893, VII, 555.
[9] Centralbl. f. Gynäk., 1894, XVIII, 225.
[10] Centralbl. f. Gynäk., 1903, XXVII, 1162.
[11] Pediatrics, 1914, XXVI, 126.
[12] Lancet, 1921, I, 964.

(*B*) Asphyxia of Extra-uterine Origin.—Treatment is of little avail where the asphyxia results from anomalies of respiration or of circulation, or from intra-uterine diseases. When it depends upon the existence of premature birth there become necessary certain modifications of the methods already detailed. Not only is the relief of the asphyxia required, but that treatment also which has been described as appropriate for premature birth (p. 291). The removal of aspirated fluid from the mouth is not usually needed, since intra-uterine efforts at respiration have not taken place. When the case is one unsuited for, or not benefited by, cutaneous stimulation, artificial respiration must be attempted. However, the weakness of the child and the lack of elasticity of the chest-wall, together with the great need for maintaining bodily heat, render Schultze's method in many cases inapplicable or unavailing, and some of the procedures are to be preferred which permit of the constant mainte-

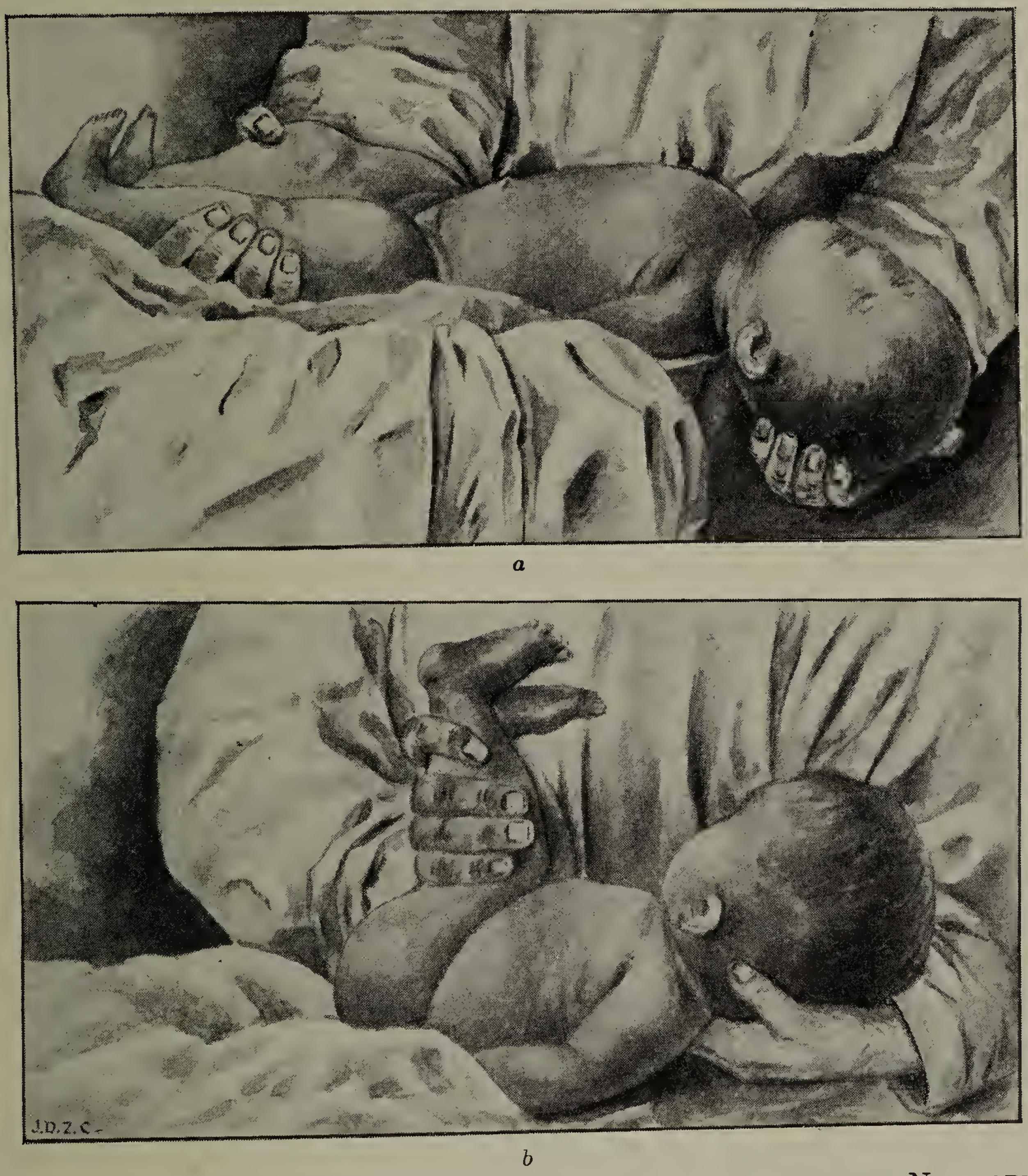

Fig. 49.—Method for Inducing Artificial Respiration in the Newborn. (*a*) Inspiration; (*b*) expiration. (*After Ssokolow, Monatsschr. f. Kinderheilk., Orig.* 1911, *X*, 459.)

nance of bodily temperature in a warm bath. The very great liability of these cases to relapse is not to be forgotten. When there is cyanosis inhalations of oxygen may be useful, but should not be employed too freely else respiratory effort by the infant will be interfered with. Alpha-lobelin in doses of gr. 1/20 (0.0032) by hypodermic injection has been recommended by Oberbeck[1] in all types of asphyxia. The employment of this remedy as a respiratory stimulant is of long standing. Close watching is required, and if the tendency appears, warm baths and cutaneous friction should be employed several times daily in order to interrupt the somnolence and produce vigorous crying. Days may pass before either the safety of the child is assured or death takes place.

CHAPTER VIII

SUDDEN ARRESTS OF RESPIRATION IN THE NEW-BORN

To this condition attention was called by Still[2] and later by Kirkwood and Myers.[3] We have encountered 1 very typical case in an infant of 6 weeks, which ended fatally. It develops usually in infants but a few days old, but sometimes of several weeks. The **cause** is obscure. There is no association of previously existing asphyxia or cyanosis, and no necessary evidence of general feebleness or malnutrition. The disease would appear to depend upon some affection of the respiratory center. No **lesions** have been found, except in the case of Kirkwood and Myers, in which hemorrhages of microscopic size were discovered just above the level of the olivary body.

The **symptoms** consist of repeated attacks of sudden failure of respiration. Without warning, and while the infant is lying quietly, there is a sudden cessation of breathing, followed by pallor or a leaden color of the skin. Breathing returns after artificial respiration is employed, and in a short time the infant seems again perfectly normal; but in a few hours a second attack develops and is relieved as before. This process continues for a day or two, or possibly even for a week or more, with several attacks each day; and finally, as a rule, the infant dies in an attack. This occurred in 4 of Still's 5 cases, and in that of Kirkwood and Myers, as well as in our own.

Treatment is difficult since the attack comes on without any warning. Consequently constant watching is the only thing possible, in order that artificial respiration and the administration of oxygen may be begun immediately.

[1] Med. Jour. and Rec., 1925, CXXII, 40.
[2] Lancet, 1923, I, 431.
[3] Lancet, 1923, II, 65.

CHAPTER IX

PULMONARY ATELECTASIS IN THE NEW-BORN

Etiology.—The name denotes a persistence of, or a return to, the unexpanded fetal condition of the lungs. It is peculiarly a disease of the new-born, although under certain circumstances it may develop in older subjects. This acquired atelectasis will be considered later (Vol. II, p. 346). In some cases the infants are born asphyxiated, respiration is absent or incomplete, and the lungs never expand properly even under the influence of artificial respiration. The asphyxia and the atelectasis may then be regarded as due to the same cause. (See Asphyxia, p. 312.) In other instances, especially in premature children or those who are weakly from other reasons, respiration is established for a time and there is at first no evidence of asphyxia. Soon, however, the poorly distended lungs become gradually more and more collapsed, owing to the weakness of the child and the yielding character of the thorax, which make satisfactory respiration difficult or impossible. Asphyxia then develops as the result of the atelectasis.

Pathological Anatomy.—In complete atelectasis, where the infant has never breathed, the lungs are dark-red or blue-red, small, very vascular, entirely collapsed, firm to the touch, do not crepitate, and sink in water. They are capable, however, of being forcibly inflated without difficulty, thus distinguishing the condition from pneumonic consolidation. If the lungs have been partially inflated during life, either through respiratory efforts of the infant or through attempts at artificial respiration, post-mortem examination shows small scattered areas of normally distended or emphysematous pulmonary tissue, characterized by the lighter color and the evident presence of air. These are oftenest seen in the upper portion of the lung or at the anterior edges. Where life has continued some weeks the anterior parts of the lungs may be found entirely distended or even emphysematous, while the posterior portions, especially the lower lobes, are completely atelectatic; or perhaps the surface seems normal but the portions beneath it are collapsed and the lung is hard. There is a striking contrast between the areas of healthy or distended lung and the depressed, dark, atelectatic portions. Both lungs are generally involved, although not necessarily equally so. Quite frequently more or less hypostatic pneumonia is combined with the atelectasis. In some cases one area may be pneumonic and another atelectatic. Other lesions are those described under Asphyxia.

Symptoms.—In the cases in which the children are asphyxiated at birth, and either show little or no tendency to recovery or relapse promptly, the symptoms of atelectasis are those already described under Asphyxia. In those in which the atelectasis develops later, being dependent upon the excessive weakness of the infant, or in which asphyxia, although present at birth, has apparently been recovered from, the symptoms of atelectasis are characteristic. The pulse is weak and slow; the respiration is rapid, irregular and shallow, and attended by evident retraction of the intercostal spaces and the lower portion of the thorax at the insertion of the diaphragm. The infant is somnolent and lies generally with eyes closed, crying but little and never loudly, and making feeble, if any, efforts to suck. The temperature of the body is persistently or only at times subnormal. The physical examination of the lungs generally gives a somewhat impaired percussion-note over the lower posterior portion of the chest. Sometimes, however, no distinct alteration of note can be discovered if numerous inflated areas of pulmonary tissue are present. The respiratory murmur is feeble, with fine râles at times, but as a rule no bronchial breathing is discovered.

This condition may continue for weeks. Recovery may take place after several relapses, or death may occur suddenly, often with convulsions and without there having been any very positive sign which indicated the actual state of the lungs.

Prognosis.—When the disease is present at birth and is accompanied by asphyxia livida, and where no congenital malformations or other pathological conditions are present the prognosis is generally favorable. When atelectasis occurs in premature infants the result is more uncertain on account on the constant danger of relapse. Yet very great care and watchfulness will often succeed in saving life.

Diagnosis.—The disease is to be distinguished principally from the hypostatic pneumonia which is liable to develop in weakly infants. The absence of fever and of bronchial respiration constitute the chief diagnostic evidences against pneumonia.

Treatment.—Although varying with the nature of the causes and symptoms, treatment is practically identical with that recommended for asphyxia. Cutaneous stimulation, artificial respiration, the maintenance of the bodily temperature, and the frequent administration of nourishment are indicated. (See Premature Birth, p. 284 and Asphyxia, p. 312.) It must never be forgotten that atelectatic infants must have an abundance of fresh warmed air and must not be allowed to lie too long in one position. They should be roused from their somnolent condition at frequent intervals and carried about cautiously; and be given massage, warm baths and similar measures to stimulate both circulation and respiration.

CHAPTER X

CONGENITAL ASTHENIA

In this category belong the infants born with a low weight, and with a power of resistance to deleterious influences and a capacity of thriving under ordinary circumstances much below normal. The condition is different from that of premature birth, although often associated with it. Premature birth is doubtless the most frequent cause, but not every case of prematurity exhibits debility, while, on the other hand, infants born at full term may be suffering from asthenia. Such cases of congenital asthenia may be the result of prenatal influences such as prolonged illness of the mother, and particularly such conditions as syphilis, tuberculosis, alcoholism, and the like. In some of these the baby, although born at term, is physically premature, the maternal condition having interfered with intra-uterine development. In other cases the fact that the infant is one of multiple births accounts for the asthenia. The condition is much the same whatever the cause, and the symptoms and treatment are fully considered in the chapter on Premature Birth. There is often a difference, however, in the prognosis. If a prematurely born infant with asthenia dependent upon no maternal disease can be maintained alive until its organs reach the power of functionating properly, the prognosis is good; whereas the infant with constitutional debility, perhaps depending upon prenatal influences, may show little increase of energy as time passes.

CHAPTER XI

DISEASES OF THE UMBILICUS

Affections of the navel are among the common pathological conditions of the new-born. In 2603 births Porak and Durante[1] found some abnormality in 832 or 32.6 per cent. although this was of an important nature in but 333; viz., 12.8 per cent. All the severer forms have become much less frequent since antisepsis has been practised more perfectly. The studies of Laurinsich[2] show that ordinarily the umbilical stump and surrounding area contain few bacteria, and that these are chiefly saprophytes. Even in the cases where pathogenic organisms are found there is not necessarily any inflammation present.

The various umbilical disorders, with the exception of hernia which will be discussed later (Vol. II, p. 264) may be subdivided as follows:

DELAYED HEALING OF THE UMBILICUS

(Excoriation; Blennorrhea; Umbilical Ulceration, etc.)

Under this title Runge[3] properly groups several minor affections of the umbilicus which, although often described as distinct conditions, are yet clearly allied.

In place of skinning over with epithelium a few days after the fall of the cord, the umbilical wound sometimes projects slightly and becomes irritated by the dressing applied, constituting the so-called **excoriatio umbilici.** If a flat, red surface is visible, resembling mucous membrane, and secreting pus more or less abundantly, **blennorrhea of the navel** is spoken of. Should the process extend in area or in depth a condition of genuine ulceration is produced with a granulating surface, sometimes covered with necrotic tissue and constituting an **ulcus umbilici.** This ulcer may develop a false membrane, and is then entitled **croup** or **diphtheria of the navel,** without the process necessarily being of a truly diphtheritic nature.

Etiology.—The causes of this delayed healing are various. There is a certain normal variation in the falling of the cord and the healing of the umbilical wound. An average time for the separation of the cord is the 5th day, and for the healing of the wound the 12th to the 15th day. In strong children the degree of reactive inflammation is liable to be more intense than in weakly ones, the cord falls sooner, and the wound heals more rapidly. It is only when the inflammation increases after the cord has separated that the process can be called abnormal. In some cases local irritation by the dressing is the cause of the inflammation. Lack of cleanliness is another factor. In many instances a local infection is without doubt present, and this is probably nearly always the case when ulceration develops. It is also likely that infection has taken place if decided increase of redness and secretion is combined with a much delayed separation of the cord. In delayed healing the possibility of a diphtheritic infection of the stump must be thought of. This is probably not very rare, and cases have been reported by Henkel[1] Foth[2] and Comby.[3]

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[1] Arch. de. méd. des enf., 1905, VIII, 465.
[2] LaPediat., 1924, XXXII, 1022.
[3] Krankh. d. ersten Lebenst., 1893, 71.

Symptoms.—No general symptoms attend these different forms of delayed healing of the umbilical wound, and no danger to life exists provided they remain purely local. Even ulceration of the umbilicus is without involvement of the general health unless it has lasted a considerable time. Where there are decided constitutional symptoms accompanying the umbilical lesions described, infection has certainly extended beyond the umbilicus.

Treatment.—Inasmuch as the persistence of the umbilical wound continues the liability to septic infection, treatment is necessary. As a prophylactic measure the stump of the cord must be kept as dry as possible in order to hasten mummification and separation. Should there be delay in healing after the cord has separated, dressings with some antiseptic powder are to be preferred. After a thorough cleansing with water followed by careful drying, powdered boric acid may be applied in a thick layer, or a mixture of salicylic acid and starch or talc in the proportion of 1:5, and this covered with a wad of absorbent cotton and a bandage. Washing with hydrogen dioxide, and painting with a 1 per cent. solution of nitrate of silver are often also of service. The dry dressing should be renewed once or twice daily. In diphtheria of the stump antitoxin should be applied locally and 500 to 1000 units given subcutaneously.

FUNGUS OF THE UMBILICUS

(Granuloma; Sarcomphalos; Umbilical Polypus)

After the separation of the cord an abnormal growth sometimes develops at the navel. This may be hidden by the overlying skin, or may project as a round, red mass the size of a pea or larger, exhibiting a granular appearance on close inspection. The surface of the mass is moist, discharges sero-purulent fluid, and bleeds slightly when irritated. Unless treated the growth may persist for months. The general health of the infant is unaffected.

Anatomically it is composed of granulation tissue which has developed upon the point of attachment of the cord. The condition can always be recognized if the folds of the skin about the navel are pulled apart in a way to render the interior visible. It is to be distinguished only from a persistent Meckel's diverticulum (Vol. II, p. 185). The tendency to recovery unaided is slight, and the umbilical wound cannot heal while the fungus is present. Treatment consists in the application of astringents, preferably nitrate of silver, followed by a dressing of powdered boric acid. This may be repeated every few days if necessary. If the fungus is of considerable size it may be ligated. When exuberant granulations have disappeared an antiseptic dressing may be applied until the wound becomes covered with epithelium.

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[1] Deutsch. med. Wochensch., 1919, XLV, 1411.

[2] Deutsch. med. Wochenschr., 1921, XLVII, 1261.

[3] Arch. de méd. des enf., 1921, XXIV, 437.

OMPHALITIS

(Periomphalitis)

Etiology.—The term is used to designate a phlegmonous inflammation of the navel and surrounding tissues. It is not of frequent occurrence, Hennig[1] finding but 12 cases among 7000 sick infants. The cause is a pyogenic infection, to which uncleanliness and lack of proper care of the navel have predisposed.

Symptoms.—The disease begins usually in the 2d or 3d week of life, after separation of the cord and as a sequel to a delayed healing of the umbilical wound, which may exhibit ulceration with secretion of pus. The skin around the navel is swollen, red, hot, shining and projecting, with a disappearance of the normal folds. The subcutaneous tissue is infiltrated and hard, and the slightest pressure causes intense pain. The inflammation may remain comparatively superficial, or may extend in depth, even involving the peritoneum. It may be confined to the neighborhood of the navel, or may attack the greater part of the abdominal wall. In some such cases it is very probable that an erysipelatous infection has been added to the omphalitis, or has been present from the beginning. The general condition of the infant is always affected. There is restlessness, loss of appetite, and fever. Pain is a prominent feature, and on this account there is little movement of the abdomen or lower limbs, the thighs are generally held rigidly flexed on the abdomen, respiration is superficial, and loud crying is avoided. The emptying of the bladder and of the bowels is also painful.

Course and Prognosis.—The duration and course of the disease is variable. In the mild cases the exudate is rapidly absorbed. A few pustules or quite small cutaneous abscesses may form in the vicinity and recovery is complete in a few days. The severe cases may last for weeks, the infiltration being slowly absorbed, or an abscess developing and discharging. The prognosis is always dubious. Those cases generally recover in which the disease spreads but little. Where the process is intense the prognosis is grave. The more quickly the severer cases advance to the formation of abscess the better for the child, since the duration of the inflammation and the consequent exhaustion are thus curtailed. The development of a complicating gangrene or the extension of the lesion to the peritoneum makes the prognosis very unfavorable. If the inflammation involves the umbilical vessels general septic infection will probably follow.

Diagnosis.—This is as a rule rendered evident by the pain and the infiltrated, hard, prominent umbilical region. The formation of an abscess is shown by the presence of fluctuation. Erysipelas is generally distinguished by the rapid spreading of the characteristic color. Yet when erysipelas exists as a complication the diagnosis of the existence of a primary omphalitis becomes most difficult.

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[1] Gerhardt's Handb. d. Kinderkr., II, 131.

Treatment.—This is first of all prophylactic. The navel must be kept aseptic and free from irritation. If omphalitis has already commenced, the umbilical wound should be cleansed carefully and then powdered thickly with boric acid, iodoform, salicylic acid and starch (1:5), or other antiseptic powder. If there is much infiltration of the tissues and abdominal pain, the inflamed area may be covered with a warm, wet antiseptic dressing. A diluted ichthyol ointment, 5 to 10 per cent. is sometimes useful. Any abscesses which form should be incised early. Attention must be given to the nourishment of the infant, forced feeding being used if necessary, and stimulants being required in many cases. The bowels should be emptied by injections, as the child cannot make any effort at pressing.

GANGRENE OF THE NAVEL

Etiology.—Gangrene may be a sequel to severe cases of omphalitis, ulcer, or inflammation of the umbilical vessels. It is now very rare as a purely local affection, since better antiseptic precautions are observed; but is still seen, though infrequently, as a secondary manifestation of a general septic infection. It may exceptionally follow severe general diseases, especially diarrhea of a choleraic nature, even in children over a month old and previously healthy. The existence of great debility, as in cases of premature birth, favors its development. An especially potent factor is lack of cleanliness about the umbilical wound.

Pathological Anatomy and Symptoms.—The inflammation already present develops into a greenish or black offensive mass surrounded by a red areola, and the edges of the wound become discolored and break down, causing a more or less rapid loss of substance, either in area or in depth. In the latter case the process may extend to the peritoneum, and even into the intestine, producing perforation and fecal fistula. If the spreading is toward the periphery the greater part of the superficial abdominal wall may be destroyed, involving more or less the muscular layer and even extending to the bladder. Severe hemorrhage may occur if the umbilical vessels are involved. General sepsis may be produced by way of the vessels or of the peritoneum. The constitutional symptoms attending gangrene are always severe. There are great prostration with coldness of the extremities, quick and weak pulse, and little or no fever. Rapid collapse is frequent. Occasionally the process is not so serious, the gangrene may not extend far, and, the reactive inflammation producing pus, the dead tissue is thrown off and the cavity fills with granulations. The diagnosis offers no difficulty.

Course and Prognosis.—The duration varies, depending upon the resisting power of the infant and the swiftness of the spread of the disease. The average duration of fatal cases, according to Fürth[1] is about 5½ days, and of those which recover about 22 days. The prognosis is exceedingly bad. If the diseased area is small, the infant's strength good, and reactive inflammation soon sets in, recovery may follow, but the mortality reaches over 85 per cent. (88.48 per cent., Fürth). Absence of the surrounding areola, involvement of the peritoneum, severe hemorrhage from the umbilical vessels, and development of general sepsis render death almost inevitable. Gangrene arising as an affection secondary to constitutional conditions and other diseases is uniformly fatal.

Treatment.—This consists in supporting the strength by means of abundant nourishment and powerful stimulation, the maintaining of the temperature, the limiting the spread of the disease, and the favoring of the casting off of the dead tissue and of the development of a reactive inflammation. Warm wet antiseptic dressings may be applied. The early employment of the thermo-cautery may be efficacious. After the gangrenous tissue has separated iodoform or ichthyol may be used.

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[1] Wiener Klinik, 1884, X, 331.

UMBILICAL ARTERITIS AND PHLEBITIS

Etiology.—Both of these conditions are of comparatively unusual occurrence. The investigations of Runge[1] show that inflammation of the arteries is far more common than that of the vein. In 55 cases of disease of the vessels the vein was found affected but once, while in all instances the arteries were involved. Of all affections of the navel arteritis appears to be much the most frequent cause of death. Phlebitis, too, usually leads to general fatal septic inflammation. The cause of disease of the umbilical vessels is always an infection of the umbilical wound. It is most likely to occur before the stump of the cord has completely separated, since the granulations which develop later form to some extent a protection against infection. It may appear as an epidemic in lying-in institutions, although this has become very much less frequent since better methods have prevailed. The infection may be acquired from the lochial discharges of the mother, or be transmitted by the hands of the physician or nurse, infected umbilical dressings, or even apparently by the air of an infected room or the water used for bathing. Any irritation of the umbilical wound predisposes to it, as does omphalitis or gangrene of the navel. On the other hand the vascular infection may occur without the umbilical wound showing anything abnormal.

Premature infants seem especially predisposed to the disease. The presence of moisture and decomposition in the stump of the umbilical cord is more favorable to the development of organisms than the normal state of mummification (Cholmogoroff).[2] Various organisms have been found in the stump of the cord and in the umbilical wound in cases of infection of the vessels, among these being varieties of staphylococci, the streptococcus pyogenes, the bacillus coli, the pneumococcus, and the bacillus pyocyaneus.

Pathological Anatomy.—The process always begins as an infection and inflammation of the perivascular tissue, according to the statements of Runge,[3] which becomes infiltrated and swollen. It next extends to the vessel-walls and a septic thrombus forms in the vessel. The disease usually remains local, resulting in suppuration in or about the affected portion of the vessel or between the abdominal wall and the peritoneum, and the final discharge of pus. In a smaller number of cases the process spreads by continuity along the perivascular tissues, the sepsis reaching the general system through the perivascular lymph channels.

On inspection at autopsy the diseased arteries are visible as thickened, stiff and discolored cords, surrounded by edematous infiltrated connective tissue. The vessels of the adventitia are abundant. Nearly always both arteries are involved either for a short distance only or throughout their extent. They contain broken-down thrombi, partially greenish in color from the purulent changes, partially still bloody. The intima of the vessel is without the natural shining character and exhibits loss of endothelium. The lesions of the umbilical vein are identical with those of the arteries, and the process usually extends throughout its entire length up to the liver and even into it. The umbilical wound may appear perfectly normal or may exhibit the lesions of ulcer or of omphalitis. Even if the stump of the cord is mummified and still persists, foci of suppuration may be discovered about the periphery of the region of beginning detachment. Some of the lesions of a general septic infection may be present, such as have been described in considering Sepsis in the New-born. (See p. 291.) In the case of phlebitis the liver may be involved directly by continuity from the umbilical vein.

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[1] Krankh. d. ersten Lebenst., 1893, 88.

[2] Zeitschr. f. Geburtsh. u. Gynäk., 1889, XVI, 16.

[3] *Loc. cit.*

Symptoms and Diagnosis.—Characteristic symptoms of the disease, apart from the pathological lesions, are practically absent. When the process remains a local one, as is true of the majority of cases, the symptoms are chiefly local. If the portion of the vessel immediately beneath the navel is involved, crusts may appear upon the umbilical wound, or there may be discharged from it on pressure pus which clearly comes from beneath the surface. In the cases where a portion of the vessel more deeply situated is the seat of the lesion, the navel is of normal appearance. In some such instances the hard and thick arteries can be felt through the abdominal wall. Sometimes abscess forms above the peritoneum and may make its way in different directions. Moderate fever and other constitutional disturbances may be present, but frequently no general symptoms whatever are discovered; and it may well happen that death occurs unexpectedly, and only at autopsy is an umbilical arteritis found. In the more severe cases, in many of which the vessels are involved throughout much of their extent and general sepsis follows, there is often no disorder of the navel visible, the symptoms are very uncharacteristic, and there are no positive means of recognizing the disease during life. Without any apparent reason, or with no previous evidence of illness, the infant becomes restless, loses appetite, develops irregular and often high fever, and passes rapidly into collapse. Less frequently the duration is more prolonged, and symptoms of grave constitutional disturbance are present. High fever and collapse alternate and wasting becomes great. There is diarrhea and distended and tender abdomen. Intense icterus is constantly present in umbilical phlebitis if the condition is at all prolonged (Widerhofer),[1] and is frequent in arteritis also. In neither case is there any reason to justify the diagnosis of extensive inflammation of the umbilical vessels unless the umbilical wound is distinctly diseased, in which event the suspicion is warrantable, although proof is absent. When evidences of diffuse general pyemia develop, the diagnosis of an umbilical arteritis or phlebitis is rendered still more probable, although not even then certain.

Complications.—Pneumonia is that oftenest seen, and may be the only one. It occurred in 22 of Runge's 55 cases. All the wide-spread local pyemic lesions of septic infection may occur as complications, among these empyema, peritonitis, subcutaneous abscess, nephritis, and the like. (See Sepsis in the New-born, p. 291.) Erysipelas may sometimes develop as a complication.

Course and Prognosis.—As we cannot discover the time when the disease begins the exact duration is uncertain. In nearly all instances it is short, ranging from a few days to several weeks. The greater number of deaths in Runge's cases occurred on the 8th day of life. The prognosis is always grave. Since the diagnosis can be made with certainty only at the autopsy, it is impossible to determine the fatality of the disorder, but it is probable that recovery does occur in many instances if the process does not spread beyond the vessels. Should the sepsis become general, death is almost inevitable.

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[1] Jahrb. f. Kinderheilk., 1862, V, 195.

Treatment.—Prophylaxis is the most important. Careful antiseptic treatment of the umbilicus is necessary from the moment of birth. The hands of the physician and nurse must be aseptic, the scissors, ligature and dressings likewise so, and precautions taken at all times against the access of germs. The child should be at once removed from the vicinity of the mother if she has any evidence of sepsis. The stump of the cord must be kept as dry as possible in order to avoid a moist decomposition, and the frequent renewal of antiseptic dressings may be necessary to ensure this. The use of sterilized absorbent cotton with a powder of salicylic acid and talcum (1:5) is useful for this purpose. The employment of powdered gypsum on cotton has been recommended by Sutugin,[1] Cholmogoroff[2] and others as the best means of accomplishing this. Such a dressing should be used as will not shut out air too greatly, since rapid drying is hindered in this way. When the cord has separated the wound should be dressed with the powder of salicylic acid and talcum, or one of salicylic acid and starch, (1:5) or with boric acid or iodoform. If the navel is already diseased in any way the treatment indicated for the condition present must be employed in order to prevent the spread of infection to the vessels.

OMPHALORRHAGIA

(Umbilical Hemorrhage)

(See also Hemorrhage in the New-born, p. 297)

This, like melena, is not a distinct disease, but a symptom of different conditions, itself of enough importance to warrant separate consideration. It is customary to recognize 2 forms (*A*) *accidental*, proceeding from the umbilical vessels, and (*B*) *idiopathic* or *spontaneous* coming from the umbilical tissues.

Etiology.—(*A*) ACCIDENTAL HEMORRHAGE.—Severe hemorrhage from the umbilical vessels may occur before the fall of the cord as a result of imperfect ligation of it. The ligature may have been too loose, or may have slipped, or have cut into the vessels. Under otherwise normal conditions hemorrhage from such causes will not take place except in the first 10 or 15 minutes of life. When, however, under the influence of well-developed asphyxia in otherwise healthy infants, or especially in the slow, unsatisfactory expansion of the lungs in premature infants, the blood is not drawn into the expanding lungs in a normal manner, there results a maintenance of the blood-pressure in the umbilical arteries, the muscle of the arterial walls fails to contract and close the lumen properly, and the occurrence of hemorrhage is possible. In like manner gangrene of the umbilicus or imperfect mummification allows the vessels to open again and blood to escape. It is possible, too, that a bath too prolonged and too hot may relax the vessels and permit of hemorrhage. Hemorrhage very rarely results from rupture of the cord during birth in the case of healthy children.

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[1] Wratsch, 1883, No. 44, Ref. Cholmogoroff, *loc. cit.*, 19.

[2] *Loc. cit.*, 28.

After the separation of the cord a slight oozing from the umbilical vessels is not uncommon. This occurs oftenest when the umbilical wound is healing slowly or has been roughly handled. Rarely the bleeding is severe.

(*B*) Idiopathic Hemorrhage.—Bleeding of this variety from the umbilical wound, decidedly more common than from the vessels, is a very dangerous affection, the causes of which are diverse and little understood. Grandidier[1] writing in 1871 collected 220 cases from medical literature. Townsend[2] reported 14 cases in 7225 births. Winckel[3] observed it in only 1 of 5000 births. The condition would appear perhaps to be more frequent than these statistics indicate, inasmuch as many cases are never reported. It occurs oftenest after the complete separation of the stump of the cord. Males exhibit it oftener than females. The general condition of the infant is of doubtful influence. It is possible that poor health of the mother during pregnancy may predispose to the development of the disease, but this, too, is doubtful. Certainly congenital syphilis appears to be a prominent cause of umbilical as of other forms of hemorrhage in the new-born. Septic disease of the new-born also produces it. (See p. 291.) Epstein[4] found well-marked sepsis in 24 out of 51 fatal cases of hemorrhage. The presence of microörganisms plays an important part in the causation of many cases. Among those found in the blood or at autopsy are the streptococcus pyogenes, pneumococcus, staphylococcus pyogenes, bacillus pyocyaneus, colon bacillus, and others (Abt).[5] Numerous cases, however, have shown no evidence whatever of sepsis. Umbilical hemorrhage is one of the symptoms of acute fatty degeneration, and especially of the hemorrhagic disease of the new-born previously referred to (p. 298). The influence of hemophilia is negligible, since this disease rarely shows itself until later in life. Larrabee[6] was able to collect but 23 cases from medical literature in which umbilical hemorrhage could be considered as dependent upon hemophilia.

Symptoms.—Severe **accidental hemorrhage** is usually sudden and profuse, and may terminate life quickly unless checked at once. The blood can often be seen flowing directly from an open vessel. It occurs generally a few minutes after birth, or, in the case of premature infants with evidences of asphyxia, during the first few hours of life, or occasionally later; sometimes even after the cord has fallen. After the separation of the cord the hemorrhage is usually in the form of oozing only. This, too can often be seen to come from a vessel.

No general symptoms attend the accidental hemorrhage other than those of anemia, nor is there bleeding from other parts of the body.

Idiopathic hemorrhage usually occurs somewhere between the 5th and the 10th days of life, although it may take place earlier or later than this. It is very frequently combined with the occurrence of bleeding in other parts of the body. Beginning slowly it becomes fairly free and saturates the dressing, yet it appears to be capillary in origin. Sometimes it is profuse from the start. A characteristic of the disease is that effort to check the bleeding has but a temporary effect, if any. Very occasionally the hemorrhage stops of itself for a time and then is renewed. The blood has little tendency to coagulate, but microscopic examination shows nothing abnormal in it. The infants may appear at first perfectly healthy in every other respect, or may have seemed not quite well before the hemorrhage began, or have looked ill and been somnolent or cyanotic or exhibited diarrhea, vomiting or decided icterus. In all cases distinct evidence of general disturbance appears sooner or later, apart from the results of the loss of blood. Icterus often becomes intense; cyanosis may develop, if not earlier present; and hemorrhage from other parts of the body often occurs, or in other instances may have antedated the umbilical hemorrhage. Not infrequently bleeding takes place not only from the navel, but into the subcutaneous tissues about it. Edema of the wrists and ankles is common. As the hemorrhage continues anemia becomes profound and death occurs, sometimes preceded by coma or convulsions.

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[1] Die freiwilligen Nabelblutungen d. Neugeb., 1871. Ref. Runge, Die Krankh. der ersten Lebenst., 1906, 224.

[2] Bost. Med. and Surg. Jour., 1891, CXXV, 218.

[3] Lehrb. d. Geburtsh., 1893, 854.

[4] Oester. Jahrb. f. Pädiat., 1876, VII, 139.

[5] Jour. Amer. Med. Assoc., 1903, XL, 284, for literature.

[6] Amer. Jour. Med. Sci., 1906, CXXXI; 497.

Prognosis.—The prognosis in accidental hemorrhage is favorable if the bleeding is not too profuse at the beginning and is discovered and checked at once. In idiopathic hemorrhage it is unfavorable. Statistics give from 25 to 35 per cent. of recoveries only. When the disease depends on sepsis, acute fatty degeneration, or syphilis, practically no hope of recovery exists. Death may occur in less than 24 hours and the average duration of life from the onset in fatal cases is only 2 to 3 days, and is seldom longer than 2 weeks.

Diagnosis.—The distinction is to be made only between the two varieties of umbilical hemorrhage. Careful inspection and the course of the case will generally make the matter clear.

Treatment.—Prophylactic treatment for **accidental hemorrhage** consists in the proper ligation of the umbilical cord not too close to the body (see page 64), in gentle handling of it afterward and in care in the removing of all evidences of asphyxia as soon as possible. Should hemorrhage occur a new ligature must be tied at once. If there is no room to apply this, or if there is free hemorrhage after the separation of the cord it may be necessary to apply a compress or even to push two needles through the skin, above and below the seat of bleeding, and ligate around these. If the bleeding is very slight the application of an astringent powder or the use of compresses moistened with tr. ferri chloridi or liquor ferri subsulphati may suffice. For the anemia which may have developed are to be employed free stimulation, abundant nourishment, the maintaining of the heat of the body, and later, measures to aid the enriching of the blood.

In the case of **idiopathic hemorrhage** prophylaxis consists, first, in attending carefully to the general health of the mother before the birth of the child and in giving to her the treatment indicated if there are any evidences of syphilis. Great care must be taken after the birth of the infant to guard against septic infection from any source. If hemorrhage begins styptic applications may be tried, such as alum or tannic acid, and especially the use of firm compresses moistened with tincture of the chloride of iron. These may be fastened in place with broad bands of adhesive plaster crossing over the navel and drawn firmly, finishing on the back. It has also been recommended to pour moistened plaster of Paris on the navel and allow it to remain for several days. Acupuncture with the application of a ligature about the needles may be employed in the manner already described.

The administration of a 5 per cent. solution of gelatine freely by enema or by the mouth is worthy of trial. Still better is its subcutaneous use after *very carefully repeated* sterilization (p. 260). If these procedures do not control the hemorrhage, the injection of serum or of blood may be resorted to (p. 300).

Against the anemia the measures already mentioned are indicated.

PROTRUDING MECKEL'S DIVERTICULUM

(See also Diseases of Meckel's Diverticulum, Vol. II, p. 285)

This rare affection at first sight resembles the Fungus of the Umbilicus (p. 324). It consists of a prolapse at the navel of the terminal portion of the omphalomesenteric duct which has failed to close and to disappear early in fetal life. It is generally patulous throughout, forming a fistulous tract from the umbilicus to the small intestine. In appearance it is usually a reddish tumor of glistening surface, moist with mucous secretion. Microscopical examination shows that it is of the same structure as the intestinal wall and that the surface is composed of mucous membrane. It is generally of the size of a pea or bean, although sometimes much larger, and shows a central opening through which a small amount of fecal matter may be discharged from time to time. This condition may last for years, or the duct may close spontaneously. In some cases the fistula is much larger, and the posterior wall of the intestine, and finally even quite a large portion of the bowel may project through it in the form of two intussusceptions each with its central opening.

The treatment of the protruding diverticulum, if small, consists in the application of a ligature. The larger protrusions are more difficult of cure and the prognosis is unfavorable.

CHAPTER XII

MASTITIS

The activity of the mammary glands so frequently present in the new-born (see p. 55), producing enlargement with secretion of fluid, may pass into an actual inflammatory condition, and is then known as mastitis.

Etiology.—The condition is not an uncommon one, and either sex may be attacked. As a rule it occurs only in a breast in which secretion has been free and has continued for some time. Sometimes an ignorant mother or nurse is led to squeeze or rub such a breast repeatedly, with the idea that it is necessary or that the manipulation induces a good development of the gland later in life. The irritation produced, combined with want of cleanliness, favors the entrance of germs through the ducts or through fissures and suppuration follows.

Symptoms.—Mastitis begins usually in the 2d or 3d week of life, but may occur later in infancy. The breast, generally only one, grows uniformly larger, red, hot, and painful on pressure (Fig. 50). As a rule suitable treatment employed at this period prevents the disease from advancing farther, and the inflammation subsides in a few days. In many cases, however, particularly if the squeezing of the breast is still continued, suppuration takes place and a circumscribed abscess forms. Less frequently there are multiple abscesses. Fever, restlessness, loss of appetite, sleeplessness, and loss of weight attend the suppurative process. Occasionally the inflammation spreads beyond the gland to the surrounding connective tissue, and in rare instances this peri-mastitis may involve much of the anterior and lateral walls of the thorax.

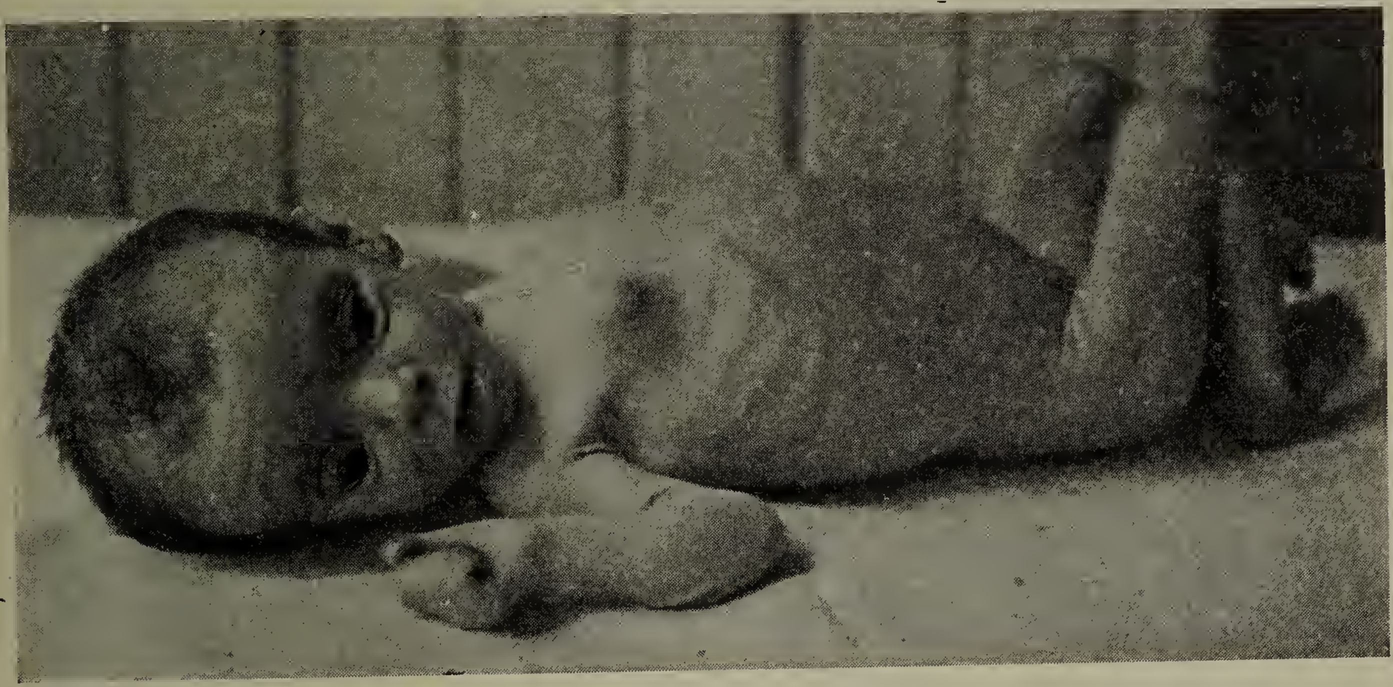

FIG. 50.—MASTITIS.
Male infant, aged 25 days.

Prognosis.—The prognosis of mastitis of the new-born is nearly always good. Even in the cases where abscess forms recovery follows unless suppuration is very extensive. Yet permanent injury may easily remain, the secreting power of the gland in later life in females being destroyed or impaired, or the nipple being retracted or otherwise distorted.

Treatment.—The disease should be prevented by careful prophylactic measures. All pressure or rubbing of the breast of the new-born is to be avoided, and cleanliness is to be enforced. If the gland is more swollen than common and inflammation is feared, a wad of aseptic absorbent cotton may be laid over it to protect from pressure and to prevent infection. If mastitis actually begins, hot, wet applications may be employed. Any abscess forming should be opened promptly and freely, and the infant given tonic treatment.

CHAPTER XIII

OPHTHALMIA NEONATORUM

Although occurring later in life as well, this form of ophthalmia is so much more prevalent in the new-born that it may properly be described in connection with the diseases of this period of life. The affection is still a common one, although less so than formerly. This is particularly true of lying-in institutions, where in earlier times it often prevailed epidemically, from 5 per cent. to even 20 per cent. of the children born being affected by it. The improvement in the statistics is shown by those of Harman[1] who found that it occurred in but 0.843 per cent. of all births in London in 1911. The greater sensitiveness of the conjunctiva in the new-born certainly predisposes to the disease.

Etiology.—Infection is the cause in all instances. This is generally acquired from the vaginal discharges at the time of birth, while the head is passing through the genital canal of the mother. In other cases the infection comes from the fingers of the physician, mother, or nurse, or from infected clothing. It is also possible for the infectious matter to penetrate beneath the lids even before birth after the amniotic sac has ruptured. Although such germs as the colon bacillus, pneumococcus and others are capable of producing the disease, yet in the great majority of cases, especially if severe, the gonococcus is the cause. Waldeck[2] reports the gonococcus present in 55 per cent. of 233 cases of ophthalmia neonatorum, and Stephenson[3] in 64.56 per cent. of 1829 cases. The milder cases may owe their origin to exposure to too bright a light, trauma, and the like, which allows a mild non-gonorrheal infection to take place.

Symptoms.—The process may affect both eyes simultaneously, or may spread from one eye to the other. Only exceptionally is but one eye involved. In the cases of a catarrhal, non-specific nature the symptoms are generally mild. They then consist of photophobia, redness of the palpebral conjunctiva, and slight serous secretion which collects in the corners or on the edges of the lids. The course is short, and the inflammation has generally disappeared in a few days.

In the gonorrheal cases the symptoms usually appear on the 3d or 4th day after birth or occasionally earlier. The first evidences are swelling and redness of the palpebral and injection of the bulbar conjunctiva, while a slight watery discharge is seen on separating the lids. The course of the disease is violent and rapid. In a few hours the lids become red, hot, stiff and extremely swollen, closing the eye tightly and being separated with difficulty by the fingers; the conjunctiva becomes much swollen with an abundant infiltration of lymphoid cells and gonococci; the secretion is somewhat more abundant and of a more purulent character; the cornea is involved, and in bad cases is liable to ulcerate, and perforation of it may occur even during the 2d day. In a few days the swelling of the lids diminishes, but the redness of the conjunctiva does not lessen, and, in cases which have not been benefited by treatment, folds and roughnesses develop on it giving the appearance of granulation tissue, while the secretion grows still more abundant and quite purulent. General symptoms are absent or slight, although considerable fever may be present in the severer cases. Recovery takes place slowly. The duration is variable, the average being 3 to 5 weeks. The severe cases run 6 to 8 weeks if not influenced by treatment.

In cases where the cornea is involved this becomes cloudy, opaque and dull in appearance, and, if ulceration occurs and is followed by perforation, the aqueous humor is discharged and the iris may prolapse. Total blindness is liable to result, or the whole eye may be destroyed by a panophthalmitis. If the ulcer does not perforate healing gradually takes place and a localized opacity develops which gradually becomes more transparent. Where treatment has been successful the severity and duration of the inflammatory process are materially lessened.

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[1] Brit. Med. Jour., 1913, I, 1099.

[2] Jour. Mich. State Med. Soc., 1922, XXI, 501.

[3] Practitioner 1914, XCIII, 374.

Complications.—Chief among the complications is the occasional development of gonorrheal arthritis. This has been recorded repeatedly. Secondary gonorrheal infection of the nose or mouth has also been observed.

Prognosis.—The prognosis of the severe form of inflammation is extremely grave as regards loss of vision in cases which have not received prompt and efficient treatment. Formerly a large number of instances of blindness were due to this disease. The statistics of Reinhardt[1] concerning the inmates of the blind asylums of Germany, Austria, Denmark and Holland combined covering the years 1865–1875, showed that 40.25 per cent. had lost their sight through ophthalmia neonatorum. Prompt and thorough treatment reduces the danger greatly, especially in the newborn. If involvement of the cornea has already commenced before treatment is begun the prognosis is graver.

Diagnosis.—The diagnosis of the gonorrheal form is based upon the rapid and great swelling of the lids and other evidences of very severe inflammation of the eye, and later upon the free secretion of pus and the discovery of the gonococcus. Diphtheritic conjunctivitis occurs very rarely in infants and can be distinguished by the presence of a false membrane and of the characteristic bacilli, as well as by the lesser degree of swelling and the absence of the granulation-like appearance of the lining of the lids.

Treatment.—Chief in the line of treatment is careful prophylaxis. The frequency of the disease in institutions has been decidedly lessened since Credé[2] urged the dropping of a 2 per cent. solution of nitrate of silver into the eyes of every child immediately after birth. Of 1160 infants treated in this manner in the Leipzig Obstetrical Clinic only 1 or possibly 2 developed ophthalmia; *i.e.* 0.086 to 0.172 per cent.; whereas earlier statistics based on 4057 infants in the same maternity had given 7.8 per cent. suffering from ophthalmia. Observations of most later investigators are in entire accord with the result obtained by Credé. The use of an antiseptic vaginal injection before labor is also to be recommended. In private practice the instillation and the vaginal douching should be employed if there exists any probability that the vaginal secretion of the mother may be gonorrheal in nature. Newer silver compounds have been recommended to replace the nitrate solution. Thus solutions of protargol (5 to 20 per cent.), argyrol (25 per cent.), neosilvol (20 to 25 per cent.) or arkase (5 per cent.) have been employed with success instead of silver nitrate, but would appear to be less certain in results. The water in which the child is bathed must never be used for the washing of the eyes. In cases where the danger of gonorrheal infection is entirely unlikely the eyes should be thoroughly cleansed after birth with cotton moistened with distilled water or a saturated solution of boric acid. To prevent the milder form of conjunctival inflammation the eyes of the new-born infant must be protected from too bright a light and from mechanical injury.

When gonorrheal ophthalmia develops in an infant in a maternity or a hospital ward the patient should at once be isolated, and the nurse in charge should have nothing to do with the other children. In all cases of the disease the cloths, cotton, and the like used about the eyes should be promptly destroyed. The sound eye, if one only is affected, must be carefully protected by covering it with a wad of antiseptic cotton and enveloping with a gauze bandage. The hands of the child must also in many cases be restrained by bandaging them to the sides or in other ways.

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[1] Zweiter europ. Blindenlehrer-Congr., Dresden, 1876 Ref. Magnus; Die Blindheit, 1883, 165.

[2] Arch. f. Gyn., 1881, XVII, 50; 1883, XXI, 179. Die Verhütung d. Augenentzündung d. Neugeborenen, 1884.

The two chief indications in treatment are the repeated thorough disinfection of the eye, and the reduction of inflammation by cold applications. In the mild cases, not of gonorrheal nature, all that is required is to wash the eye thoroughly several times a day with a saturated solution of boric acid dropped into it from a blunt-pointed medicine dropper. In the gonorrheal cases the treatment must be more energetic. The lids should be separated, and the boric acid solution instilled every ½ to 1 hour. The solution should be dropped in the corner nearest the nose with the child's head turned a little toward the diseased side so that the fluid may run into the eye, and then out as far removed as possible from the unaffected eye. Care must be taken that the fluid penetrates beneath the upper as well as the lower lid. Thorough frequent removal of pus must be accomplished in this way, since the retention of the secretion is very harmful. A solution of corrosive sublimate of a strength of 1:10,000 may be used instead of boric acid. In addition to the washing there should be instilled into the eye once a day 2 or 3 drops of a 1 per cent. solution of nitrate of silver; or a 10 to 25 per cent. solution of protargol or argyrol more frequently. To reduce the inflammation and swelling of the eye cold compresses must be applied. These are best made of little squares composed of 1 or 2 layers of soft linen cloth cooled on a block of ice, and changed every 2 or 3 minutes for 30 minutes or more, with intermissions of 2 or 3 hours. In very bad cases the application must be kept up continuously until the swelling has abated. Gonorrheal ophthalmia is, however, such a serious affection that whenever possible the treatment is more safely entrusted to an ophthalmologist.

CHAPTER XIV

SCLEREMA AND EDEMA

SCLEREMA NEONATORUM

(Sclerosis; Scleroma; Sclerema adiposum)

Probably several different conditions were formerly considered to be identical with that which we now designate as sclerema. The affection is sometimes wrongly called scleroderma, which is at present believed to be entirely distinct from it. Sclerema neonatorum in its modern sense appears first to have been described by Uzembez.[1]

Etiology and Pathology.—Although not a common disease anywhere, it is observed much more frequently in Europe than in America. Writing in 1897, I[2] was able to discover but 5 undoubted and fully detailed cases published in the United States, and to these I added a 6th, and later reported another.[3] It is most often seen in premature or in greatly debilitated new-born infants, living under very unfavorable hygienic conditions, and it has occurred oftenest in institutions for foundlings. At a slightly later period, in infants a few weeks or months old, it occasionally develops following a severe diarrheal affection or other exhausting disease. The nature of the change which takes place is undetermined. An explanation with much in its favor is that the great lowering of the body-temperature dependent upon the condition of inanition present produces a hardening of the subcutaneous adipose tissue (Knöpfelmacher).[1] Smith[2] found that in scleremic fat there is a higher percentage of fatty acids than in normal fat. That this hardening can occur might be due to the low percentage of olein and the high percentage of stearin and palmatin in the fat of infants, and the consequently higher melting point as compared with the fat of adults. Yet it is to be noted that the great majority of new-born infants who develop collapse-temperatures do not suffer from sclerema. Finkelstein and Sommerfeld[3] believe that there is a change in the colloids of the tissues. They do not think that chilling of the fat in subnormal temperatures has any etiologic influence. Various microorganisms have been considered to be the active causative agents, but the existence of any such influence is very uncertain. The etiology is still undetermined.

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[1] Ephemerides naturæ curiosorum, 1722. Ref. Hennig, Gerhardt's Handb. d. Kinderkr., 1877, II, 141.

[2] Medical News, 1897, LXXI, 428.

[3] Arch. of Pediat., 1906., XXIII, 97.

Pathological Anatomy.—There are no characteristic lesions. Incision of the skin is not followed by any exudation of blood or serum as in œdema neonatorum. The subcutaneous connective and fatty tissues appear unusually dry. Microscopical examination showed nothing whatever abnormal in a case reported by Northrup,[4] while Ballentyne[5] discovered a growth of bands of connective tissue and an atrophy of the fat-cells. The connective-tissue hypertrophy was confirmed by Sarvonat.[6]

Symptoms.—The two characteristic symptoms are fall of temperature and a hard swelling of a portion of the skin. Occasionally at birth, but oftener when the greatly debilitated child is a few days old, induration of the skin is discovered. This change begins usually in the feet and calves and rapidly spreads perhaps over the whole body. It is generally most decided in the cheeks, buttocks, back and thighs. The swollen tissues seem to have an almost stony coldness and hardness and will not pit on pressure. The limbs, and sometimes the whole body, are more or less stiff and immovable. The skin cannot be lifted from the subcutaneous tissue. It is pale, waxy, and sometimes in places discolored bluish or yellowish, resembling an old bruise. The temperature of the body is generally very low, sometimes not over 90°F. (32.2°C.) in the axilla, or even less than this. The child has an almost inaudible cry. The respiration and cardiac action are very feeble; the fontanelle is sunken; the infant becomes somnolent and will not take nourishment. Exceptionally the disease is complicated by edema (Parrot).[7] Atelectasis is very prone to develop.

Occasionally cases are not so severe, the temperature is not markedly low, there is an absence of discoloration and the child does not show so great a degree of inanition and debility. I have observed at least 3 cases of this nature, which could hardly be placed in any other category, and which were yet clearly not instances of the typical sclerema described. To such cases the title *scleredema* might be conveniently applied.

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[1] Jahrb. für Kinderheilk., 1897, XLV, 177.
[2] Jour. Cutan. Dis., 1918, XXXVI, 436.
[3] Monatsschr. f. Kinderheilk., 1923, XXV, 105.
[4] Transac. Amer. Pediat. Soc., 1889, 140.
[5] Antenatal Pathology and Hygiene, 1902, I, 75.
[6] Arch. de. méd. des enf., 1906, IX, 22.
[7] La clinique des nouveau-nés, 1877, 127.

Prognosis.—This is extremely unfavorable in typical cases. The great majority die in a few days. Occasionally when the disease is not too extensive, the general condition of the infant better, and treatment commenced early, recovery will slowly take place. This was true of the 3 personal cases referred to.

Diagnosis.—Sclerema is to be distinguished by the adherence of the skin to the subcutaneous tissues and the fact that it cannot be pitted, the very low temperature, and the marked rigidity of the body. The first two symptoms serve to distinguish it from edema. Scleroderma is generally considered a disease of adults, but it is possible that some of the milder cases called sclerema may properly be placed in this category. In scleroderma the process of hardening is more local, the course is chronic and there are no general nutritional symptoms or fall of temperature. (See studies by Cockayne[1] and by Mayerhofer.[2])

Treatment.—The only treatment possible is the maintenance of the bodily temperature, the employment of general massage with oil, and the sustaining of life by food and stimulants. The continuous application of hot water-bags to the affected regions has been of benefit in some of the milder cases, and Wolff[3] has had good results in severer ones by submitting the infant to a continued temperature of 40° to 42°C. (104° to 107.6°F.). Bourne[4] reports cure in an infant with sclerema whom he treated by giving ¼ grain (.016) of thyroid extract daily combined with external heat. The original cause, if discovered, and complications, ecially atelectasis, must receive treatment appropriate for them.

ŒDEMA NEONATORUM

(Scleredema; Acute Edema)

Although edema from various causes may develop in infancy, there is a form oftenest seen in the new-born to which the name Œdema Neonatorum is given. This condition is considered by some writers to be an edematous form of sclerema.

Etiology.—The disease is uncommon. In some cases septic infection appears perhaps to have been responsible and in others the existence of a specific infectious agent has been suspected. Exposure to cold has also been considered to be sometimes the cause. It is more likely to develop in weakly or premature children, in foundling asylums, or under bad hygienic conditions, but hearty infants are exceptionally attacked. Disturbance of the respiratory and circulatory functions and beginning nephritis have each been considered the causative agent.

There has further been described by Schridde[5] and the observation confirmed by others (Chiari;[6] Wienskowitz[7]) a special form of widespread edema which is congenital and which has been designated by Chiari "fetal erythroblastosis," characterized by a universal anasarca; hydrops of the serous cavities; enlargement of the liver and spleen; a very large number of erythroblasts in the blood, with a great diminution of other elements; numerous erythroblasts in the liver, spleen, kidneys and other organs; and an abnormal deposit of hemosiderin in the spleen and liver. This form of edema is seen chiefly in premature infants born of mothers with nephritis, which has been the cause probably of a toxic disturbance of the blood-making functions of the child. Still another form is a chronic genital edema occurring in premature new-born male infants, or at a slightly later period. This will be discussed elsewhere (Vol. II, p. 488).

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[1] Brit. Jour. Child. Dis., 1916, XIII, 225.
[2] Jahrb. f. Kinderheilk., 1915, LXXXI, 348.
[3] Monatsschr. f. Kinderheilk. Ref., 1914, XIV, 66.
[4] Lancet, 1922, I, 368.
[5] Münch. med. Wochenschr., 1910, LVII, 397.
[6] Jahrb. f. Kinderheilk., 1914, LXXX, 561.
[7] Berl. klin. Wochenschr., 1914, LI, 1725.

Pathological Anatomy.—The skin is hard as in sclerema, but incision into it after death allows a serous fluid which is colorless, or of a slightly bloody tint to exude from the subcutaneous tissue. The other findings in the skin are uncharacteristic and vary with the case. The lesions of the form described by Schridde have already been mentioned. The alterations in other parts of the body are chiefly those of atelectasis and inanition.

Symptoms.—In the congenital cases a universal edema is seen at birth. In other cases the disease usually begins in the first 3 or 4 days of life, although sometimes not until the infant is some weeks or even 1 or 2 years old. It is ushered in, as a rule, by swelling of the calves, which rapidly spreads over the lower extremities and in the course of at most 2 or 3 days over the rest of the body. The skin is pale, shining and tense. Pressure gives a doughy sensation and produces pitting. The skin is not immovable over the underlying tissues as in sclerema. The limbs are very cold, stiff and difficult to move; the face is stiff and expressionless. The infant is in a condition of collapse, with feeble pulse and respiration, feeble cry, inability to suck, very low bodily temperature, and diminished secretion of urine. At first there is restlessness, but finally a soporous state. In fatal cases the condition gradually grows worse with rapid wasting, and the child dies after a few days, or at most 1 or 2 weeks. The disease may be complicated by pneumonia, atelectasis or septic conditions. It may also occur in connection with sclerema. Icterus and gastrointestinal disturbances may be present.

Prognosis.—The prognosis of œdema neonatorum is, as a rule, bad. Cases recover but rarely.

Diagnosis.—The disorder resembles sclerema in many particulars. It is distinguished from it, however, by the ability to pit the skin and to move it over the underlying tissues. It is to be remembered also that an edema is seen in erysipelas and in acute nephritis in the new-born, and is not uncommon in advancing marantic states in many infants. In the latter condition, however, the dropsy comes on more slowly, and is situated oftenest about the ankles, hands and scrotum, although frequently in the face as well. The temperature, too, is usually not so low, and the cause of the edema is generally evident.

Treatment.—The condition can better be prevented than cured Feeble children must be kept warm and nourished in the best manner possible, and all depressing diseases treated energetically. If edema has already developed these same methods are still to be employed. Alcoholic stimulation and remedies to sustain the heart's action are necessary, together with gentle massage of the body with oil. Complications require treatment appropriate to them.

CHAPTER XV

TRANSITORY FEVER IN THE NEW-BORN

(Inanition-fever; Hunger-fever Thirst-fever, etc.)

The natural irregularity of the temperature in the early weeks of life readily predisposes to a febrile elevation from very slight causes. Temporary rises of temperature are probably much more common than is usually supposed; the failure to detect them being the result of the common absence of any systematic frequent temperature-taking in infants who appear to be in health. Thus Lo Cicero[1] by making observations on 83 infants every 3 hours during the first 8 days of life found that in 49 there were sudden rises of temperature to from 37.5°C. to 39°C. (99.5° to 102.2°F.), with sudden falls, without any objective signs except somnolence or sometimes restlessness. Eröss[2] noted elevation of temperature in 431 out of 956 new-born infants, viz.; 45.08 per cent. Other statistics would place the incidence of febrile reaction in the new-born as low as 4 per cent.

The fever usually appears from the 2d to the 4th day of life. The elevation may last but a few hours or continue for several days. (Fig. 51.) The general condition of the infant seems to be but little influenced, children strong and of good weight at birth being affected as readily as others. The **causes** are not clearly understood. That the fever is of so transitory a nature and without discoverable lesions largely eliminates such factors as general sepsis, pneumonia, and other grave inflammatory conditions; although Eröss believed that in many instances the elevation depends upon a slight temporary infection from local putrefactive changes in the umbilical cord, and in others upon a disturbance of the digestive tract. This explanation doubtless applies to a number of cases; but the rapid development of the rise of temperature and its rapid disappearance, as well as the uniform limitation to the same period of life, makes it unlikely that the majority of cases can be attributed to such causes. It has also been maintained that the fever depends upon irritation of the kidneys by uric acid present in them, but there is lacking sufficient evidence in support of this view. The fact that the fever is liable to develop at the time when the initial loss of weight is at its greatest is a suggestion that it may be associated with a condition of inanition from hunger (Holt)[3] or may depend upon thirst with consequent desiccation of the tissues (Müller).[4] Bakwin[5] has found that this transitory fever in new-born infants is associated with increase in the concentration of the serum-protein and that fall in temperature was coincident with an increase in the plasma-water. Those infants who lose most in weight are the ones oftenest showing elevation of temperature. It has been found, too, that the giving of breast-milk freely, or even of water, will often

[1] La Pediat., 1915, XXIII, 768.
[2] Jahrb. f. Kinderheilk., 1891, XXXII, 68.
[3] Arch. of Pediat., 1895, XII, 561.
[4] Berl. klin. Wochenschr., 1910, XLVII, 673.
[5] Amer. Jour. Dis. Child., 1922, XXIV, 508.

cause the fever to disappear promptly. There seems every reason to believe that these causes are operative in many instances; but against the view that they are the sole cause is the fact that in hunger-states after the period from the 2d to the 4th day, the starving infant with drying tissues is more liable to exhibit subnormal bodily temperature. The fact that the fever appears, as stated, only upon certain days of life, and that it does not attack with greater frequency those already feeble or of low temperature at birth, would support the suggestion which has been made that it may depend upon an autointoxication, the result of some of the physiological processes which are especially active at this time, and that the giving of water or breast-milk freely may act not directly by relieving hunger or thirst and consequent inanition, but by favoring a washing out of these toxic products from the system. This is supported by the opinions of Grulee and Bonar[1] and of Sherman and Lohnes,[2] who attribute the rise of temperature to the absorption of protein-products, bacterial or otherwise, from the intestinal tract. The addition of breast-milk to the diet reduces the putrefactive processes in the intestine and the temperature falls. Jaschke[3] considers it to be dependent on the absorption of pyogenic substances, and Plantenga[4] upon bacterial action. Finkelstein[5] attributed it to the influence of the sodium chloride of the food, and

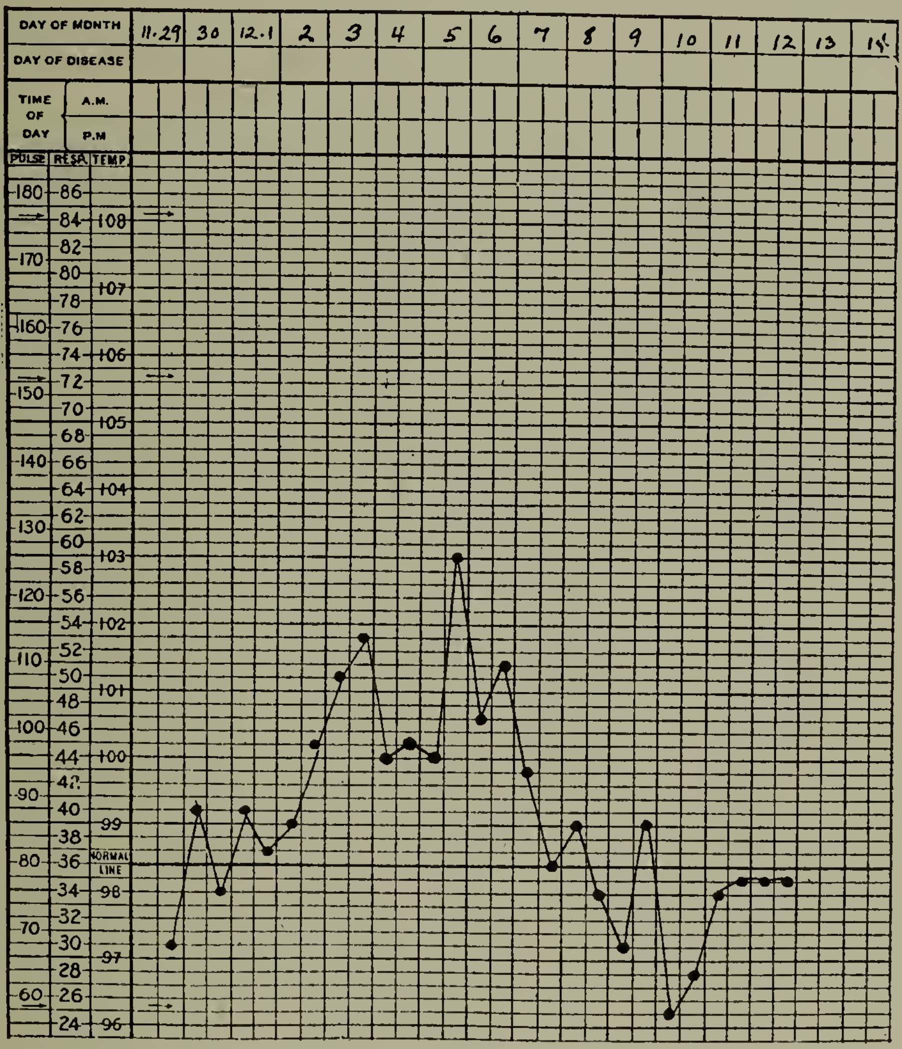

Fig. 51.—Transitory Fever in the New-born.

Baby M., born Nov. 29. Persistent fever and loss of weight. Weighing before and after each nursing showed an insufficient amount of milk furnished. After supplemental feeding was commenced Dec. 6 the temperature fell and gain in weight began.

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[1] Amer. Jour. Dis. Child., 1922, XXII, 44.

[2] Jour. Amer. Med. Assoc., 1921, LXXVII, 1720.

[3] Zeitschr. f. Geburtsh. u. Gynäk., 1916, LXXVIII, 119.

[4] Jahrb. f. Kinderheilk., 1917, LXXXVI, 175.

[5] Lehrb. der Sauglingskr. 2 Aufl., 1921, 252.

Hirsch and Moro[1] conclude that sugar is a prominent cause, but that more than one substance is capable of producing the fever. It is evident that, despite the numerous investigations, the etiology is yet far from clear, and that doubtless different causes are operative in different cases.

The **symptoms** attending transitory fever are more or less characteristic. The appetite is often diminished and the children nurse poorly from the breast or the bottle. In other cases they suck with avidity and an evidence of hunger. There is restlessness or in more severe cases prostration and little movement. Loss of weight continues while the fever lasts, and is usually greater than in normal infants. The temperature reaches 39°C. (102.2°F.) or often more. Its duration is 2 to 3 days, sometimes with intermissions, and the fall is generally rapid.

The **prognosis** is good in the majority of cases, except that the continued and increasing loss of weight may exert a serious influence upon the infant, and even predispose to a fatal termination of any other affection which may arise. The **diagnosis** is on this account very important. A careful examination will usually reveal the causes of fever of any other nature than that now under consideration, and such an examination must, of course, invariably be made. Septic inflammation, if other than purely local, is generally excluded by the disposition to develop at a slightly later period; although this is by no means an invariable rule. The fever also is not of such short duration and the infant seems more ill. The **treatment** in all cases dependent upon insufficient food or liquid is simple. In addition to the free supply of breast-milk or of water, other measures are entirely symptomatic.

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[1] Jahrb. f. Kinderheilk., 1917, LXXXVI, 34; 1918, LXXXVIII, 313.

SECTION II

THE INFECTIOUS DISEASES

CHAPTER I

GENERAL

DEFINITION

Much confusion has existed regarding the employment of the terms "infectious" and "contagious" as applied to disease. In the widest sense an "infectious" disease is one due to a specific living organism of some sort which "infects" the human body; while "contagious" indicates merely that the disease may be contracted by one individual by contact, directly or indirectly, with another. As a matter of fact most of the infectious diseases are contagious also. A notable exception to this is seen in the case of malaria. Certain diseases, such as some of those dependent upon the presence of various parasites in the gastroenteric tract, the contagious parasitic affections of the skin, and certain others, while clearly infectious in the broader sense are not ordinarily classified among the acute infectious disorders; that term, in the narrower sense, and for the sake of convenience, being reserved for those dependent upon a more general infection of the human organism by microscopic germs, in which the symptoms are of a more general nature. Those with a comparatively short and self-limited course are called the "acute infectious diseases;" or, if febrile, the "acute infectious fevers." The others are designated "chronic infectious diseases." Many of the first class are attended by special cutaneous eruptions, and to those the title "acute exanthemata" is often applied. The course of the infectious diseases is often marked by certain stages or periods designated by special terms. That during which the germs are developing in the body without characteristic manifestations is called the *period of incubation.* On this follows the *prodromal stage* or *period of invasion,* during which the *initial symptoms* appear. After this is the stage of the fully developed disease, or *stage of florition,* in the case of the exanthemata called the *period of eruption.* Last comes the *stage of decline,* in the eruptive fevers sometimes designated the *period of desquamation.*

Any accurate classification or even enumeration of the infectious disorders is impossible at this time of rapidly advancing knowledge regarding them. The list is constantly growing. Among them are included here: scarlatina; rubeola; rubella; variola; vaccinia; varicella; typhoid fever; erysipelas; cerebrospinal fever; diphtheria; grippe; pertussis; mumps; malaria; tetanus; epidemic encephalitis, poliomyelitis and a few less important ailments. The chronic infectious diseases include tuberculosis and syphilis.

Pneumonia appears to be quite certainly an infectious disorder, rheumatism and perhaps chorea probably so as well; but for the sake of convenience and for other reasons I have considered them elsewhere. So, too, certain intestinal infections, such as cholera and some forms of enterocolitis, are clearly infectious and communicable; yet they are more conveniently studied in the section of Diseases of the Intestine. The septic infections of the new-born have for the most part already been described in a preceding section, and gonorrhea is discussed under the headings of the organs affected.

At times 2 or even more acute infectious diseases may affect a patient simultaneously or one shortly after the other. The following of pertussis upon rubeola is of great frequency, and the development of diphtheria with or immediately after scarlet fever or especially measles is also often seen. Measles and scarlet fever may occasionally occur together, and even epidemics of this have been observed. The combination of typhoid fever with scarlet fever is less often reported, and that of typhoid fever with measles still less frequently. In one instance I saw rubeola and diphtheria with varicella which took on a gangrenous form occur nearly simultaneously, and in another typhoid fever, measles and cerebrospinal fever. Hoyne[1] reports a patient exhibiting diphtheria, varicella, measles, and scarlet fever within the course of 4 or 5 days. Many of these multiple infections are simply accidental; in others, such as the combination of scarlet fever and diphtheria, it may well be that the lowered local resistance occasioned by one disease predisposes to the other.

METHOD OF DISSEMINATION

The older views were long accepted without question, that the chief means of the dissemination of the germs of infectious diseases was by the air-currents, the dust of the room, the playthings, books and clothing of the patient, and the clothing of attendants or other third persons. More careful study of recent years has proven with fair satisfaction that in most of the diseases, although these methods are theoretically possible, in practice they occupy a very limited position. The transmission by a third person is practically only by the direct handling of a child, as by a nurse who has just left an infectious patient close by. This could take place only with great carelessness or in hospital wards. The chief source of infection is close contact with or proximity to the patient himself, the disease being communicated to others either before it has been recognized and the individual isolated, or given after quarantine has been removed and the patient believed to be well. It is usually by means of the mucous secretion of the infected person; often through the microscopic drops of mucus which are coughed or sneezed out by the patient or carried upon the breath; these reaching the respiratory tract of the previously healthy individual. In numerous instances the disorder has been perhaps so mild that it has not been recognized at all, and the child has remained an undiscovered source of infection. Those who have recovered from an infectious disease but who still harbor its active infectious germs are called "carriers," and the same title is applied to the smaller, but still quite large, class comprising those who have not suffered, but who have been in close contact with patients, and who carry the germs for a considerable time upon their mucous membranes. It is almost solely in this way that the virus is transmitted by a third healthy person, and the danger is probably less than from those who have suffered from the disease. The possibility of the dissemination by air, dust, water, milk, insects, and in other ways will be considered under the headings of the

[1] Arch. of Pediat., 1920, XXXVII, 606.

various infectious disorders. It varies with the disease; tuberculosis, for instance, being undoubtedly readily spread by infected dust upon the hands or in the food; typhoid fever by water and by insects.

GENERAL RULES FOR THE HYGIENIC CARE AND THE QUARANTINE OF INFECTIOUS DISEASES

The diseases under consideration vary greatly in their relative infectiousness, and in the mode by which the germs are chiefly conveyed. The following rules are those to be applied where rigorous prophylactic measures are to be carried out. Circumstances may alter either the possibility or the necessity of their application. It must be understood clearly that the methods to be described constitute in some respects the ultimate limit of precautions to be followed; not those which are considered by the leading authorities as actually necessary. It has, for instance, repeatedly been shown in hospital-practice that it is perfectly possible, with proper precaution by the aid of the "box method," to treat many of the infectious diseases in the general wards without extension taking place. (See Koplik,[1] von Pirquet,[2] Richardson,[3] Rundle and Burton,[4] Feer[5] and others on the Box-System and similar methods in hospital practice.) However, inasmuch as the requirements of local Boards of Health vary and are often rigorous, and since our knowledge of the method of spread of infectious diseases is still incomplete, it is better to err upon the safe side, and the full details of quarantine may well be described.

Room.—The room selected should be preferably in the upper portion of the house. Here the patient is less liable to be disturbed by noises, and here, too, isolation can be better carried out. It should be well-lighted and well-warmed, and capable of being well-ventilated. It should open into another room, which we may call the anteroom. This should be, if possible, a bath-room or a room opening into it. The temperature of the sick-room should be from 65 to 70°F. (18.3° to 21.1°C.). The door entering it from the hallway should be locked, and the cracks stuffed with paper or cotton. The door from the anteroom into the hallway should be kept closed except at the time of exit or entry. A sheet should be hung here which should be kept moistened with a solution of corrosive sublimate (1:1000) or one of carbolic acid (5 per cent.) or its equivalent in other antiseptics. The real value of this measure against the spread of the disease is more than questionable, but it serves at least as a reminder, and satisfies the demands of some Boards of Health.

Ventilation is best obtained by a fireplace, or, in the absence of this, from the anteroom. Screens should be used at the windows of the sick-room to prevent the direct action of draughts upon the child and the entrance of insects. If the weather permits, the window of the anteroom should be opened constantly, thus practically cutting off the sick-room from the rest of the house. It is to be understood that the action of draughts in producing chilling of the surface and any consequent damage is not so much to be feared during the febrile state of the disease. It is after fever is past that injury from this may follow.

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[1] Arch. of Pediat., 1911, XXVIII, 728; 1912, XXIX, 5.
[2] Zeitschr. f. Kinderheilk., Orig., 1913, V, 213.
[3] Jour. Amer. Med. Assoc., 1913, LXI, 1882.
[4] Lancet, 1912, I, 720.
[5] Correspondbl. f. Schweiz. Aerzte, 1919, XLIX, 777.

The sick-room should have all unnecessary articles removed, especially all clothing, carpets, curtains, upholstered furniture, and pictures. Only such books and toys may remain as shall afterward be destroyed. The room should be kept very clean, all dust being removed by wiping with a 1:1000 bichloride or other antiseptic solution. The bichloride solution must not be used upon metal.

Patient.—The food for the patient and nurse should be brought to the door of the anteroom by an attendant. After the dishes, spoons or other articles have been used, they should be washed, and either boiled, or disinfected with a 5 per cent. solution of carbolic acid in which they should lie for some time. All bed-clothes and body-clothing should be changed frequently and should be immersed in a 5 per cent. solution of carbolic acid or a 1:1000 solution of corrosive sublimate kept in the anteroom. After thorough soaking they should be wrung out, placed in a bucket and taken by an attendant from the nurse at the door of the anteroom. They may now be washed with the other linen of the house without danger. Old linen or muslin cloths and absorbent cotton may well replace handkerchiefs, since they can be destroyed at once after use. The utmost care should be taken to receive and disinfect or destroy promptly all discharges from the eyes, nose, mouth and, in the case of typhoid fever, the intestine and bladder. When there is free expectoration, paper sputum-cups or others holding a 5 per cent. solution of carbolic acid or similar disinfectant may be employed.

Attendants.—The nurse should be dressed in washable material, and should have a cap to cover thoroughly the hair and the neck. She should have her meals in the sick-room or, better, anteroom, and leave it only when about to pass through the house on her way out. Before doing this she should slip off her outer garment, disinfect her hands and face with a 2 per cent. solution of carbolic acid or a 1:5000 of corrosive sublimate, put on her outer street clothes, which should be kept in the anteroom, and go directly out without stopping to talk with members of the family. Those of the family who must necessarily enter the room to relieve the nurse should adopt similar precautions. With proper care the confinement of the nurse to the sick-room during the whole of the disease is entirely unnecessary and constitutes a superfluous hardship. The physician, too, should on entering the anteroom array himself in a linen or rubber garment. A linen dust-coat is very serviceable for this purpose. He should wear an oil-silk or other cap upon his head. Before leaving the anteroom he should remove these articles, and disinfect his hands and face carefully. Regarding the wearing by the attendants of face-masks of gauze, unless these are so thick and firmly applied that respiration would be decidedly interfered with, it is questionable whether they are sufficiently effective to render their employment worth while (Kellogg and MacMillan).[1]

Family.—Other non-immune children should, when possible, be removed from the house, and should not be allowed to go to school or mingle with others until a time has elapsed equal to the duration of incubation, for fear they may have the disease already in their systems. If they are obliged to continue to reside in the house they should not attend school or associate with other children until the patient is removed from quarantine. In case, however, they have already had the disease, there is no actual reason why they may not safely be with other children if the measures for isolation of the patient are being properly carried out. The feeling of other parents regarding the matter is, however, so strong that it is useless to insist upon the truth of this.

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[1] Amer. Jour. Publ. Health, 1920, X, 34.

Final Disinfection.—When the disease is over the child should receive a disinfecting bath of corrosive sublimate, 1:10,000, particular attention being paid to the hair. (See p. 271.) He should then be dressed in entirely clean underclothing and removed to another room. The outer clothing in use at the time the disease began should have been meanwhile thoroughly exposed to fresh air and sunlight. The sick-room and all others used during the illness should be thoroughly disinfected. The carpets, if they have not been removed previously, and the mattresses and pillows should be subjected to superheated steam in a municipal disinfecting plant, when this is practicable. When not, they may be allowed to stay in the room during fumigation, and then exposed to the open air and sunlight for several days. The walls, if papered, should be scraped, and, if painted, washed with a 1:1000 solution of corrosive sublimate. The floor should be scrubbed with soap, and then washed with the solution. The furniture should be wiped carefully with the solution, excepting in the case of metal, when carbolic acid, 5 per cent., must be used instead. All small objects, such as toys, books and the like should be burned. Fumigation with formaldehyde has been much in vogue, but is being with reason abandoned in many quarters. As ordinarily performed it is questionable whether it possesses any value. It may be regarded as an additional precaution. Preparatory to the fumigation the cracks of the windows should be plugged with cotton, a formaldehyde lamp or candle started or the solution scattered about, the outer door then closed and the cracks plugged in the same way, and the room left undisturbed for 12 hours.

CHAPTER II

SCARLET FEVER

(Scarlatina)

History.—Although by some believed to have been known to the ancients, or at least to early Christian or Arabian writers, it is more probable that scarlet fever was not distinguished from other eruptive fevers until about the middle of the 17th century. At this time Sennert[1] gave a good clinical description of an epidemic. Sydenham[2] appears to have been the first to distinguish it clearly from measles, and to apply to it its present name. The disease has certainly increased greatly in frequency since the 17th century, and now is one of the most common of the infectious fevers, without any discoverable diminution in its incidence (Donnally).[3]

Etiology. Predisposing Causes.—Climatic and geographical conditions seem to possess but little influence. The disease appears capable of developing wherever the infection is brought, although it still remains far most common in Europe and America. The season of the year is perhaps not without importance, the greatest number of cases occurring in the winter and especially in the autumn months. This is, however, a much disputed point, and the influence of season, if it exists, is certainly not very considerable.

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[1] Med. pract. Wittemberg, 1654, II, cap. 12. Ref. Rilliet and Barthez (Sanné) Mal. des enf., 1891, III, 74.

[2] Processus Integri. Ref. Williams, 20th Cent. Pract. of Med., 1898, XIV, 117.

[3] Amer. Jour. Dis. Child., 1916, XII, 205.

Sex, race, social conditions, and sanitary surroundings possess no certain predisposing causal relationship, the statements regarding them being entirely contradictory. So, too, the previous state of health of the patient has no discoverable connection with the susceptibility, except that children with the exudative diathesis are thought to be especially predisposed and that patients with wounds are particularly likely to contract the disease. (See Surgical Scarlet Fever, p. 355.)

On the other hand, the influence of *age* is very great. Although scarlet fever may possibly occur congenitally (Ugon;[1] Liddell and Tangye;[2] Comolmi[3]), or may develop in old age, by far the most frequent occurrence is in childhood. The greatest number of cases were found by Gresswell[4] between the ages of 5 and 10 years, and the next between 2 and 5 years. Donnally[5] in a review based upon some millions of cases placed about 50 per cent. between 3 and 8 years, and 90 per cent. in the first 10 years of life. Under the age of 1 year, and especially under 6 months it is very much less common than at other periods of early life. Gresswell gives only 0.5 per cent. in the 1st year in 568 cases; Caiger and Dudgeon[6] 0.3 per cent. in 157,149 cases of the Metropolitan Asylums Board's Hospitals, and Heiberg[7] 0.4 per cent. in 1000 cases. The youngest patient under my own observation was 3 months of age. Lemarquand[8] records 22 personal observations, and gives a number of others from medical literature, in which nursing women with scarlet fever did not communicate the disease to their infants in the early months of life. We have seen 2 nursing infants under 6 months of age admitted to a scarlet fever ward yet who escaped the disease entirely. In fact, the danger at this period is so slight that there is no need for weaning, so far as the infant is concerned. Dorner,[9] however, while admitting the ordinary lack of susceptibility, reports an instance of the contraction from the mother of the disease in a mild form by a nursing infant of 7 days.

The *individual susceptibility* to the disease varies greatly. Some individuals appear immune, although repeatedly exposed. According to the studies of Stickler,[10] Kobrak[11] and others, the incidence, severity and mortality of scarlet fever are less in poorly nourished children than in well-fed ones with the exudative diathesis. The disease certainly has a less general tendency to spread than some of the other infectious disorders possess. In the experience in the Faroe Islands (Hoff),[12] for instance, while 99 per cent. of the population of one of the chief towns showed susceptibility to measles, only 38.3 per cent. of unprotected persons contracted scarlatina. In Boston during 5 years the number of cases varied from 16.77 to 62.87 per 10,000 of the population (McCollom).[1] My own experience has always shown the spread of scarlatina to be much more readily controllable than that of measles.

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[1] Jahrb. f. Kinderheilk., 1910, LXXII, 676.

[2] Brit. Med. Jour., 1923, II, 380.

[3] La Pediat., 1922, XXX, 491.

[4] A Contrib. to the Nat. Hist. of Scarlatina, 1890.

[5] Loc. cit.

[6] Allbutt and Rolleston's Syst. of Med., 1906, II, I, 429.

[7] Zeitschr. f. Hyg. u. Infektionskrankh., 1910, LXV, 237.

[8] Thèse de Paris, 1908, July.

[9] Deutsch. med. Wochenschr., 1920, XLVI, 734.

[10] Arch. f. Kinderheilk., 1922, LXVII, 15.

[11] Zeitschr. f. Kinderheilk. Orig., 1920, XXVI, 137.

[12] [illegible] Beobachtung 1874. Ref. von Jürgensen in Nothnagel's Encyclopedia, Amer. Edit., Article Measles 228; Scarlet fever 382.

The comparatively small number of cases occurring in early infancy and after childhood is passed is probably in part due, in the first instance, to a lesser frequency of exposure, and perhaps to the transmission of protective bodies from the mother; and, in the second, to the protection already given by a previous attack. Still, a greater degree of susceptibility certainly does appear to exist in childhood, since in the Faroe's epidemic, where no previous immunity existed, 56.3 per cent. of the cases were under 20 years of age and only 7.6 over 40 years. This immunity of older persons may be due to the protection which could be afforded by repeated exposure to the causative organism, this exposure being insufficient actually to produce active symptoms but enough to initiate antibody-formation.

An *epidemic influence* is also discoverable. This is especially seen in country districts, for in many of the larger cities the disease is practically endemic, cases constantly appearing although in very different numbers at different seasons and in different years. The severity of the disease and the consequent mortality vary greatly with the epidemic.

Exciting Cause.—The direct cause of the disease is infection, and analogy is convincing that this is by means of some form of living organism. Meyer[2] and others have regarded it as an anaphylactic reaction and Szontagh[3] as an auto-infection dependent upon the pharyngeal inflammation; but these views have been adopted by few investigators. The *nature of the germ* has been much discussed, but seems to be approaching some definite recognition. From the time of the report by Klein[4] of the streptococcus scarlatinæ to the present, various germs have been described. Among the different investigators Class[5] discovered a diplococcus which he believed to be pathogenic, and Baginsky and Sommerfeld[6] reported a similar germ as being uniformly present in the pharynx and as found in the blood and organs of fatal cases. Salge[7] and others have produced agglutination of the scarlatinal streptococci by the blood from a case of scarlet fever, but Kolmer[8] found agglutinins and antibodies present in only a small percentage of cases. Mallory[9] reported a protozoan-like body, the cyclastis scarlatinalis, resembling the malarial parasite and found in and between the epithelial cells of the skin and in the lymph-vessels and spaces of the corium; and in a later publication with Medlar[10] described a Gram-positive bacillus constantly present early in the case in the crypts of the tonsils. Later this disappears and is replaced by streptococci. Pryer[11] writes of a large coccus-shaped organism isolated from scarlet fever patients, which he thinks may be the cause of the disease. A great deal of work has been done by di Cristina[1] and other Italian investigators (Vitetti;[2] Caronia and Sindoni[3]) with a small anaerobic diplococcus which has been obtained from the naso-pharyngeal secretion, blood, bone-marrow, spleen and the epidermal scales of patients suffering from scarlet fever. It is claimed that this organism, when inoculated into rabbits, produces a disease resembling scarlet fever. Agglutination and complement-fixation tests are specific, and in a few experiments immunity seems to have been conferred upon patients inoculated with killed cultures of this diplococcus. These results are at variance with those obtained by others indicating a relationship to the special hemolytic streptococcus presently to be discussed, and only further study can decide the matter.

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[1] Boston City Hospital Reports, 1899.

[2] Deutsch. med. Wochenschr., 1923, XLIX, 509.

[3] Arch. f. Kinderheilk., 1910, LIV, 111; Jahrb. f. Kinderheilk, 1912, LXXVI, Ergänzungsh., 1.

[4] Proc. Royal Soc., London, 1887, XLII, 158.

[5] New York Med. Record, 1899, LVI, 330.

[6] Berl. klin. Wochenschr., 1900, XXXVII, 588.

[7] Münch. med. Wochenschr., 1902, Oct. 14, 1729.

[8] Arch. Int. Med., 1912, IX, 220.

[9] Jour. Med. Research, 1904, X, 483.

[10] Jour. Med. Research, 1916, XXXIV, 127.

[11] Jour. Lab. and Clin. Med., 1922, VII, 592.

The chief point of interest is connected with the possible causative influence of the streptococcus. For a long time it had been known that streptococci can be obtained from the throats, blood, lymphatic glands, and the complicating lesions of scarlet fever patients. Successful transmission to animals by inoculation had been reported by Landsteiner, Levaditi and Prasek;[4] Bernhardt;[5] and Schleissner,[6] but this had not been confirmed by the studies of Draper and Hanford,[7] Krumwiede, Nicoll and Pratt,[8] Klimenko[9] and others. It had usually been considered, as pointed out by Hektoen,[10] Jochmann[11] and others, that, although a germ of this class is frequently present, it is rather the cause of the complicating conditions than of the disease itself. Later, however, the development by Dochez, Avery and Lancefield[12] of a method for the differentiation of biologic types of streptococcus hemolyticus has led to more exact knowledge. Work by Tunnicliff,[13] Gordon,[14] Bliss,[15] Dochez and Sherman,[16] Herrold and Tunnicliff,[17] Dick and Dick and others has demonstrated quite clearly that there is a specific type of streptococcus hemolyticus associated with scarlet fever. By inoculation of guinea pigs with this organism a disease somewhat resembling scarlet fever can be produced (Dochez and Sherman),[18] and experimental scarlet fever has been developed with it in man (Dick and Dick).[19] By immunization of the horse an antiserum has been obtained which possesses marked curative value (Dochez and Sherman;[20] Blake, Trask and Lynch[21]). Dick and Dick[22] have also been able to produce with a filtrate of the culture a positive cutaneous test in persons who have never had scarlet fever. (See p. 375.) All of this convincing study would seem to indicate that a specific type of hemolytic streptococcus is the cause of scarlet fever.

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[1] La Pediat., 1921, XXIX, 1105; 1923, XXXI, 1.

[2] La Pediat., 1923, XXXI, 989.

[3] La Pediat., 1923, XXXI, 745.

[4] Ann. de. l'instit. Pasteur, 1911, XXV, 754.

[5] Deutsch. med. Wochenschr., 1911, XXXVII, 791.

[6] Jahrb. f. Kinderheilk., 1915, LXXXII, 225.

[7] Jour. Exper. Med., 1913, XVII, 517.

[8] Arch. Int. Med., 1914, XIII, 909.

[9] Jahrb. f. Kinderheilk., 1913, LXXVII, 679.

[10] Jour. Amer. Med. Assoc., 1903, XL, 685; 1907, XLVIII, 1158; 1923, LXXX, 84

[11] Deutsch. Arch. f. klin. Med., 1903, LXXVIII, 209.

[12] Jour. Exper. Med., 1919, XXX, 179.

[13] Jour. Amer. Med. Assoc., 1920, LXXIV, 1386.

[14] Brit. Med. Jour., 1921, I, 632.

[15] Jour. Exper. Med., 1922, XXXVI, 575.

[16] Proc. Soc. Exper. Biol. and Med., 1924, XXI, 184; Jour. Amer. Med. Assoc., 1924, LXXXII, 542.

[17] Jour. Infect. Dis., 1924, XXXIV, 209.

[18] *Loc. cit.*

[19] Jour. Amer. Med. Assoc., 1923, LXXXI, 1166; 1924, LXXXII, 301

[20] *Loc. cit.*

[21] Jour. Amer. Med. Assoc., 1924, LXXXII, 712.

[22] Jour. Amer. Med. Assoc., 1924, LXXXII, 265.

Method of Transmission.—The disease never originates spontaneously, but is always transmitted from the sick to the well. The transmission is either direct, and this is true of the great majority of cases, or mediately through clothing, carpets, bedding, and the like, or a third person. The mediate infection is certainly uncommon. (See Infectious Diseases, p. 344.) The transmission occurs with great frequency in schools, where children with the disease about to manifest itself, and who consequently possess the power of infecting, communicate it directly to others.

Numerous instances are on record where the disease has been carried even for sometime and distance in the clothing of physicians or nurses, or been transmitted by letters, toys and books, or carried by animals. These methods of dissemination are, however, very exceptional. There are many instances of its spread by milk coming from dairies where scarlet fever existed among the families of the employees. Kober[1] found the disease at the dairy or milk-farm in 68 out of 99 reported epidemics of scarlet fever. Although instances are reported, it is yet doubtful whether an immune inmate of a house where scarlet fever exists, who does not come into intimate association with the patient, can transmit it to another individual. Similarly it is uncertain whether there are carriers in the ordinary sense. Indeed, close and recent contact with the patient or with the infected article seems to be necessary, as the germs do not appear to be carried to any distance by the air. The disease, as stated, has been communicated by direct inoculation (Strickler;[2] Dick and Dick [3]).

Portal of Entry.—That the throat is so early and prominently affected indicates that the germs enter by way of the naso-pharyngeal mucous membrane. It is likely, however, that in case of an open wound they may find entrance in this situation (see Surgical Scarlet Fever, p. 355). In fact, the usual entry by the throat may be the result of the existence of an irritated or broken mucous membrane in that situation.

Seat of the Virus.—The parts of the body in which the infectious matter is chiefly situated is a subject still incompletely understood. The fresh or dried mucus from the nose and throat is undoubtedly infectious, as is the pus from a complicating otitis, empyema or suppurating adenitis. The infectiousness of the urine and feces is in doubt. There is no proof of its existence. It is questionable whether the expired air carries the germs. The scales of the epidermis were formerly considered extremely infectious, but this is now generally denied, and there is little if any danger from them except as they may be merely the holders of contagious material derived from the secretions, which has attached itself to them.[4] It is certain that the disease can be given by persons suffering from a scarlatinal angina who have never exhibited eruption or desquamation.

Period of Maximum Infectivity.—With this uncertainty as to the chief method of dissemination of the contagion, the question regarding the most infectious stage must remain as yet undetermined. The ease with which infection spreads in schools shows that the latter portion of the stage of incubation or certainly the earliest part of the stage of invasion must posses infectious power. Probably the most active period is that of the height of the eruption. The disease is also communicable during the stage of desquamation and after it. This is proved by the frequency with which it occurs in other children in the family, after patients who have suffered from scarlet fever in hospitals have returned to their homes ("Return cases"). Thus Pugh[1] found that 2.9 per cent. out of 6507 hospital cases later communicated the disease to others at home after 6 weeks in the hospital. In many of these instances the virus had probably persisted in the nasal or faucial secretion of a mucous membrane still slightly diseased, or in the pus of a discharging ear.

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[1] Amer. Jour. Med. Sci., 1901, 552.

[2] N. Y. Med. Record, 1899, LVI, 363.

[3] *Loc. cit.*

[4] Discussion, Brit. Med. Jour., 1902, 1, 777.

Tenacity of Life.—The remarkable tenacity of life which it has been believed that the germs possess renders difficult the solution of the question of the time of greatest infectiousness. There are numerous carefully observed instances on record which seemed to prove that the germs may live and communicate the disease after months or even after several years. Murchison[2] gives an instance of the persistence of vitality for 4 months; Lommel[3] for 133 days; and Sanné[4] for 71 days. Others of still longer duration are on record but are open to doubt. Indeed, in view of later experience with the disease, any extreme tenacity of life possessed by the germs seems doubtful.

Pathological Anatomy.—There are no definite lesions characteristic of the disease except that of the skin and of the mucous membrane of the mouth and throat. The intense hyperemia present during life disappears after death. There is found, however, a dilatation of the lymphatics and blood-vessels of the *skin* (Pearce),[5] with a swelling of the cells of the rete, sometimes with extravasations of blood between them. An infiltration of all the layers of the skin by serum and leucocytes occurs, especially in the corium and around the hair-follicles, sweat-glands and blood-vessels. As a result of this process there is a rapid destruction of the upper cells of the epidermis which results in desquamation.

The *mucous membrane* of the mouth and throat suffers an analogous inflammation with desquamation, especially well seen on the tongue and producing, in typical cases, the strawberry appearance. The inflammation, in average cases merely catarrhal, becomes in many instances pseudomembranous or even gangrenous in the fauces. The pseudomembranous inflammation of the throat exhibits various organisms, generally streptococci. The faucial inflammation may spread to the nose and ears, and thence to the mastoid cells and even the meninges.

There is wide-spread involvement of the *lymphatic tissue* throughout the body. This is especially marked in the cervical lymphatic glands, which often suppurate, the pus containing streptococci. Swelling and edematous infiltration of the cellular tissue of the neck is common. The spleen is often enlarged and shows hyperplasia of the lymph-follicles. The lymphatic tissue of the gastroenteric tract is hyperplastic, Peyer's patches being often much enlarged and prominent. The liver may be enlarged.

Endocarditis and pericarditis are not uncommon, and pleuritis and pneumonia may occur. Parenchymatous changes may be found in any of the organs of the body. These are not characteristic, but are due partly to the fever, as in all febrile diseases, partly to the septic involvement, and partly to the direct action of the scarlatinal poison. All these lesions, as well as the involvement of the kidneys so often present, are conveniently considered under Complications.

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[1] Lancet, 1905, 1, 273.

[2] Lancet, 1864, II, 176.

[3] Münch med. Wochenschr., 1901, XLVIII, 1165.

[4] Rilliet and Barthez, Mal. des enf., 1891, III, 164.

[5] Boston City Hosp. Reports, 1899.

Symptoms. TYPICAL SCARLET FEVER. **Period of Incubation.**—This period, between infection and the development of the first symptoms, is irregular within certain limits. In general it may be placed at from 1 day to 1 week, in by far the majority of cases from 2 to 4 days. Even periods of but a few hours have been recorded (Russeger).[1] On the other hand, periods of incubation lasting 12 days are not hncommon, and even as much as 20 days or more are on record (Hagenback-Burckhardt).[2] As a rule the danger of developing the disease may be considered over after 7 days from exposure has elapsed. In cases of surgical scarlet fever, as for instance after tracheotomy or tonsillectomy, the period of incubation is usually very short. Severe cases also appear to have a shorter period of incubation than others.

Period of Invasion.—The onset is generally of great suddenness. Except in older children there is usually no distinct initial chill, although there may be chilliness and pallor. Convulsions are often among the first symptoms in quite young children. Vomiting, often repeated, occurs at the onset in the great majority of cases. Diarrhea occasionally accompanies it. The temperature rapidly rises to 103° or 104°F. (39.4° or 40°C.) or more, and the face becomes flushed. The pulse is rapid and there is headache and generally sore throat, of which, however, young children often make no complaint. The child looks and feels ill, sleeps badly, and is restless and decidedly prostrated. The lymphatic glands of the neck begin to enlarge. The eruption is now found in the throat (enanthem), giving a characteristic appearance; the mucous membrane, namely, of the hard and soft palate, the tonsils, and the anterior pillars of the fauces being unusually red, due to the presence here of closely packed, minute, dark-red macules. This condition rapidly spreads to the lining of the cheeks and gums. The macules may be distinctly visible or they may have fused to such an extent that only a uniform red flush is perceptible. The tongue is coated with the edges sometimes reddened. The tonsils are swollen and often exhibit whitish spots due to secretion retained in the follicles.

The duration of the invasion is short, from 12 hours or occasionally less up to 24 hours. It is of very common occurrence for a child to exhibit the febrile symptoms and vomiting during the night and to be found with the cutaneous eruption well-developed by morning. A period of invasion lasting 36 or 48 hours and even longer is exceptionally observed (Fig. 52). In very mild cases there may be no prodromes whatever or none discovered.

Period of Eruption.—*The Rash.*—The termination of the period of invasion is marked by the appearance of the rash on the skin. This develops first on the neck and upper portion of the chest; thence spreading rapidly to the rest of the trunk, the arms, and finally the lower extremities. Generally the face is little or not at all involved, the forehead and especially the circle about the mouth standing out prominently of a pale-white color in contrast with the rest of the body; the cheeks being flushed, but not the seat of a truly punctate eruption. In exceptional cases, however, there is an extensive development of eruption on the face, but even then the pale oral circle is usually to be found. Viewed superficially the rash seems to be of a uniformly red color, but careful inspection shows it to consist of very minute, closely packed red points situated on a white base. Later the red hue of the punctæ grows duskier while the white base takes on a shade of red as well. Though now more confluent the punctate character can still always be discovered. The eruption generally has spread to its full extent in 24 hours and often less. Sometimes, however, 2 or 3 days are required for this. Its greatest intensity of color may also be reached within 24 hours, but is not generally attained for 2 or 3 days. Where the integument is softest and finest the eruption is generally especially intense and most confluent. The skin is now hot, dry and somewhat swollen, the swelling being frequently particularly marked on the hands and feet. It often feels slightly rough to the hand rubbed over it. Decided itching is common. Drawing the fingernail over the red skin leaves for a moment a strikingly white line. This though very suggestive is not absolutely characteristic.

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[1] Med. Jahrb. d. k. k. Österr. Staates, 1848, LXIII, 277.

[2] Jahrb. f. Kinderheilk., 1886, XXIV, 105.

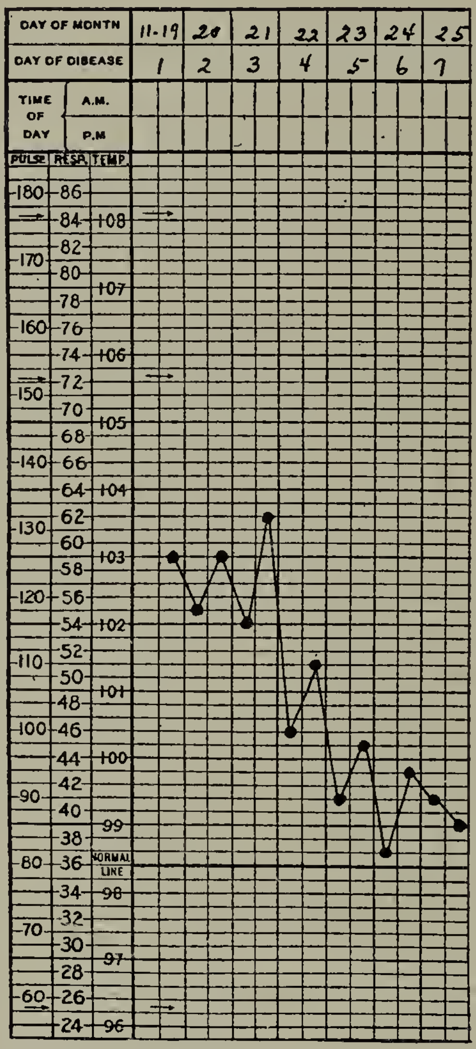

Fig. 52.

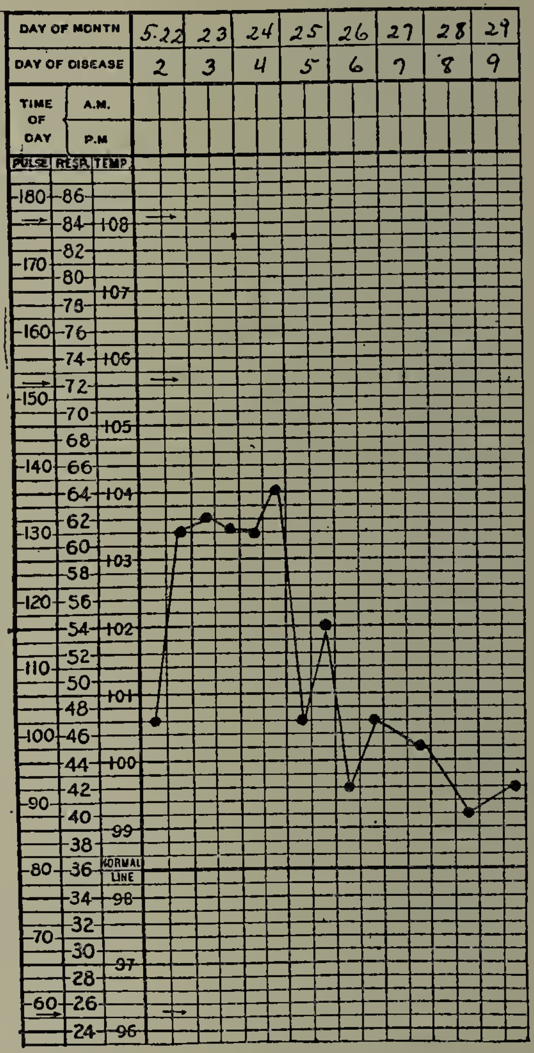

Fig. 53.

Fig. 52.—Scarlatina. Average Case Except for Prolonged Invasion, with Rash on the 3d Day.

Helen S., aged 2 years. Nov. 19, vomited, fever; Nov. 20, no complaint, throat very bright-red, suspiciously like scarlatina; Nov. 21, rash appearing, vomited; Nov. 22, general condition excellent, rash typical, abundant, throat less red; Nov. 23, rash fading, tongue red at tip, rash nearly gone.

Fig. 53.—Scarlatina. Average Case.

Andreas L., aged 8½ years. May 21, nausea in evening, fever, sore throat; May 22, vomiting, diarrhea, secretion in tonsillar crypts, eruption on body; May 23, sore throat, restlessness; May 24, eruption very abundant; May 25, eruption duskier, tongue entirely denuded; May 27, eruption fading, throat and tongue sore; May 29, sore throat no longer complained of.

The rash maintains its intensity for from 1 to 3 days and then fades rapidly, following the order of its appearance. The total duration of the eruption is extremely variable. An average may be said to be from 3 to 7 days. A faint underlying yellowish tinge is often discovered when the skin is pressed upon or as the eruption fades.

Normal Variations of the Eruption.—A number of variations occur in the eruption within the limits of the typical case. The color varies through many shades of red, from a pale rose to that of a boiled lobster, or to a deep red with a slightly brownish or bluish tint. When the eruption is not very extensive it may appear in smaller or larger blotches in some part of the body leaving other parts, especially areas on the extremities, entirely free (*Scarlatina variegata*). Sometimes it is macular in some portions and here strikingly suggests measles (Fig. 54). In many cases the skin is unusually rough, due to the presence of much infiltration in the minute red punctæ (*Scarlatina papulosa*), while in a large number there is a greater or less development of miliary vesicles (*Scarlatina miliaris*). These vesicles may occur in any part of the body. I[1] have seen them so abundant that the scarlatinal color of the underlying skin was almost totally concealed and the diagnosis made difficult. As a rule, however, they are few in number. Their occurrence does not appear to me to bear any relationship to the intensity of the ordinary scarlatinal rash, the amount of desquamation, or the severity of the case. In rare cases the vesicles may coalesce to a considerable extent, forming small blebs (*Scarlatina pemphigoides*).

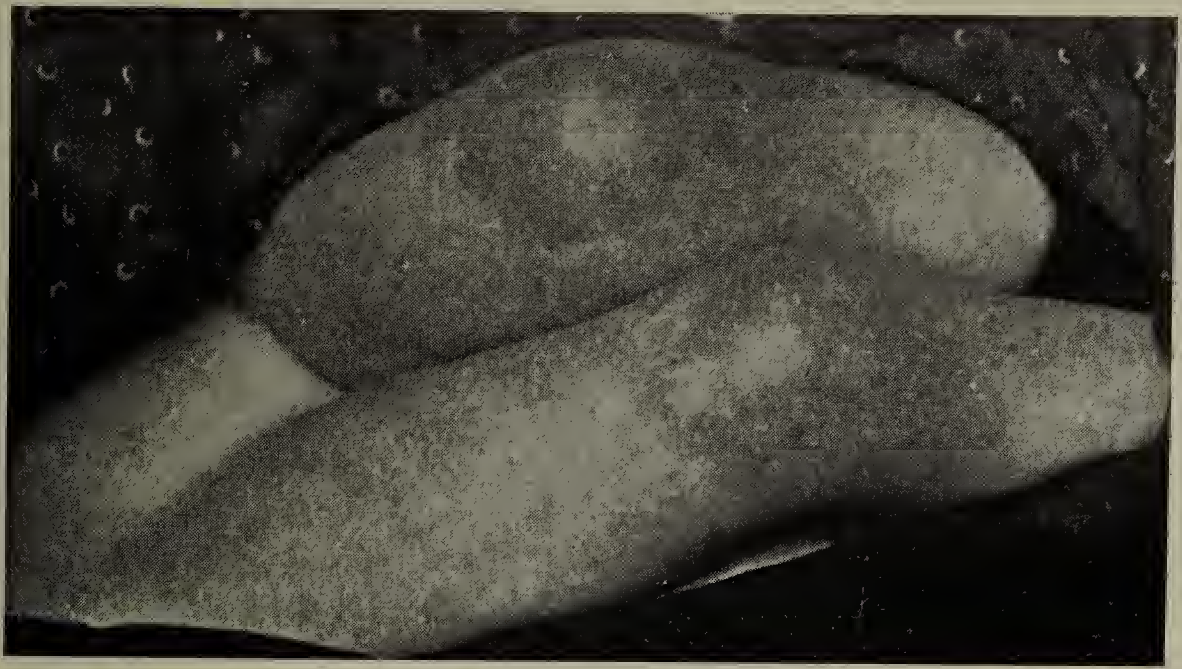

Fig. 54.—Scarlet Fever.
Blotchy rash suggesting measles. (*From a photograph.*)

In the so-called *Surgical Scarlet Fever* the rash develops sometimes first in the neighborhood of a wound, including lesions of the puerperium, and thence spreads over the body; or very soon after the receiving of the wound the rash may appear first on the neck or chest in the ordinary way, and follow the usual sequence in its spread; it being supposed that the infection gained entrance through the wound. The symptoms do not differ materially from those of the disease as usually seen. There has been much discussion as to the reality of surgical scarlet fever. There is reason to believe that it does exist, but that the large majority of the cases supposed to be of this nature are probably either instances of

[1] Jacobi's Festschrift, 1900, 182.

septic erythema or of mere coincidental development of scarlet fever in a surgical patient.

Retrocession of the Eruption.—It happens often that the rash soon after its appearance suddenly fades, or, as it is commonly called, has "struck in," the other symptoms persisting or growing worse. This disappearance is often the result of a feeble action of the heart. It is not the cause of unfavorable symptoms, but the accompaniment of them.

Other Symptoms of the Stage of Eruption.—The eruption of the mucous membrane of the *mouth and throat* (enanthem) is reaching its height when that of the skin begins to appear. The intense redness of the mucous membrane persists, and the tonsils, if not already affected, generally exhibit lacunæ filled with secretion, while swallowing is painful. The tongue begins to lose its white coating, and by the 3d to the 5th day of the disease has become entirely denuded, bright-red and with the fungiform papillæ swollen and prominent. The result is the "strawberry" or "raspberry" tongue. These titles have often been misapplied to the condition seen during invasion and in many other diseases, where the red papillæ, especially on the edges and tip, show prominently through the white coating. In many cases the tongue never becomes denuded throughout, yet the enlargement of the papillæ, at least of its tip and edges, is claimed by McCollom[1] to be invariably present and to constitute a valuable diagnostic sign in cases with doubtful or absent eruption. In typical cases the throat improves as the cutaneous rash and the other symptoms abate. By the 7th or 8th day of the disease the normal appearance of the tongue is nearly restored. Close examination, however, will show a persistence of some degree of redness and swelling of the papillæ at the edges and tip of the organ after other symptoms of the stage of eruption have entirely disappeared. The nose often exhibits a mucopurulent discharge. The lymphatic glands of the superfices of the body are found swollen, those of the neck, groins and axillæ and those below the body of the jaw, being most noticeably so. Some degree of tenderness on pressure is present. The spleen is often palpable. The *temperature* (Fig. 53) is subject to great variations, and no typical curve exists. As a rule it reaches its height from the 2d to the 3d day and continues at 102° to 104°F. (38.9° to 40°C.) with little variation between morning and evening, its elevation being generally in proportion to the severity of the attack. It begins to diminish as the eruption fades, falling by lysis, and reaching normal about the 9th or 10th day. Very often the elevation of temperature lasts a much shorter time than this. The pulse is rapid, often out of proportion to the elevation of temperature. This is generally considered one of the characteristics of the disease. The blood-pressure is sometimes reduced (Rolleston)[2] but its condition possesses little practical significance unless nephritis occurs. The bowels are usually not disturbed during this stage. There is thirst and loss of appetite. Vomiting is not common during the eruptive stage. Slight delirium may develop during the height of the fever. Either somnolence or restlessness may be present.

The *urine* is high-colored and diminished in amount and, if the temperature is high, may exhibit febrile albuminuria with cylindroids and possibly a few hyaline casts; but blood-cells and granular casts do not occur unless a complicating nephritis develops. Woody and Kolmer[3] found the diazo-reaction present in 8.53 per cent. of 375 cases examined. Urobilin is found in the majority of instances (Rach and Reuss;[1] Gromski[2]).

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[1] Boston City Hospital Reports, 1899.

[2] Brit. Jour. Child. Dis., 1912, IX, 444

[3] Arch. of Pediat., 1912, XXIX, 12.

The *blood* shows a quite decided diminution in the number of red blood-cells and in hemoglobin; the latter occurring more promptly and with a greater rapidity (Schiff),[3] and the diminution not being in proportion to the intensity of the eruption. The original percentages are restored after several weeks. According to Tileston and Locke[4] a hyperleucocytosis begins from the 2d to the 8th day reaching from 18,000 to 40,000 per c.mm. This falls gradually and reaches normal at the end of from 3 to 6 weeks. The polymorphonuclear cells are increased absolutely and relatively during the stages of invasion and eruption, reaching 85 to 90 per cent., and decrease to normal with the disappearance of the leucocytosis. In our own experience the relative polymorphonuclear increase is more characteristic than the total increase in white blood-cells. The mononuclear cells are at first diminished, sometimes to even 4 or 5 per cent. The eosinophiles nearly or quite disappear, but rise much above normal with defervescence and continue so into convalescence. The number may even exceed 20 per cent. These observations confirm for the most part the earlier studies of Kotschetkow,[5] Sacquépée,[6] Bowie,[7] and others. Iodophilia is reported by Neutra[8] and Magi.[9] There is an increase of blood-platelets above normal, occurring after the disappearance of the eruption (Schiff and Mátyás).[10] Great interest in recent years has attached to the discovery by Döhle[11] of certain "inclusion bodies" constantly found in the polymorphonuclear leucocytes of the blood in scarlet fever, as well as in the internal organs and lymphatic glands in fatal cases. Except in fatal toxic cases they are always present up to the 4th day. They were at first supposed to be microörganisms and believed to be the cause of the disease; but this view has been abandoned, and their nature is not known. Their absence is an indication that the condition is other than scarlet fever; but their presence is not specific, since they have been shown to occur, although in smaller numbers, in diphtheria, measles, lacunar tonsillitis and sepsis. It has frequently been observed that the Wassermann reaction may be positive in scarlet fever, oftenest in the 2nd or 3rd week. Thomas and Pesch[12] found it positive in 36 of 150 cases, and doubtful in 15. Christensen[13] in 110 cases obtained 10 positive reactions, but points out that this was only with cholesterolized antigen, and then the positiveness was only transitory. The sensibility to cutaneous tuberculin-reaction is often diminished during the eruptive period.

Period of Desquamation.—As other symptoms abate and the eruption fades the skin is left dry and rough and desquamation then begins, starting in the localities first invaded by the rash. Speaking very gen-

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[1] Jahrb. f. Kinderheilk., 1910, LXXII, 422.
[2] Przegl. pedyat. Ref. Monatsschr. f. Kinderheilk, Referat., 1914, XIII, 423.
[3] Monatsschr. f. Kinderheilk. Orig., 1917, XIV, 273.
[4] Jour. of Infect. Dis., 1905, II, 375.
[5] Wratch, 1891, No. 41. Ref. Jahrb. f. Kinderheilk., 1893, XXXVI, 409
[6] Arch. de méd. experiment., 1902, XIV, 101.
[7] Jour. of Path. and Bact., 1902, VIII, 82.
[8] Zeitschr. f. Heilk., 1906, XXVII, 433.
[9] Gaz. degli Osped., 1908, XXIX, 433.
[10] Monatsschr. f. Kinderheilk., Orig., 1919, XV, 259
[11] Centralbl. f. Bakt. u. Parasit., 1892, XII, 909.
[12] Zeitschr. f. Kinderheilk., 1921, XXVIII, 208.
[13] Jour. Amer. Med. Assoc., 1923, LXXX, 1118.

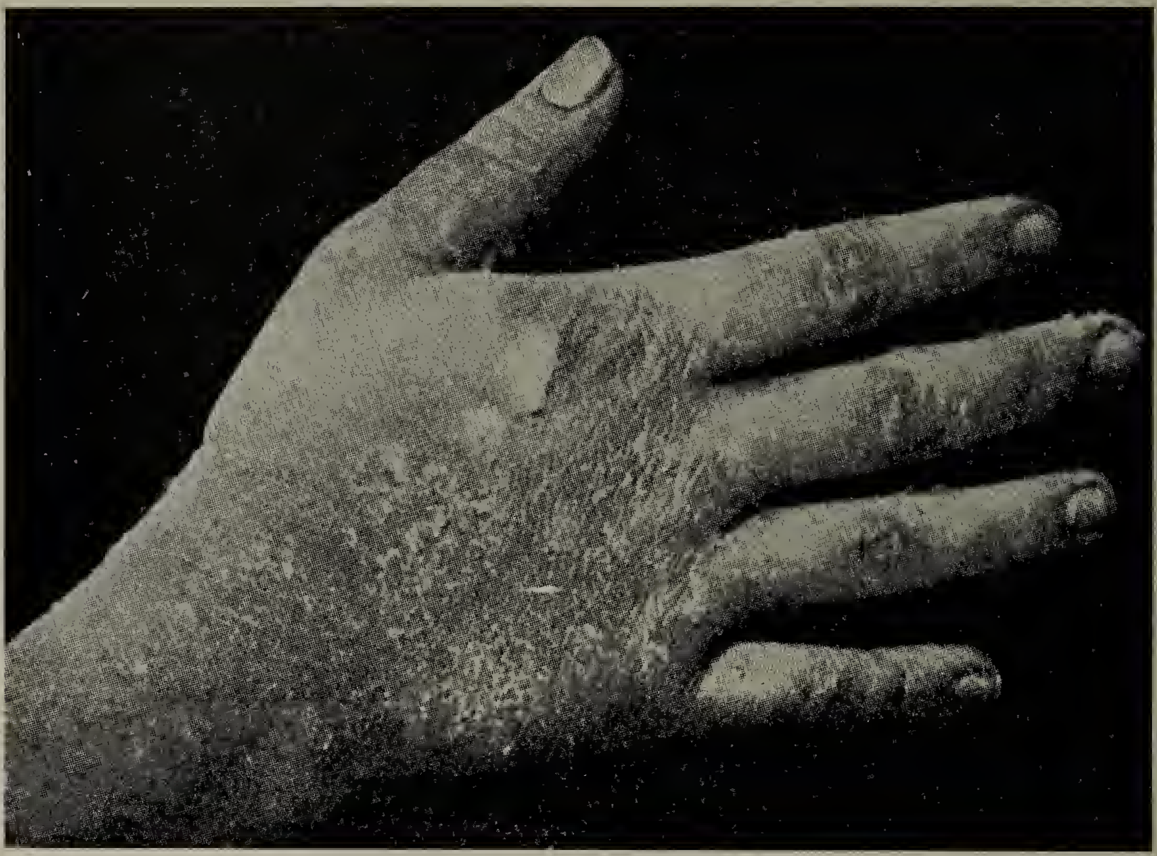

Fig. 55.—Scarlet Fever.

Well-marked desquamation upon the dorsum of the hands and fingers, showing the lamellar peeling. (*Welch and Schamberg, Acute Contagious Diseases*, 1905, 377.)

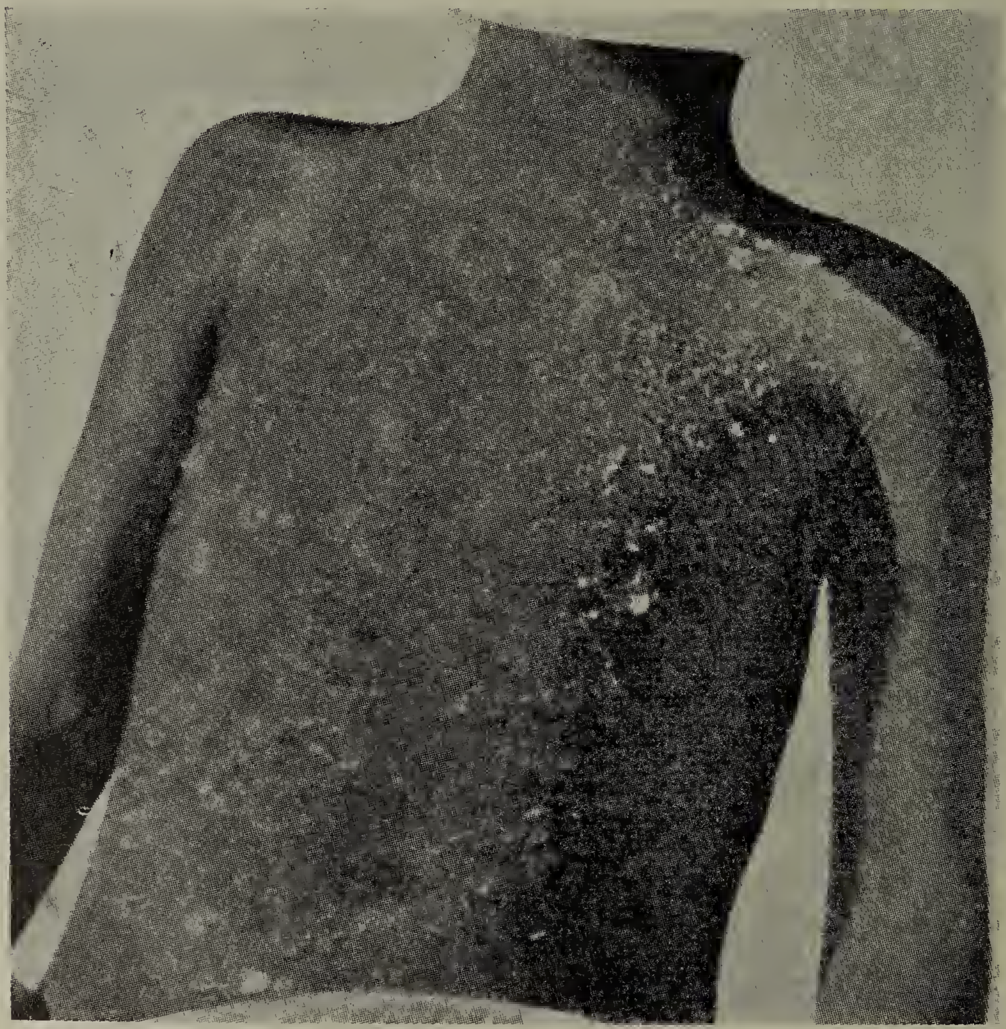

Fig. 56.—Scarlet Fever with Unusually Severe Desquamation.

The enlarging scaling rings are well shown. (*Welch and Schamberg, Acute Contagious Diseases*, 1905, 376.)

erally only, the process may be said to commence at the end of the 1st week of the disease. In many cases it is earlier, and in some much later. The scaling is of 2 forms. The first is a branny desquamation, somewhat similar to that seen in measles, and occurs on the head, neck and upper portion of the trunk. Upon the hands and feet the second form is shown by a very characteristic lamellar peeling, the dead skin coming off in larger or smaller strips (Fig. 55) leaving a sharp contrast between the pink new skin exposed and the remaining old greyish-white skin. It begins here often at the tips of the fingers and toes and especially about the roots of the nails. Occasionally the skin is shed from the hands and feet in the form of true casts. The desquamation of the hands and feet is so characteristic that it is often possible to make the diagnosis on this appearance alone in cases not seen earlier in the attack. On the rest of the body and especially well-marked on the trunk the branny desquamation begins as a small white scale which separates, leaving a pinhole-like opening to the new skin beneath. The skin surrounding this gradually peels off, enlarging the pin-holes to wider circles which finally fuse (Fig. 56).

In some instances the two forms may be combined to a large extent throughout the body, except on the hands and feet where only the lamellar form occurs. Very frequently desquamation is absent or difficult to discover. This is especially true if the body has been bathed or oiled often. The scaling continues for from 10 days or less up to 2 or 3 weeks. It begins last and continues longest on the hands and feet, and here 4 or even 7 or 8 weeks may not see it completed. The average day of the disease on which the desquamation ceased in 91 cases in the Hospital for Scarlet Fever and Diphtheria of New York (1901) was the 47th.[1] Often it may not have commenced on the hands and feet by the time it is nearly or quite over on the rest of the body. Not infrequently after scaling has apparently ceased it recommences, and this process may be repeated several times. The duration and degree of desquamation usually bears some, but no necessary, relationship to the severity of the case and intensity of the eruption. There is reason to believe that it is even possible for desquamation to occur in regions where there has been no rash whatever; consequently other causes than the mere hyperemia of the skin would appear to be active agents in producing it. Nevertheless, as a rule, an intense eruption is attended by abundant desquamation, and vice versa.

Desquamation affects other epithelial structures as well. The nails show the effect of the disorder to a greater or less extent, perhaps in the form of a transverse furrow observed after convalescence, and the hair becomes brittle and falls. The teeth, too, are sometimes affected by the disease and exhibit erosions. In cases where miliaria has been extensive a fine branny scaling may take place early. This is not to be confounded with the true scarlatinal desquamation which occurs later. During the stage of desquamation all other symptoms are absent in normal cases and the patient feels well.

ATYPICAL TYPES OF SCARLET FEVER. (Mild; Severe; Anginose; Malignant; Hemorrhagic).—Different classifications of the varieties of scarlet fever have been made. There are no strict boundaries separating one from another, but a convenient division is the following:

Mild Forms.—The attack may be unusually mild throughout, or some of the symptoms may be absent entirely. There may be a sudden onset with fever, which may disappear completely in the course of a very short time. The eruption may be so slightly developed that its nature is questionable, or it may be so evanescent or limited to so small an area that it is entirely overlooked. The throat may have exhibited only slight traces of redness, and the tongue may never have shown a decidedly characteristic appearance. The child may seem little, if at all, ill. In many of these cases it is only on the occurrence later of desquamation, or perhaps of nephritis as a sequel, that a tardy diagnosis is made. Different varieties of the mild form are seen:

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[1] Report Hospital Scarlet Fever and Diphtheria Patients, 1901, 22.

(*a*) In some cases the diagnosis from the beginning is unquestionable, but the disease runs a very short and mild course (Fig. 57), and the whole process is over in from 2 to 4 days, except perhaps for the desquamation later, although even this is often absent (*Abortive scarlet fever*).

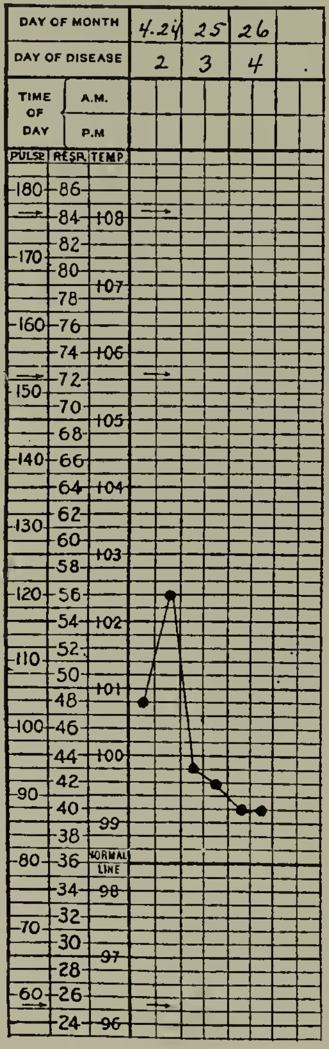

FIG. 57.—SCARLATINA; ABORTIVE FORM.

Weightman F., aged 9 years. Apr. 23, vomited; Apr. 24, rash appearing; Apr. 25, rash at height, well developed; Apr. 26, tongue beginning to peel; May 13, desquamation on hands and feet discovered; none found elsewhere.

(*b*) There seems to be no doubt that the eruption need not develop at all (*Scarlatina sine eruptione*). This is especially often seen in those persons associated with scarlet fever cases who have had the disease before. Such individuals exhibit only some fever and an angina with more or less involvement of the tongue. The condition has been denominated "*scarlatinal sore throat.*" Stevens and Dochez[1] have shown that this may occur in persons with negative Dick tests. Cases of this nature are as capable as others of spreading the disease.

(*c*) Another mild form shows an entire or nearly complete absence of fever (*Scarlatina afebrilis*). With this there is generally a very meagre development of the rash, and of other symptoms. Sometimes, however, the rash is very typical, yet with a temperature normal or rarely exceeding 100°F. (37.8°C.). I have seen this attended by an absolutely characteristic tongue and by subsequent nephritis, making the diagnosis certain (Figs. 58 and 59).

(*d*) In still another form inflammation of the pharynx is absent or represented only by a very slight redness of the pillars without swelling of the tonsils (*Scarlatina sine angina*).

The term "rudimentary form" is sometimes used to denote many of these mild cases with some symptoms undeveloped. It is however equally applicable to some of the severest atypical forms in which only certain of the symptoms of the disease are present although these are severe. The title is therefore confusing.

Severe Forms.—(*a*) In the ordinary severe form as most commonly seen all the symptoms of the disease may be present to an unusual degree. The onset is rapid; there may be violent vomiting; the eruption is very intense and wide-spread and soon becomes dark-red in tint; the temperature is persistently high; the glands of the neck are much swollen; the inflammation of the throat and mouth is severe; delirium is present; there is great restlessness; convulsions may occur and coma supervene; the pulse is rapid and weak; prostration is great and dyspnea may be marked. Death may take place in a few days, or the attack may last for weeks, the patient convalescing very slowly, if at all. The severity of the attack may appear to be the direct result of the scarlatinal poison, or the symptoms may be the varying ones of sepsis.

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[1] Jour. Amer. Med. Assoc., 1926, LXXXVI, 1110.

The severe forms of the disease show numerous variations from this type. These are not sharply differentiated from each other. Thus:

(*b*) The severity may depend, perhaps, on the special prominence of one or a few symptoms or complications, the attack being "rudimentary" so far as the complete development of the disease is concerned.

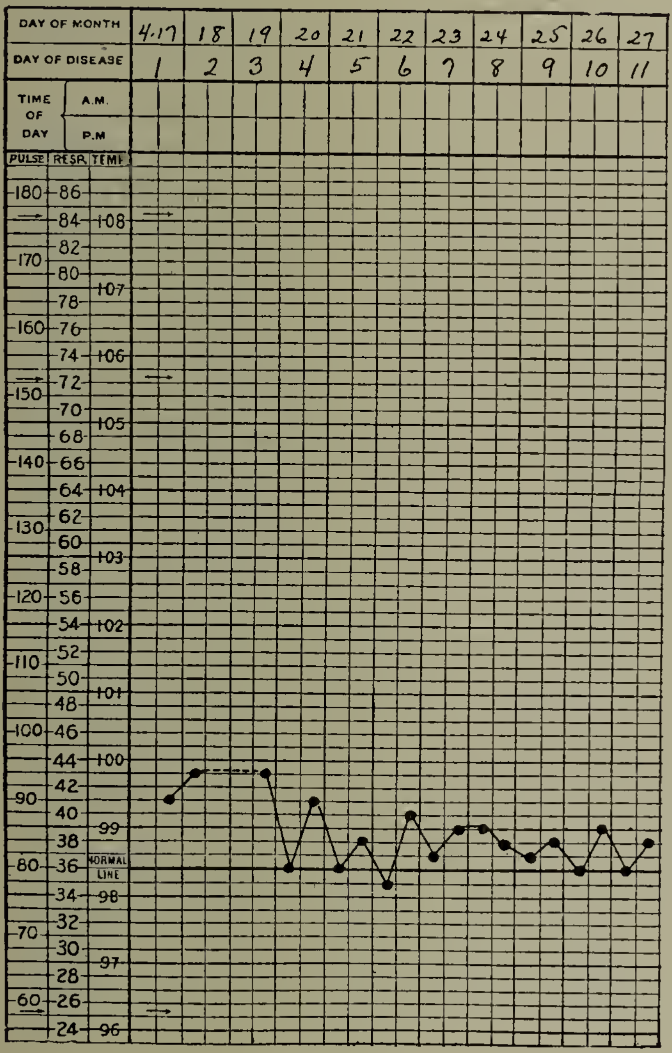

FIG. 58.

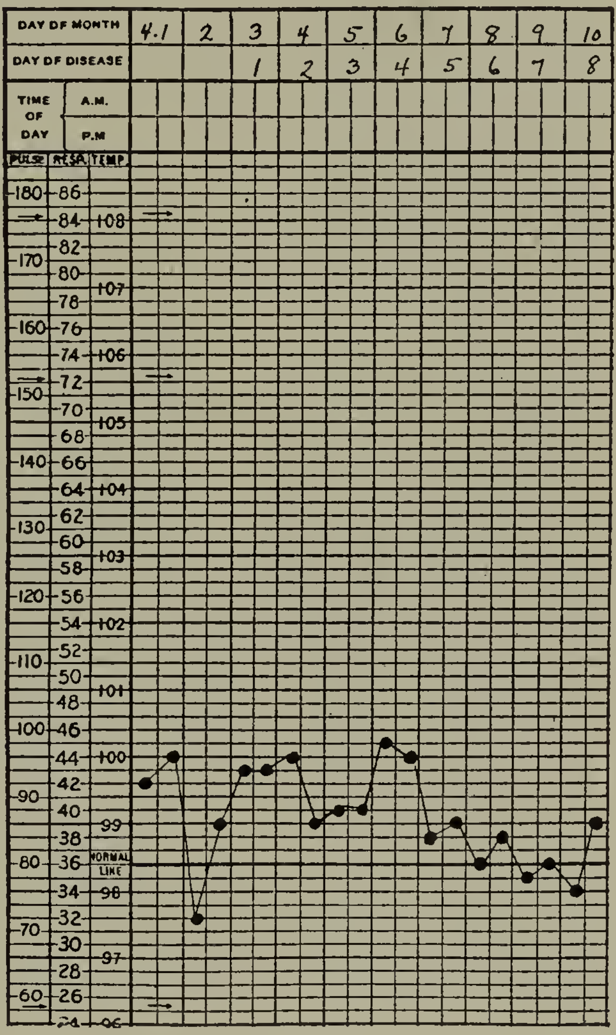

FIG. 59.

FIG. 58.—SCARLATINA AFEBRILIS.

Henry P., aged 9 years. Typical rash, slight redness of throat, moderate development of strawberry tongue. Later slight scarlatinal desquamation and albuminuria with casts.

FIG. 59.—SCARLATINA AFEBRILIS.

Frank M., aged 3 years. Recovering from typhoid fever. Apr. 3, typical miliary scarlatinal eruption, tonsils red, secretion in follicles, tongue beginning to peel; Apr. 5, rash still very bright, miliaria drying; Apr. 8, profuse branny desquamation from the miliaria; Apr. 11, typical scarlatinal desquamation.

(*c*) There occur differences in the development of the eruption. In many severe cases instead of being unusually well-marked it is faint, localized, blotchy, and tends constantly to fade.

(*d*) In one variety the disease is characterized not so much by development of any localized symptoms as by long continuance of the fever without discoverable cause, there occurring such nervous symptoms as headache, delirium, apathy, etc., or stupor and the symptoms in general of the typhoid state, on account of which the title *Scarlatina typhosa* has been applied to it.

(*e*) A fairly distinctive variety, dependent in reality on a complication, yet so common that it may well be described here, is the anginose form (*Scarlatina anginosa*) (Fig. 60). In this the disease usually starts as an ordinary case and it is not until the 3d or 4th day or later that the characteristic symptoms appear. Sometimes the fever has commenced to fall, and the child seems about to convalesce, when, with an increase of temperature, the anginose symptoms begin. Pseudomembrane, the result probably of invasion by streptococci, then develops on the tonsils and may spread to the uvula and pillars, the posterior wall of the pharynx, the nose, and occasionally the mouth. The submaxillary lymphatic glands become very much swollen. The appearance may be that typical of faucial diphtheria, and inasmuch as scarlet fever is so often complicated by a true diphtheritic invasion, the diagnosis can be made only by a bacteriological examination. In severe cases fever remains high, the pulse becomes weak, nervous symptoms are marked, and there develops a profound general septic state with great prostration. In other instances the lesions may go on to necrosis, and be attended by extensive sloughing with abscesses.

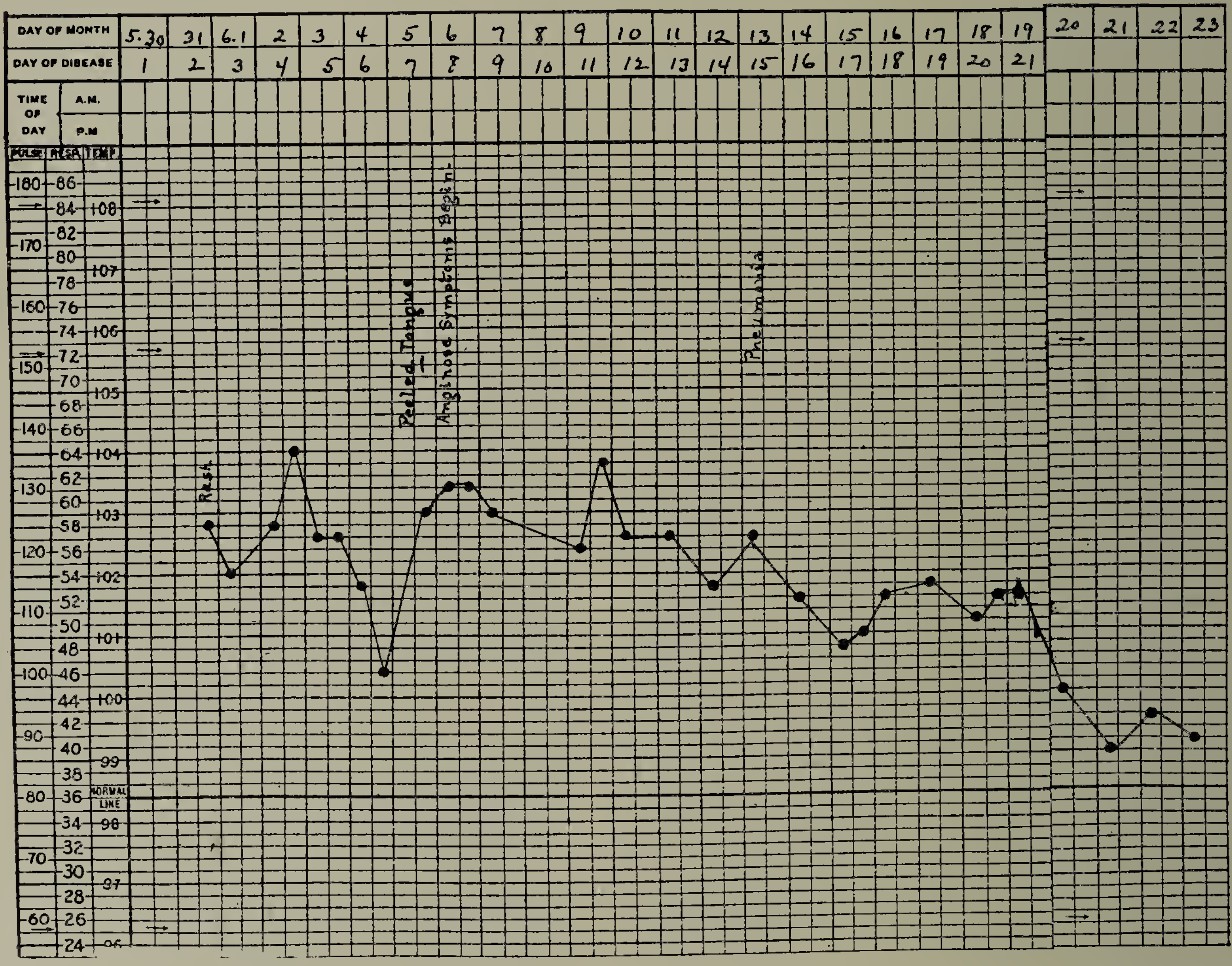

Fig. 60.—Scarlet Fever Followed by Anginose Symptoms and Later by Pneumonia

Minnie L., aged 7 years. May 30, vomiting in the evening and night; May 31, rash appeared, throat inflamed; June 2, throat more inflamed; June 5, tongue peeling; June 7, rash disappearing, inflammation of throat has increased, tonsils now thickly covered by white exudate; June 12, throat still inflamed, but better; pneumonia of low grade developed

Even in the milder cases of anginose scarlet fever the local condition lasts for a week, more or less, and decidedly prolongs the attack while it adds greatly to the discomfort and debility of the patient and to the danger of involvement of the ears.

(*f*) One of the severe types of scarlet fever is the *malignant* or *cerebral variety* (*Scarlatina maligna*). The onset of this form is extremely sudden with a preponderance of nervous symptoms, the child appearing to suffer from an over-powering intoxication. There occur repeated vomiting, intense headache, very high fever, and restlessness and delirium soon passing into coma, perhaps with convulsions. The urine may be suppressed, the rash may be very intense, dark-red and often of a purplish cast from the presence of numerous petechiæ. (Fig. 61.) Sometimes there are hematuria, purpura, epistaxis, and hemorrhages from other parts of the body (*Scarlatina hemorrhagica*). In other cases the rash is but little developed or even entirely absent. The inflammation of the throat likewise may not be present. In such cases there may be nothing on which to base a diagnosis, unless the case occur in connection with others. Death may take place in a few days, or even in a few hours. In still other instances the disease may begin in the ordinary manner and the evidence of malignancy not appear until the 2d or 3d day.

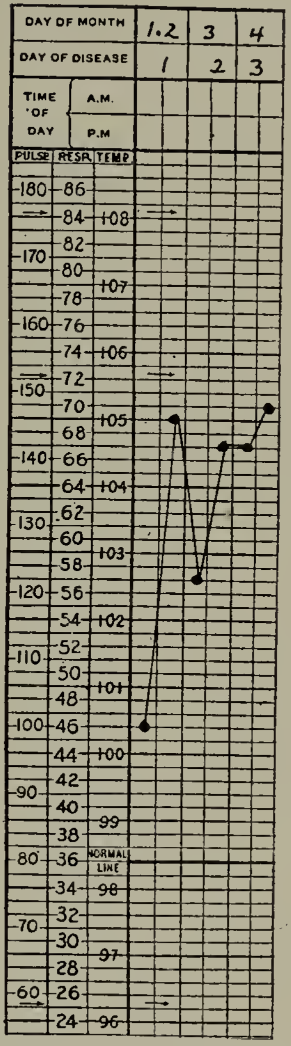

FIG. 61.—MALIGNANT SCARLET FEVER.

Domenick S., aged 2 years. Jan. 2, vomiting, dyspnea and irregular respiration; Jan. 3, red, uncharacteristic eruption, throat red, tonsils enlarged; Jan. 4, wide-spread, abundant petechial eruption combined with the scarlatinal rash, coma, respiration rapid and dyspneic, pulse rapid, tonsillar exudate; Jan. 5, death at 1 A. M.

Complications and Sequels.—Numerous disordered conditions may accompany or follow scarlet fever, but not many of these are of frequent occurrence. In 153,607 cases reported by the Metropolitan Asylums Board from 1900 to 1909 (Goodall)[1] the incidence of the more important complications equalled: otitis 13.1 per cent., adenitis 8.1 per cent., nephritis other than simple albuminuria 4.6 per cent., and arthritis 3.5 per cent.

Throat and Nose.—Different varieties of faucial involvement have been described, chief among them being the erythematous, pseudomembranous, and gangrenous forms. There exists, however, no sharp boundary line and they shade into each other. The erythematous throat, combined often with some engorgement of the tonsillar follicles with secretion, is always seen in typically normal cases. It is only when the process goes on to the development of a pseudomembrane, perhaps followed by extensive necrotic changes, that the condition can be considered as a complication. This occurs so often that it characterizes one of the variant forms of the disease. There may even be produced a condition which is clinically indistinguishable from that seen in diphtheria. My own experience has been that of others; that severe and even finally fatal cases of this condition may occur in which it is doubtful whether a scarlatinal rash has ever been present. The nose is often involved by the pseudomembrane, and an irritating thin discharge takes place from it which excoriates the upper lip. Nasal respiration may become impossible. Only occasionally is the larynx invaded. The symptoms attending this inflammation of the nose and throat have already been described (p. 362).

Ears.—Otitis media is one of the frequent complications. It is especially prone to develop when the inflammation of the throat has been severe, although by no means only then, the disease spreading along the Eustachian tube to the ear. Younger children are most disposed to it. Otitis may occur either at the beginning of the disease, or oftener as a sequel after desquamation has commenced or convalescence been entirely established. The proportion of cases developing it varies greatly with the epidemic. Caiger[1] reports suppurating otitis media in 11.05 per cent. of 4015 cases, Pugh[2] in 15 per cent. of 11,000 cases, and Borden[3] in 11 per cent. of 2232 cases and 454 autopsies. Baum[4] found it in 1.7 per cent. of 628 cases although some pain in the ears was experienced in about 35 per cent. Both ears are generally affected. The greater frequency of perforative otitis in the severe cases of scarlet fever is shown by the analysis of Holmgren[5] of 9590 subjects with this disease. In 511 fatal cases perforation had occurred in 36 per cent.

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[1] Garrod, Batten and Thursfield, Diseases of Children, 1913, 1009.

Otitis may terminate in complete recovery, generally after perforation, but many instances of some degree of permanent deafness owe their origin to a scarlatinal otitis. This had been the history in 21.17 per cent. of Burkhardt-Merian's[6] 85 cases of deafness. In older children with normal cerebral condition the otitis is generally accompanied by severe earache and deafness; yet pain may be entirely absent. In those, however, in which mental dullness exists, and in all younger patients, the symptoms are very obscure. Sometimes a return of fever with fretfulness is the only indication until spontaneous rupture of the drum-membrane occurs. The presence of tenderness over the region in front of the tragus or behind the ear is often the chief symptom unless the drum-membrane be frequently examined. The greatest watchfulness of these cases is therefore necessary. One of the chief dangers is the development of mastoid disease with subsequent purulent meningitis and septic symptoms. Gardiner[7] reports that of 300 cases of otitis following scarlet fever, mastoid disease developed in 21 (7 per cent.). The inflammation of the mastoid may come on without the previous existence of otitis having been recognized at all. Even in cases where perforation of the drum-membrane has occurred the presence of a free flow of pus does not insure against the development of mastoiditis.

Cervical Adenitis.—The lymphatic glands of the neck are always somewhat enlarged in all cases where sore throat is a prominent symptom. Sometimes they become greatly so, and may advance to the formation of abscess. This takes place oftenest as a sequel in the 2d week or later. It is attended by fever, but not by any marked septic symptoms, and is relieved by rupture or by incision, but most often in our experience subsides spontaneously in a few days. In bad cases of the anginose form, however, the engorgement and subsequent necrosis of the lymphatic glands is contemporaneous with the angina. The swelling may become extreme and a cellulitis of the tissues of the neck may develop, which can reach such an extent that the natural depression beneath the jaw is everywhere completely obliterated. The head is thrown back and respiration may be much interfered with. This may go on to the formation of pus, which makes its way either toward the surface or along the sheaths of the muscles or the great vessels. Very extensive sloughing may take place, and blood-vessels may be perforated. The attending symptoms are always of a gravely septic nature. Adenitis of a degree sufficient to be called a complication was found by Schick[1] in 7.2 per cent. of 990 cases; and by Caiger and Dudgeon[2] in 11.4 per cent. of 10,983 cases.

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[1] Allbutt's System of Med., 1897, III, 150.

[2] Lancet, 1905, I, 273.

[3] Bost. Med. and Surg. Jour., 1913, CLXVIII, 221.

[4] Jour. Amer. Med. Assoc., 1903, XLI, 906.

[5] Otolaryngologiska Medelanden, 1912, I, 1. Ref. Jahrb. f. Kinderheilk., 1913 LXXVIII, 475.

[6] Volkmann's Samml. klin. Vortr.; Chir., No. 54, 1489.

[7] Jour. Laryngol. and Otol., 1922, XXXVII, 497.

Arthritis (Fig. 62).—This may often complicate or follow scarlet fever, generally in those past early childhood. Heubner[3] saw it in 8 per cent. of his 358 cases; Ashby[4] in 2.4 per cent. of 500 cases; Heiberg[5] in 19.1 per cent. of 1000 cases; Baum[6] in 3.7 per cent. of 628 cases, Brade[7] in 6.9 per cent. of 868 cases; Morgenstern[8] in 3 per cent. of 470 cases, and Joe[9] in 3.76 per cent. of 24,012 cases. In the majority of instances it is a synovitis (*scarlatinal rheumatism*) occurring about the end of the 1st week, sometimes earlier or later. It involves especially the hands, fingers and elbows, although any of the joints may be affected. The attack lasts generally not more than 3 or 4 days. It is a mild affection characterized by pain and with generally moderate swelling and redness. There may, however, be nothing visible on inspection of the joints. The

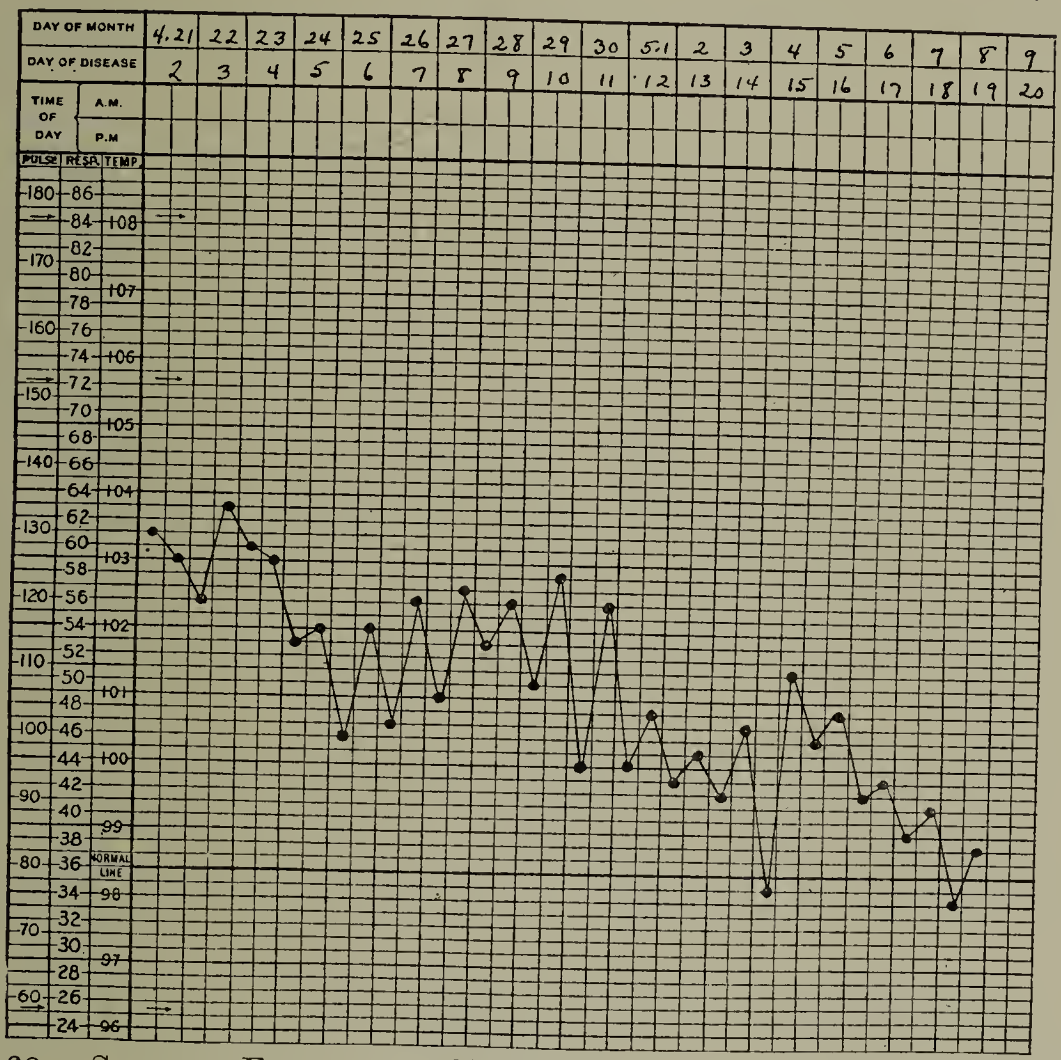

FIG. 62.—SCARLET FEVER WITH TEMPERATURE PROLONGED BY ARTHRITIS.

Genette F., aged 5 years. Apr. 20, fever, vomiting; Apr. 21, well-developed eruption; Apr. 22, a few white spots on tonsils; Apr. 23, rash at maximum, tongue peeling; Apr. 24, rash fading; Apr. 26, multiple arthritis, involving fingers, elbows, knees and ankles; May 5, joints improving.

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[1] Arch. f. Kinderheilk., 1905, XLII, 459.

[2] *Loc. cit.*, 452.

[3] Deutsche Klinik, 1905, VII, 286.

[4] Brit. Med. Jour., 1883, II, 514.

[5] Zeitschr. f. Hyg. u. Infektionskrankh., 1910, LXV, 237.

[6] *Loc. cit.*

[7] Inaug. Dissert. Leipzig, 1904. Ref. Monatsschr. f. Kinderheilk., 1904, III, 348.

[8] Zeitschr. f. Kinderheilk., Orig., 1918, XIX, 56.

[9] Edinburgh Med. Jour. 1924, XXXI, 341.

likelihood of its developing bears no relationship to the severity of the scarlatinal attack. It is probably not truly rheumatic in nature, although cases are occasionally seen in which true rheumatism and scarlet fever appear to be combined.

Another less frequent form of arthritis is apparently septic in nature and goes on to suppuration. It may attack one or more of the larger joints, and possesses a graver prognosis.

Nephritis.—This is one of the most common and often most serious of the complications or sequels of scarlet fever. Some writers regard a degree of involvement of the kidneys as one of the constant symptoms of the disease. The presence, however, of slight albuminuria, perhaps with cylindroids and even hyaline casts, as often seen in the first days of an attack of scarlet fever when the temperature is high, is what is liable to occur in any of the febrile infectious diseases, and cannot properly be looked upon as evidence of nephritis. In the severer cases of scarlet fever, however, particularly when any septic process is present, as for instance in the throat, an acute diffuse nephritis frequently develops during the height of the disease. It is seen with great frequency in the malignant cases. The nephritis may come on with suddenness and severity, with great diminution of the urinary secretion and the presence in it of much albumin and with casts and blood. The symptoms are those of uremia rather than of dropsy. Convulsions often occur. In cases not so severe there may be no special symptoms, or those present may be masked by the graver ones of the scarlet fever itself. It is noteworthy that even in the milder cases the urine is liable to be hemorrhagic.

The most frequent form of nephritis, however, is that denominated *post-scarlatinal*, occurring as a sequel. This develops in the 3d or 4th week after the onset of the scarlet fever, and is just as liable to follow the mildest cases as any others. The lesions are oftenest those of a glomerular nephritis. The onset may be sudden with wide-spread dropsy and with very scanty secretion of a smoky or coffee-colored urine containing much albumin, blood-cells, granular epithelium and very numerous casts of different forms. Convulsions, vomiting and other uremic symptoms may develop. Complete anuria may last for a time (Northrup[1] 5 days; recovery). Death may follow promptly, or there may be a gradual amelioration of the symptoms, the urine becoming more abundant and of lower specific gravity, the fever lessening, and the child showing evidences of anemia. In the majority of cases, however, the symptoms are much less severe and their onset often insiduous. There may be only slight edema, especially of the hands, feet and eyelids; a moderate degree of fever, irritability and restlessness; and but a small amount of albumin in the urine with a few casts of different sorts. As in the severer but not fatal cases, convalescence is slow, the albumin and casts continuing perhaps for months and finally disappearing, and recovery being complete in the majority of instances. There is noted a decided tendency for the nephritis to relapse, and this may persist for many months. There have been instances reported, and even some epidemics, in which edema has developed subsequent to scarlet fever yet without any evidence in the urine of an inflammation of the kidneys (Quincke).[2] In some such cases, however, the lesions of nephritis have been found post-mortem In only comparatively few instances does the nephritis become chronic.

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[1] Med. Rec., 1910, LXXVIII, 706.

[2] Berlin. klin. Wochenschr., 1882, XIX, 409.

A study by Rosenfeld and Schrutka v. Rechtenstamm[1] of 94 individuals who had had acute scarlatinal nephritis several years previously failed to reveal a single instance of severe chronic nephritis remaining.

Nephritis may, of course, lead to other complications, such as cardiac hypertrophy and dilatation, effusion into the pleural and pericardial sacs, and the like. The relative frequency of the occurrence of nephritis in connection with scarlet fever varies greatly with the epidemic. The views of writers as to what constitutes the evidence of nephritis also influence the statistics. Ashby and Wright[2] estimate the average proportion of patients attacked as 6 per cent. Caiger and Dudgeon[3] give 4 per cent. in 10,983 cases; Royer[4] 7.76 per cent. of 756 cases. Others have given higher figures as, for instance, Barash,[5] 16.1 per cent. of 1438 cases.

The causes of nephritis in scarlet fever are not certainly known. In the cases associated with septic processes some pus-germ is probably the active agent. In those developing during malignant scarlet fever and in the post-scarlatinal cases it is probably the toxic products of the scarlet fever germ. The predisposing influence of external conditions is uncertain. (Fuller reference to the pathology, prognosis and treatment of scarlatinal nephritis will be found under the heading of Nephritis.)

Circulatory System.—Functional transitory disturbances of the heart are not uncommon during scarlet fever, but serious lesions directly produced by the disease are less frequent. Endocarditis and pericarditis occur occasionally as the result of a complicating nephritis or a rheumatic synovitis, or as one of other septic manifestations. I have seen very few cases in which chronic valvular disease could be without question attributed to an antecedent scarlatina. Embolism of the pulmonary or other arteries has been observed. Phlebitis, perhaps followed by gangrene, may rarely occur. Huenekens and Siperstein[6] report an instance and collect several other cases from medical literature. Silberstein[7] added 1 case of symmetrical gangrene to 13 other recorded instances. Myocardial degeneration is present to some extent in many cases, especially if these are severe and accompanied by high fever. If considerable, it may produce acute dilatation. Dilatation, however, and especially hypertrophy, generally depend upon a nephritis and are sequels rather than complications. Hypertrophy the result of this cause comes on rapidly and may present demonstrable physical signs even within a week from the onset of the renal inflammation. Pospischill[8] believed that the heart is often characteristically affected in scarlet fever, there being a splitting of the first sound and a murmur suggesting pericarditis; these depending upon myocardial changes. The symptom is observed especially at the beginning of the attack. Lederer and Stolte[9] confirm this observation. The average frequency of involvement of the heart in scarlet fever is illustrated by Rosenbaum's[10] series of 1770 cases, in which cardiac complications occurred in 106. Twelve of these had had previous endocarditis. In the remaining 94 the following diagnoses were made: myocarditis in 88; endocarditis in 4; pericarditis in 3.

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[1] Zeitschr. f. Kinderheilk., Orig., 1912, IV, 265.
[2] Diseases of Children, 1893, 247.
[3] *Loc. cit.*
[4] Penna. Med. Jour., 1906–7, X, 286.
[5] Deutsch. med. Wochenschr., 1915, XLI, 4.
[6] Amer. Jour. Dis. Child., 1923, XXVI, 447.
[7] Jahrb. f. Kinderheilk., 1912, LXXV, 350.
[8] Wien. klin. Wochenschr., 1907, XX, 1089.
[9] Jahrb. f. Kinderheilk., 1911, LXXIV, 395.
[10] Arch. Int. Med., 1920, XXVI, 424.

Respiratory System.—The general opinion is that pulmonary complications and sequels are not nearly so frequent in scarlet fever as in some other diseases. Hutinel,[1] however, found that about ⅓ of the deaths in 2500 cases were from bronchopneumonia, and that croupous pneumonia and pleurisy also were frequent complications or sequels. This does not, however, express the incidence of the pulmonary complications except in fatal cases. The involvement of the nasal mucous membrane has been spoken of in connection with the throat. Pseudomembranous laryngitis not associated with the germs of diphtheria is of rare occurrence. Pleurisy with effusion, often purulent, is an occasional sequel. Its onset is frequently very insidious and entirely unsuspected. Bronchitis sometimes develops. Bronchopneumonia is a dangerous complication, occurring usually only in association with nephritis. Croupous pneumonia is generally believed to be uncommon except in combination with nephritis. The opinion of Hutinel opposes this. Edema of the lungs and edema of the glottis sometimes occur as a result of disease of the kidneys.

Gastrointestinal Complications.—These are not, as a rule, troublesome. Vomiting is very common at the beginning of the attack, and ulcerative stomatitis is a complication occasionally seen in young children. It is usually mild, but sometimes as a sequel is severe. Diarrhea is a frequent complication. It is generally of a catarrhal nature, but may be inflammatory. In some cases it is very obstinate and debilitating. It may develop during the height of the disease, or be one of the earliest symptoms of the eruptive stage. Slight icterus is very common.

Nervous System.—Among nervous complications and sequels are especially to be mentioned repeated convulsions. At the onset of the attack these generally have little significance, but later they are suggestive of uremia. Encephalitis sometimes occurs with its various manifestations. Meningitis and thrombosis of the cerebral veins or sinuses may develop as sequels depending upon an otitis. Occasionally a meningitis occurs earlier in the disease and independently of any affection of the ear. Hemiplegia has been known to follow meningeal hemorrhage or be caused by embolism dependent upon an endocarditis. Paralysis has also been seen as a result of neuritis or of disease of the spinal cord. Chorea and epilepsy are sequels occasionally reported, and various psychoses are on record. Amaurosis may develop as a result of nephritis.

Cutaneous Complications.—Urticaria and erythema are sometimes observed after the disappearance of the scarlatinal eruption, and eczema is not an uncommon sequel. Rolleston[2] found herpes facialis 27 times in 413 cases.

Other infectious diseases may occur in combination with scarlet fever or as sequels to it. One of the most frequent and serious combinations is that of scarlet fever and diphtheria, the latter developing generally in a patient already suffering from the former. It is more prone to appear from the 3d to the 6th week after the attack of scarlet fever, sometimes earlier or later. Measles and scarlet fever have often occurred together. Among other combinations are those with varicella, variola, pertussis, erysipelas, typhoid fever, etc. Either disease may develop first. Tuberculosis already in the system may be brought into activity by the occurrence of scarlet fever.

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[1] Arch. de. méd. des enf., 1916, XIX, 57.

[2] Brit. Jour. of Dermatol., 1910, XXII, 309.

Among other rarer complications or sequels which have been reported may be mentioned perichondritis of the larynx; esophagitis; necrotic inflammation of the stomach; gastric hemorrhage; optic neuritis; periostitis; peritonitis; noma; furunculosis; vulvo-vaginitis; myositis; pemphigus; and glycosuria.

Relapse.—Relapse is occasionally seen, but is rare. McCollom[1] found it 4 times in 1000 cases (0.4 per cent.) and Sloan[2] 154 times in 14,143 cases (1.8 per cent.) of the Hospitals of the Metropolitan Asylums' Board. It consists in the return of some or all of the symptoms after the disease is apparently over, but while the original infection is doubtless still in the system. The mere reappearance of the eruption after a brief fading is not to be classified here; nor is the simple return of temperature an indication of relapse, since this often occurs, sometimes without the discovery of the cause being possible. There must be a combination of symptoms sufficient to justify the diagnosis of a true relapse. It is seen oftenest in the middle or at the end of the 4th week and may be either milder or more severe than the primary attack. Lettry[3] in analyzing 46 collected instances found relapse oftenest between the 15th and 35th days.

Recurrence.—Recurrence of the disease in the form of a distinct second attack after a considerable interval is rarely seen. One attack usually protects from subsequent ones. One hears of instances frequently, especially from the laity, but most of these are certainly mistakes in diagnosis. Thomas[4] reports a number of undoubted cases collected from medical literature, but has himself observed it but once in hundreds of cases, and Henoch[5] saw it but once. J. McCrae[6] reported 9 instances said to be second attacks in 850 cases (1 per cent.), and Weissenberg[7] 7 cases. I have treated 1 child with 2 distinct and undoubted attacks separated by an interval of a year.

In speaking of recurrence no reference is intended here to that which might with some propriety be called this; viz. the development of a scarlatinal angina in exposed adults who had previously had the disease.

Prognosis.—The continuously mild cases nearly invariably recover; yet cases beginning in this way may later become severe, or a dangerous complication or sequel may arise. The prognosis in every instance must, therefore, be always most guarded throughout. Only after all danger of sequels is over can one feel at ease. The mortality appears to vary greatly with the epidemic and is different in different countries. Thus in 11,216 cases reported in Philadelphia during 5 years the mortality was 5.4 per cent. (Graham);[8] and in 1072 cases reported in 1915, 2.4 per cent. (Ostheimer).[9] Of 21,834 cases occurring in Hamburg during 10 years 6.8 per cent. died (Reincke).[10] Of 15,137 cases occurring in Stockholm during 21 years 16.3 per cent. died (Carlsson),[1] the lowest yearly mortality being 2.8 per cent. and the highest 28.8 per cent. Of 1598 cases in the Manchester Children's Hospital during 10 years 11.8 per cent. died (Ashby and Wright).[2] In 1000 cases in the Boston City Hospital the mortality was 9.8 per cent. (McCollom).[3] Of 84,380 cases in Norway collected by Johannessen[4] 14.17 per cent. died, while of 167,840 cases in the Metropolitan Asylums Board's Hospitals during 13 years but 4.3 per cent. died (Caiger).[5]

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[1] Boston City Hosp. Reports, 1899.
[2] Lancet, 1903, I, 436.
[3] Thèse de Paris, 1907–8.
[4] Ziemssen's Handb., 1874, B. II, Th II, 176.
[5] Kinderkrankheiten, 1895, 675.
[6] Canadian Med. Assoc. Jour., 1911, 1, 293.
[7] Arch. f. Kinderheilk., 1909, LII, 17.
[8] Jour. Amer. Med. Assoc., 1916, LXVII, 1272.
[9] Amer. Jour. Pub. Health, 1916, VI, 1104.
[10] Bericht d. Medicinalraths, etc., 1894, 58. Ref. v. Jürgensen, Nothnagel's Encyclopedia of Pract. Med., Scarlatina, 603.

These figures show the great variation in statistics. It is to be noted that the more recent of them give the lowest death-rate, and it seems certain that the mortality of the disease has been in general diminishing. Rolleston[6] reports the mortality in the Hospitals of the Metropolitan Asylums' Board as only 1 per cent. in 1921. There is, however, a great range between different epidemics and localities, and the ordinary mortality might be said to range from 1 to 10 per cent. In Amsterdam, Herderschee[7] notes that the mortality was 4.8 per cent. in 1896 and only 0.45 per cent. in 1912. In the Philadelphia Hospital for Contagious Diseases, during a period of years up to and including 1914, there were 9,591 cases with a mortality of 6.1 per cent.; and, in the 10 years from 1915 to 1924 inclusive, in 16,727 cases the mortality was 2.5 per cent. (Woody).[8]

Certain conditions, however, affect the prognosis unfavorably. The influence of epidemic and locality has already been mentioned. The disease in hospitals has usually a higher death-rate than in ordinary practice, probably because the worst cases are treated there. The age is a prominent factor. In general the younger the patient the greater is the danger of death. An average death-rate in the 1st year may be placed at from 20 to 40 per cent., and from 2 to 5 years at about 10 per cent. A possible exception is to be made for the very early months of life, at which time scarlet fever is believed to be generally mild. The development of severe nephritis during the attack greatly increases the death-rate. Post-scarlatinal nephritis does so to some extent, although to a much less degree. Symptoms of sepsis, especially those dependent on severe involvement of the throat, are unfavorable. In proportion also to the intensity of the action of the scarlatina toxin the danger grows. Hence malignant cases are nearly invariably fatal. A very rapid pulse, an unusually high temperature, evidence of decided involvement of the nervous system or of the throat, and an irregularly or poorly developed eruption if accompanied by severe symptoms all point to the existence of a grave case. The presence of the exudative-lymphatic diathesis tends to the production of severe anginose and lymphatic complications. Sudden death in scarlet fever is usually due to myocardial weakness. The literature on this subject is reviewed by Krauss.[9]

Diagnosis.—This is not difficult in typical cases, and rests chiefly on the sudden onset with vomiting, high fever and sore throat, followed by the rapid appearance and characteristic spread of the punctate eruption, and later the peculiar desquamation. In very mild cases, however, as well as in the rapidly malignant ones, the diagnosis is often extremely difficult or even impossible. In the latter the rash may be entirely uncharacteristic or even absent. In the former it may be limited to a small area, poorly developed, or transitory and entirely overlooked, the tongue and throat may not present typical changes, and desquamation may not be discovered. Other cases may present only the affection of the throat without the cutaneous eruption. In some instances no desquamation can be found at any time, especially if inunction has been employed. In Negroes the diagnosis is often very difficult, owing to the concealment of the eruption by the dark hue of the skin. In some such cases the redness of the palms and soles is suspicious; in others only the occurrence of nephritis, otitis, or other sequel, or of the characteristic desquamation, makes the diagnosis clear. It is the clinical picture as a whole rather than any one symptom upon which the diagnosis must be based.

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[1] Ref. v. Jürgensen, *loc. cit.*, 603.
[2] Dis. of Child., 1893, 240.
[3] Report Boston City Hospital, 1899.
[4] Die epidem. Verbreitung d. Scharlachfiebers in Norwegen, 1884.
[5] Allbutt and Rolleston, Syst. of Med., 1906, II, 1, 429.
[6] Acute Infect. Dis., 1925, 259.
[7] Zentralbl. f. inner. Med., 1924, XLV, 569.
[8] Personal communication.
[9] Jour. Amer. Med. Assoc., 1923, LXXX, 454.

As an aid to the recognition of the scarlatinal eruption Pastia[1] described a uniform linear redness in the transverse fold of the skin at the elbow, more intense than the scarlatinal eruption. It is present at the very beginning of the eruptive stage and lasts after the rash has faded. It appears to be produced by capillary hemorrhages. Its presence in scarlet fever and its absence in measles is confirmed by Bizzarri,[2] Lippmann[3] and Mouriquand.[4] The Rumpel-Leede sign[5] is another supposed evidence of the existence of scarlet fever. It consists in the development of punctate hemorrhages into the skin of the elbow-fold after compression of the upper arm has continued from 5 to 20 minutes. It is claimed to be especially applicable early in the disease. Experiments by Michael,[6] Stephan[7] and others showed that a positive sign could be elicited in normal children and in many diseased conditions, and was, therefore, no proof of the existence of scarlet fever. Leede believed that a negative result excludes scarlet fever. Schultz and Charlton's[8] extinction phenomenon has been found of practical aid by many (Neumann;[9] Reymond;[10] Rojo;[11] Toomey and Nourse[12]). It would appear that a positive reaction points strongly to the existence of scarlet fever, but that a negative reaction does not certainly exclude the disease. The nature of the test is as follows: Intradermal injection of 0.5 to 1 c.c. of normal human serum blanches or dispells the eruption over an area of a few sq. cm. after 6 to 8 hours, the blanching persisting until the rash has finally faded. This local pallor occurs in no other rash than that of scarlet fever. The test should be made in the first 3 days of the disease. Any human serum may be used except that taken from the patient himself or from other cases at the height of scarlet fever. Such serum does not induce blanching, nor does that of other normal animals. If normal serum is employed in this test it would seem better to use that from an individual whose Dick test is negative, and who therefore possesses scarlet fever immune-bodies. It has been found by Blake, Trask and Lynch,[1] however, that an anti-scarlatinal serum from horses immunized with the special hemolytic streptococcus will also cause persistent local disappearance of the rash.

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[1] Arch. de méd. des enf., 1911, XIV, 130.

[2] La Pediat., 1912, XX, 898.

[3] Pediatrics, 1912, XXIV, 358.

[4] La Médecine, 1923, IV, 836.

[5] Rumpel, Münch. med. Wochenschr., 1909, LVI, 1404; Leede, Münch. med. Wochenschr., 1911, LVIII, 293; 1673.

[6] Arch. of Pediat., 1912, XXIX, 298.

[7] Berl. klin. Wochenschr., 1920, LVII, 437.

[8] Zeitschr. f. Kinderheilk., Orig., 1918, XVII, 328.

[9] Deutsch. med. Wochenschr., 1920, XLVI, 566.

[10] Schweiz. med. Wochenschr., 1921, LI, 719.

[11] Semana med., 1922, I, 776.

[12] Amer. Jour. Dis. Child., 1924, XXVII, 95.

The fact that the Dick test is positive in the early days of scarlet fever and becomes negative about the 7th to the 10th day of the disease (Zingher)[2] may be a helpful point in diagnosis.

Certain diseases are especially to be distinguished from scarlet fever. *Rubella* presents at times the closest possible resemblance, and even careful and repeated observation may not solve the question in isolated cases, or even in localized epidemics. The absence of general symptoms and of scarlatinal changes in the mouth and throat aid in its recognition. So, too, there may nearly always be found somewhere on the body the characteristic macular eruption of rubella, even when the greater part of the surface is covered by a scarlatiniform rash. (See Rubella, p. 409.) Yet I do not know of other two affections where differential diagnosis can be at times the source of more perplexity. *Measles* seldom causes any confusion. When it presents a poorly developed eruption it may, however, be distinguished from some cases of scarlet fever only with the greatest difficulty. The longer incubation and invasion and the catarrhal symptoms of the latter, the buccal eruption, and the slower development of the rash are suggestive.

Diphtheria, if attended by an erythema in the early stages, may not be at first distinguishable from scarlet fever with severe anginose symptoms. The difficulty arises, too, in the cases of scarlet fever in which no rash has been discoverable. The close study of the sequence of symptoms will often be of service. The rash in diphtheria is not very frequent, and is generally confined to the trunk. The discovery of the Klebs-Loeffler bacilli would settle the question, were it not that the two diseases may be combined. Consequently a positive diagnosis is sometimes impossible.

Scarlatiniform erythema is one of the most puzzling conditions so far as diagnosis is concerned. This erythema may be due to sepsis, other infectious diseases, various medicaments, or to acute desquamative dermatitis. It is not infrequent in children with slight disturbance of digestion. Sepsis is the cause in diphtheria, and probably also in the majority of the cases called "surgical scarlet fever." The rash resembles that of scarlet fever in appearance, and may be followed by desquamation. The history of the case, the failure of development of the eruption in the usual sequence and extent, and the absence of the other symptoms of scarlatina aid in the diagnosis. Typhoid fever, grippe and varicella occasionally exhibit an erythema early in the attack and this may make the diagnosis at first obscure. The absence of the scarlatinal symptoms, such as the characteristic throat and tongue, the transitory nature of the rash, and the appearance later of the other manifestations characteristic of these different diseases remove the difficulty in diagnosis. The prodromal erythema of variola is distinguished by its peculiar localization. Various erythemata caused by drugs are at times perplexing. Among these are especially those due to quinine, chloral, salicylic acid compounds, belladonna, antipyrine, and animal sera, especially the diphtheria antitoxin. Local irritants such as mustard-plasters have often been the

[1] Jour. Amer. Med. Assoc., 1924, LXXXII, 712.

Jour. Amer. Med. Assoc., 1924, LXXXIII, 432.

source of confusion. The occurrence of the atropine eruption has especially been a cause of alarm to parents. In none of these are the other symptoms of scarlet fever present, except that occasionally the eruption may be accompanied by fever and vomiting. Quinine will sometimes produce a scarlatiniform eruption followed by very characteristic scarlatiniform desquamation. Acute desquamative dermatitis (recurrent exfoliating dermatitis) is a peculiar affection which may resemble scarlet fever very closely. Probably cases supposed to be second and third attacks of scarlet fever are really instances of the cutaneous disease. It is attended by fever, a wide-spread erythematous eruption, and often by desquamation which may even occur in the form of casts of the skin of the hands and feet, as in some cases of scarlet fever. In these respects it cannot be distinguished from scarlet fever. Pharyngeal symptoms are, however, absent. In all these conditions the extinction phenomenon is absent. (See p. 371.)

Treatment. (*A*) **Prophylaxis. Quarantine.**—Every case of suspected scarlet fever should be isolated at once, and kept so until all danger of the infection of others is over. The attempt to limit the spread of the disease offers much greater chance of success than in the case of some of the other infectious fevers. How long the quarantine should last is uncertain. At least 6 weeks should be the rule, and longer than this if desquamation or purulent discharge from the ears or nose is present. This is in view of the infectiousness which possibly inheres in contaminated scales and certainly in the discharges. The studies of a number of observers, however (Millard;[1] Lauder[2]), indicate that this time is unnecessarily long; and that 4 or 5 weeks insures as great safety as 6, provided there is no abnormal discharge from the nose or ears. Priestley[3] sent home from the hospitals 120 individuals still desquamating; with no development of the disease following in other members of the families. Gregory[4] suggests that removal of the tonsils may act in the nature of a prophylaxis against scarlet fever. The studies of Bullowa[5] certainly indicate that persons with buried tonsils who develop scarlet fever are more liable to cervical adenitis, and that this adenitis doubles the percentage of deaths. The general methods for preventing the spread of the disease, the selection of the room and care of the patient, and the management of other children in the family, are described under General Care of Infectious Diseases (p. 345). Preventive inoculation was attempted years ago by various physicians without satisfactory results. In more recent years similar efforts have been made and good results reported. Gabritschewsky[6] employed a form of preventive vaccination in about 50,000 children and claimed success. This has been confirmed by some observers, but found without value by others (Kolmer).[7] Di Cristina,[8] Caronia[9] and other Italian workers have employed a serovaccine which they claim has rendered children immune. Fifteen c.c. (0.51 fl. oz.) of convalescent serum are mixed with 10 gm. (.35 oz.) of desquamated scarlet fever scales; 0.8 per cent. of phenol is added, and 1 c.c. (.034 fl. oz.) of guinea-pig complement. This is incubated for 15 minutes at body-temperature, centrifuged, and sealed in vials. One-tenth of a c.c. (.003 fl. oz.) of this mixture is injected subcutaneously every 2nd day for 3 doses. Most encouraging results in prophylaxis with the injection of convalescent serum alone were reported by Degwitz,[1] all but 3 of 509 exposed persons failing to develop the disease after the treatment. The serum was prepared according to the method employed by him for the obtaining of convalescent serum for use in measles (see. p. 400). From 5 to 10 c.c. (.17 to .34 fl. oz.) were given subcutaneously as early as possible after exposure. The serum was obtained from the 4th to the 6th week of the disease. Neff[2] also reports good results with a dosage of from 15 to 30 c.c. (.51 to 1.01 fl. oz.), (see also p. 376). The treatment should be given in the first 4 days after exposure. Inasmuch as normal human serum has also appeared to possess immunizing power, it is questionable how much of the value of convalescent serum depends upon a non-specific protein-therapy. Certainly prophylaxis with convalescent serum may well be tried when practicable in outbreaks in institutions or in families. Abundant testimony shows that it is valuable, and that the attack will be avoided or at least rendered very mild. In our experience even the smaller dose of 5 to 10 c.c. (.17 to .34 fl. oz.) is protective. The protection given is, however, only temporary, lasting but a few weeks.

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[1] Lancet, 1902, I, 959.
[2] Lancet, 1904, I, 712.
[3] Transac. Epid. Soc., 1894–5, XIV, 71.
[4] Illinois Med. Jour., 1921, XL, 210.
[5] Amer. Jour. Dis. Child., 1921, XXII, 29.
[6] Ref. Wladimiroff, Arch. f. Kinderheilk., 1909, LII, 28.
[7] Arch. Int. Med. 1912, IX, 220.
[8] La Pediat., 1916, XXIV, 385.
[9] La Pediat., 1918, XXVI, 406.

The work of Dick and Dick[3] has shown that it is possible to create active immunization against scarlet fever by the injection of increasing doses of scarlet fever toxin. The immunity usually develops after one or two weeks. The injection consists of the amount of toxin corresponding to 500 skin-test-doses at the first injection, 1000 at the second, and 1500–5000 at the third. If at the end of a week the Dick test, presently to be described, is still positive 1 or 2 more injections should be given of the same strength as the third. In this way practically all individuals can be immunized, or at least can be made to give a negative Dick test. Other workers have employed smaller doses such as 100, 250 and 500 skin-test-doses. Under these conditions not all individuals will become immune. The immunity produced by these injections probably lasts for at least 2 or 3 years. It should be stated, however, that the reactions with this method of prophylactic treatment are often somewhat violent, especially in older individuals. It is probable that the degree of reaction in the Dick test is an indication of the amount of antitoxin possessed by the individual, a markedly positive reaction indicating that little neutralizing toxin is present. To patients responding in this way immunizing doses of toxin smaller than usual should be given (Lindsay, Rice and Selinger).[4] Larson, Huenekens and Colby[5] have been able to detoxify scarlet fever toxin with a solution of sodium ricinoleate. Intramuscular injection of from 3000 to 5000 skin-test-doses of this can be given with little or no subsequent reaction.

Immunizing by the Dick method has been extensively studied by Zingher[6] with very encouraging results. The immunization produced in this way is of an active nature; that produced by scarlatinal antitoxin is merely passive and of short duration.

[1] Münch. med. Wochenschr., 1922, LXIX, 955.

[2] Arch. of Pediat., 1922, XXXIX, 250.

[3] Jour. Amer. Med. Assoc. 1924, LXXXII, 265; 544; 1924, LXXXIII, 84; 1925, LXXXIV, 1481; Amer. Jour. Dis. Child., 1925, XXVIII, 484.

[4] Jour. Amer. Med. Assoc., 1926, LXXXVI, 1191.

[5] Jour. Amer. Med. Assoc., 1926, LXXXVI, 1000.

[6] Jour. Amer. Med. Assoc., 1924, LXXXIII, 432.

To determine whether a patient is immune to the disease, and consequently does not need prophylactic treatment, Dick and Dick recommend the employment of a preliminary test. This consists in the intradermal injection of 0.1 c.c. (.003 fl. oz.) of a 1:1000 dilution of the filtrate obtained as described. In individuals who have never had scarlet fever a distinct local reaction appears, which consists of a bright-red swelling about 3 cm. (1.18 in.) or less in diameter with raised edges. This develops at the end of 24 hours. In 125 exposed persons the cutaneous test was positive in 52, and these were then immunized. None of the 125 developed scarlet fever. The reliability of this test has been confirmed by many studies.

(*B*) **Treatment of the Attack.**—The treatment, other than the specific, is purely symptomatic. The patient should be kept absolutely in bed until desquamation is over. The confinement to bed should continue for at least 3 weeks even in the mild cases. The diet should if possible be liquid or soft until the temperature is normal, milk and amylaceous foods being the best, reserving eggs, meat and green vegetables until the end of at least the 3d week of the disease. This plan of treatment is believed to throw less work upon the kidneys. The injuriousness of meat in this disease has been, however, disputed. Gerstley[1] in a trial upon 306 cases, half of them given the ordinary full diet, could see no difference in the incidence of nephritis. These results confirm the observations of Pospischill and Weiss,[2] but are opposed to the opinion and practice of perhaps the majority of pediatrists. Water to drink should be administered freely. Daily warm ablution should be given to every child for the sake of cleanliness and comfort, with proper precautions against exposure, washing being done under the bed-clothes, or with the uncovering of but one portion of the body at a time.

The room should not be hot, and fresh air is an essential, but draughts must be carefully avoided during the apyretic period of convalescence. Much has been written of the innocuousness of exposure to draughts of air in this disease. While it is undoubtedly true that infection is the active cause in the production of complications, there seems no good reason why surface-chilling may not act as a predisposing cause, and why no precaution should be taken against it. The bed-covering should be light, the sensations of the patient, when these can be ascertained, being the best guide. The skin should be kept oiled with petrolatum or a weak carbolized oil (2 per cent.) or, in the case of young children, with a 1 per cent. thymol ointment. This allays the itching and prevents also the dissemination of the scales, at the same time disinfecting them, if they have become soiled by mucus or purulent secretion, and favors a more rapid desquamation. The mouth, throat, and nose should be examined daily in order that appropriate treatment may be commenced at once if indicated. (See p. 335.) The employment of mild antiseptic gargles and sprays aids in preventing the development of any serious trouble here. The urine also should be examined frequently.

Little internal medication is needed. Stimulation is not required in ordinary cases. A mild diuretic, such as citrate of potash, may be of advantage in maintaining diuresis without irritation of the kidneys. Various drugs have at different times been recommended as specifics. None of these have proven of any certain value.

Much has been written regarding the *serum-treatment* of scarlet fever, but until recent years without much that was encouraging. Marmorek's[3] antistreptococcic serum was early tried, but did not become established. Later a serum was advocated by Aronson,[1] and then a somewhat similar one by Moser;[2] the latter being a polyvalent serum from horses which had been inoculated with cultures from the blood of scarlet fever patients. Excellent results have been claimed for this by many physicians, while others failed entirely to obtain them. The dose was large and the serum-reaction often severe. (For a careful review see article of Fedinski.[3]) More recently Dochez and Sherman[4] have produced an antiscarlatinal antistreptococcic serum by the immunization of horses with a specific type of hemolytic streptococcus isolated from human cases of the disease. The curative properties of this serum have been attested by Blake, Trask and Lynch[5] and others. A single intramuscular injection of 40 to 60 c.c. (1.35 to 2.03 fl. oz.) resulted in a critical fall of temperature within 12 hours, a complete fading of the rash in 24 hours, rapid improvement of the angina, and disappearance of toxic symptoms. The earlier the serum is given the greater is the likelihood of doing good and of preventing septic manifestations. A marked serum-reaction with cutaneous manifestation almost invariably follows the use of the serum. The antitoxic serum has since been concentrated, thus producing fewer general reactions.

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[1] Monatsschr. f. Kinderheilk., Orig., 1913, XII, 121.

[2] Ueber Scharlach, 1911. Ref. Gerstley, *loc. cit.*

[3] Ann. de l'inst. Pasteur, 1896, X, 47.

Treatment by the injection of serum from those recovering from scarlet fever has offered much promise. It appears to have been first used by Weisbecker[6] and since then by numerous others with decided benefit. The subject has been reviewed by Weaver.[7]

In the preparation, blood is obtained from a patient from the 2nd up to the 4th week or even the 7th week of the disease, the serum separated from the clot and mixed with serum from several other patients and with 0.3 per cent. of tricresol, tested for sterility, and preserved on ice in small bottles. It should be injected intramuscularly within a few weeks after it is obtained in doses of from 10 to 60 c.c. (0.34 to 2.03 fl. oz.) or much larger amounts, and the injection repeated once or twice in severe cases. It should be administered if possible before the 4th or 5th day of the disease. Inasmuch as the reaction is sometimes severe, it is better to reserve the treatment for the graver cases. Since improvement in symptoms has also been reported following the injection of normal human serum, the question arises as to what portion of the benefit from convalescent serum depends upon the introduction of the protein. It seems likely, however, that the only effective serum is that from normal individuals who have scarlet fever immune-substances.

Dick and Dick[8] have prepared a scarlet fever antitoxin by immunizing horses with subcutaneous injections of sterile filtrate from broth-cultures of the strains of streptococci that had produced experimental scarlet fever. Later studies[9] have convinced them that it is very efficacious if administered early, before complications have developed.

Young and Birkhaug[10] report cure by the intravenous injection of mercurochrome, using a 1 per cent. solution and injecting 7.5 mg. of the drug per kilogram of body weight (0.05 grain per pound).

[1] Verhandl. Berl. med. Ges., 1902, XXXIII, 253.
[2] Jahrb. f. Kinderheilk., 1903, LVII, 1.
[3] Jahrb. f. Kinderheilk., 1910, LXXI, 189.
[4] Jour. Amer. Med. Assoc., 1924, LXXXII, 542.
[5] Jour. Amer. Med. Assoc., 1924, LXXXII, 712.
[6] Zeitschr. f. klin. Med., 1897, XXXII, 188.
[7] Jour. Amer. Med. Assoc., 1921, LXXVII, 1420.
[8] Jour. Amer. Med. Assoc., 1924, LXXXII, 1246.
[9] Jour. Amer. Med. Assoc., 1925, LXXXIV, 803.; LXXXV, 1693.
[10] Jour. Amer. Med. Assoc., 1924, LXXXIII, 402.

The treatment of some of the more important symptoms and complications may be considered separately:

Fever.—A temperature of 104°F. (40°C.) lasting for a short time is generally a matter of no special consequence. When, however, high fever is prolonged and is accompanied by nervous symptoms, treatment is required. Hydrotherapeutic measures are generally much to be preferred to internal medication. Sponging with luke-warm water or with alcohol and water may be employed. If this is not sufficient, sponging with water at a temperature of 70° to 80°F. (21.1° to 26.7°C.) may be tried, or in urgent cases cooler than this. Great discretion must be exercised, however, in young children and especially in infants, since these often do not bear cold water well in any febrile disorder. Every case must be treated as an individual and all depression of pulse-strength assiduously avoided. Very often submersion in a tepid bath of 85° to 90°F. (29.4° to 32.2°C.) or even in a warm bath of 90° to 100°F. (32.2° to 37.8°C.) will effect decided reduction of temperature. In very urgent cases the graduated bath may be employed, the temperature of the water after the child is in the tub being gradually reduced to even 70°F. (21.1°C.); rarely lower than this. The warm or cold pack frequently repeated is often very useful and better tolerated than the bath. Hydrotherapy must always be used with especial caution, if at all, where decided cardiac weakness exists. When, too, good reaction does not take place, or when a young child fights violently against the treatment, it may sometimes do more harm than good.

The employment of antipyretic drugs of the coal-tar series for the reduction of temperature is only exceptionally indicated. There are times, however, when they are very serviceable, as when for any reason hydrotherapy cannot be employed. They should be given in small doses, frequently repeated if necessary.

Nervous symptoms attending high temperature are often much benefited by hydrotherapy. Indeed, it is the combating of these, rather than the simple reduction of temperature, which is the chief aim of hydrotherapeutic measures. An ice-cap to the head is frequently of advantage under these circumstances. The coal-tar derivatives employed to combat nervous symptoms have a value much greater than that of the mere reduction of fever which they effect. Their administration is often followed by the relief of delirium, stupor, jactitation or a convulsive condition, which is greater than can be attributed to the fall of temperature. My own preference is for phenacetin or antipyrine. It is important to give repeated small doses rather than a single larger one, since a decided fall of temperature may be attended by prostration. In the majority of instances hydrotherapy is to be preferred. For the convulsions developing during scarlatinal nephritis both Allaria[1] and Sheffield[2] have seen benefit from the employment of lumbar puncture.

Cardiac weakness, as shown by the rapid and feeble or irregular pulse, weak heart-sounds, or persistent coldness and cyanosis of the extremities, demands the prompt and energetic use of stimulants. Even before these symptoms have actually made their appearance, but where, as in septic cases or those with prolonged high temperature, it is feared that weakness of the heart may develop, stimulants may well be employed. Digitalis, strychnine and alcohol by the mouth, or camphor and caffeine given hypodermically, may be selected according to the demands of the case.

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[1] Gaz. degli. osp. e del. clin., 1911, XXXII, 1291.

[2] Pediatrics, 1912, XXIV, 99.

Inflammation of the throat and nose when mild requires no special medication. When more severe, as in anginose cases, it often demands active treatment, the nature and frequency of which varies with the case. The nose may be sprayed or syringed gently with normal salt solution or with mild antiseptic solutions (boric acid, liquor antisepticus alkalinus, etc.) repeated several times a day. Antiseptic gargles should be employed, or, in case the child cannot or will not use these, swabbing of the throat with cleansing and antiseptic solutions as for pharyngitis and tonsillitis of other nature. Diluted peroxide of hydrogen is often to be recommended, or solutions of potassium permanganate or of corrosive sublimate. When a pseudodiphtheritic membrane has formed application of diluted peroxide of hydrogen (1:2 of water) may be used, followed by diluted tincture of the chloride of iron (1:4) or of bichloride of mercury (1:5000), or nitrate of silver (gr. 5 or 10:oz. 1) (0.324 or 0.648:30). When local treatment is attended by great resistance on the part of the child, as is often the case, the advisability of continuing it is questionable. Its importance must be determined for each individual.

Otitis can probably be prevented to some extent by persistent disinfection of the throat and nose. The constant wearing of a flannel cap over the ears, the instillation of a few drops of a 10 per cent. carbolized glycerin, or the use of a hot water bag or of hot water douching of the canal, is of benefit as a preventive or if the disease has developed. In the latter event paracentesis may become necessary. If perforation of the drum-head has taken place the usual treatment of suppurating otitis media is indicated. (See Vol. II, p. 925.)

Adenitis may often be aborted by the application of an ice-bag, tincture of iodine, flexible collodion, or a 15 per cent. ointment of ichthyol. The ice-bag should be of thin rubber and kept constantly in position, with a thin layer of cloth between it and the skin. If it is evident that suppuration will take place hot compresses or poultices may be employed to hasten the process and to relieve the pain. Early evacuation of the pus is indicated.

Nephritis requires prompt treatment. Whether anything can be done to prevent its development is a much disputed question. The avoidance of surface-chilling and the employment of an unirritating diet have already been mentioned. A salt-free diet has been urged by Pater,[1] Delearde[2] and others, but Nobécourt and Merklen[3] found albuminuria less frequent when milk alone was employed. Widowitz,[4] Buttersack[5] and others have strongly recommended the administration of hexamethylenamine as a preventive measure, and Royer[6] confirmed previously published reports upon the value of chloral given for the same purpose.

When nephritis has developed, if of but slight severity, the frequently repeated application of hot poultices over the renal region, the use of warm baths, and the administration of mild diuretics, such as acetate of potash, citrate of potash and sometimes of digitalis, are sufficient. Whether the potash salts are of any benefit is, however, doubtful, and it is even possible that they may be irritant. Stimulating diuretics must never be employed. The patient must be kept strictly in bed. When the nephritis is more severe, with anuria or great diminution of the amount of urine, convulsions, vomiting, and the symptoms of uremia, the treatment must be energetic. This is discussed more fully in the chapter upon Acute Nephritis, Vol. II, p. 448.

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[1] Bull. et. mém. soc. méd. des hôp., 1906, XXIII, 93.

[2] L'Echo méd. du nord., 1907, XI, 25.

[3] Arch. de méd. des enf., 1908, XI, 81.

[4] Wien. klin. Wochenschr., 1903, XVI, 1113.

[5] Deutsch. Arch. klin. Med., 1904, LXXX, 356.

[6] Penna. Med. Jour., 1907, X, 286.

Arthritis may well be treated with salicylic acid combinations internally or externally on the ground that it may be rheumatic in nature; and in any event because full doses may relieve the pain. If it is but slight, local protection of the joints may be all that is required. Purulent inflammation of the joints is to be subjected to surgical measures.

The treatment of other complications and sequels does not require special consideration here. During convalescence, especially from a severe attack, tonic measures are indicated, including the administration of iron to combat the anemia which often results.

CHAPTER III

MEASLES

(Rubeola. Morbilli)

Measles was undoubtedly known to the ancients, but was confounded by them with several other diseases. It was differentiated clearly from small-pox by Rhazes[1] about 900 A. D. and from scarlet fever by Sydenham[2] in the middle of the 17th Century.

Etiology. Predisposing Causes.—Race, sex, climate and locality, social position, and sanitation appear to exert no influence on the occurrence of the disease. The presence of other acute disorders occasionally delays the appearance of the symptoms of measles, but does not prevent it. *Age* is a very important factor. The degree of susceptibility in the latter half of the 1st year is disputed, but it is agreed that infants in the first 6 months are much less often attacked than later. Herrman's[3] experience in a large number of cases confirms this. Infants in the first 2 months of life whose mothers had had measles were absolutely immune, and all under 5 months relatively so; but after 9 months the susceptibility was as great as in older children. Yet even instances of congenital measles are recorded, Ballantyne[4] having collected 21 such from medical literature. Additional cases of the presence of measles at or developing a few days after birth have been reported by Mason,[5] Steinschneider,[6] Rocaz,[7] Schulze[8] and others. We have seen 3 undoubted instances of measles which developed in the first few days of life, the mother suffering from the disease during the last week of the puerperium.

Whether the infrequency in nurslings is due to lesser susceptibility, or to lesser frequency of exposure is not certainly determined. Herrman's

[1] De variolis et morbillis, 1756.
[2] Processus integri. Ref. Williams, 20th Cent. Pract. Med., 1898, XIV, 117.
[3] Arch. of Pediat., 1923, XL, 678.
[4] Arch. of Pediat., 1893, X, 301.
[5] Bost. Med. and Surg. Journ., 1908, CLIX, 436.
[6] Deutsch. med. Wochenschr., 1914, XL, 441.
[7] Gaz. hebdom. des sci. méd. de Bordeaux, 1906, XXVII, 260.
[8] Deutsch. med. Wochenschr., 1921, XLVII, 271.

experience showed no evidence that any important part of the immunity is due to the ingestion of breast-milk. (See p. 97.) Lichtenstein[1] observed 40 nurslings attacked out of 47 exposed to infection, this appearing to be contrary to the usual experience. That few exposed adults suffer from the disease clearly depends on the fact that so many have already had it. In the epidemic of 1846 in the Faroe Islands, described by Panum,[2] where no one was thus protected, adults of every age proved as susceptible as children, and no instance was met with where an unprotected adult exposed did not take the disease.

The *individual susceptibility* is very great, exceeding that of most of the other infectious diseases. In the Faroe Islands' epidemic over 6000 of 7782 inhabitants were attacked, 1500 escaping only through the institution of absolute quarantine in certain villages. Later, in the epidemic of 1875, as described by Hoff,[3] 99 per cent. of the unprotected inhabitants of Thorshaven was affected. In my own experience measles is one of the most readily contracted of the diseases of its class, the susceptibility to it being so great that spread of the infection is little controllable. Yet a certain individual immunity, it is true, temporary or permanent, is occasionally met with. A decided *epidemic influence* exists also, measles being much more frequent or severe in certain years than in others, especially in localities where it is always endemic to some extent. This may depend upon a greater virulence of the poison in some years, and a consequent greater tendency to spread. There is no regularity in the return of these epidemic outbreaks. *Season* seems to possess a slight influence, the disease being more prevalent in winter, and especially in spring, than in summer. Of 530 epidemics studied by Hirsch[4] 339 (64 per cent.) occurred in the colder part of the year, and 191 (36 per cent.) in the warmer. In 213 of the epidemics the height was reached in 76 in spring, 59 in winter, 48 in autumn and 30 in summer. Yet the greater prevalence in the cooler months may depend upon the closer confinement and consequent more intimate association in houses and especially in schools.

Exciting Cause.—This is without doubt a germ of some sort. Canon and Pielicke[5] and Czajkowski[6] described a short bacillus constantly present in the blood and the nasal secretion. Bacilli have also been found in the blood by Arsamakov,[7] Zlatogoroff,[8] Von Niessen,[9] Giarre and Picchi[10] and others. Micrococci have been reported in the blood and the nasal mucus by Lesage[11] and in the blood, throat, nose, and eyes by Tunnicliff.[12] Tunnicliff and Moody[13] have described a green-producing streptococcus which they have obtained from the nose, throat and blood of patients with the disease. In a later study by Tunnicliff[1] it was found that a culture of this germ produced a subcutaneous reaction in individuals who had not had measles. Ferry and Fisher[2] have likewise obtained in susceptible persons positive cutaneous tests with the toxin of the green-producing streptococcus. Caronia[3] observed a small diplococcus which he believes from inoculation-experiments to be the cause of measles, and Sellards and Bigelow[4] a small pleomorphic Gram-staining bacillus. An ameba-like body was found in the red corpuscles by Doehle,[5] Behla,[6] and later by Rosenberger.[7] The true nature of the microörganism is evidently not yet entirely determined. It seems certain that, under the influence of its presence, other germs, such as the hemolytic streptococcus, pneumococcus, the bacillus of influenza and that of diphtheria, are enabled to produce complications.

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[1] Hygiea, 1914, LXXVI, 1022. Ref. Monatsschr. f. Kinderheilk. Ref., 1915, XIV, 455.

[2] Verhandl d. physic.-med. Gesellsch. in Würzburg, 1851, II, 292, 293; Virchow's Archiv. 1847, 1, 492.

[3] Sundhedskollegiets Aarsberetning 1876. Ref. von Jürgensen, Nothnagel's Encyclop. Amer. Ed., Measles, 228.

[4] Handb. d. histor. geog. Path. 1881, 116.

[5] Berl. klin. Wochenschr., 1892, XXIX, 377.

[6] Centralbl. f. Bakt., 1895, XVIII, 517.

[7] Bolnitch Gaz. Botk. 1898, 40. Ref. Rev. de méd., 1899, XIX, 561.

[8] Centralbl. f. Bakt. 1904, XXXVII, 249.

[9] Arch. f. Derm. u. Syph., 1902, LX, 429.

[10] Monatsschr. f. Kinderheilk., 1902, I, 577.

[11] Compt. rend. de la soc. de biol., 1900, LII, 203

[12] Jour. Amer. Med. Assoc., 1917, LXVIII, 1028.

[13] Jour. Infect. Dis., 1922, XXXI, 382.

In this connection the various experiments upon the possibility of the transmission of the disease to other animals are of interest and importance. Goldberger and Anderson[8] succeeded in producing the disease in monkeys by inoculating with virus obtained from the secretions and the blood. As this was accomplished after passing the material through a Berkfeld filter, the etiological relationship of any of the microörganisms previously reported would appear to be disproved, and the germ must be ultramicroscopic. The transmissibility to monkeys has been confirmed by Hektoen and Eggers[9] and by Lucas and Prizer.[10] Recent experimental work has been carried out in two ways: By intravenous injections into monkeys, rabbits and guinea-pigs of blood from human cases of measles a specific reaction resembling measles has been developed in them (Nevin and Bittman;[11] Duval and D'Aunoy[12]). Second, by intratracheal injections into monkeys, rabbits and guinea-pigs of filtered or unfiltered nasopharyngeal washings from cases of measles symptoms resembling the disease have been produced (Blake and Trask;[13] Duval and D'Aunoy;[14] Tunnicliff and Moody;[15] Grund[16]), and in some cases it was possible to transmit the disease from monkey to monkey. Sellards[17] who has critically reviewed the literature on the experimental investigations believes that, while occasionally individual monkeys may develop a mild reaction when inoculated with the virus of measles, this does not consist in symptoms which are pathognomonic of measles, and that there is no convincing proof of the susceptibility of these animals to the disease. This is in accord with the earlier review by Hektoen.[18] It is evident that although transmission in some degree to other animals seems very probably to exist, additional investigations are required.

[1] Jour. Infect. Dis,. 1925, XXXVII, 193.
[2] Jour. Amer. Med. Assoc., 1926, LXXXVI, 932.
[3] La Pediat., 1923, XXXI, 801.
[4] Jour. Med. Res., 1921, XLI, 241.
[5] Centralbl. f. allg. Path. u. path. Anat., 1892, III, 150.
[6] Centralbl. f. Bakt., 1896, XX, 561.
[7] Amer. Med., 1906, XII, 139.
[8] Jour. Amer. Med. Assoc., 1911, LVII, 476; 971; 1612.
[9] Jour. Amer. Med. Assoc., 1911, LVII, 1833.
[10] Jour. Med. Research, 1912, XXVI, 181.
[11] Jour. Infect. Dis., 1921, XXIX, 429.
[12] Jour. Exper. Med., 1922, XXXV, 257.
[13] Jour Exper. Med., 1921, XXXIII, 385; 413.
[14] Jour. Exper. Med., 1922, XXXVI, 231.
[15] *Loc. cit.*
[16] Jour. Infect. Dis., 1922, XXX, 86.
[17] Medicine, 1924, III, 99.
[18] Jour. Amer. Med. Assoc., 1919, LXXII, 177.

The germ is very short-lived in measles as compared with scarlet fever and small-pox. Positive data are necessarily lacking, but it is probable that none are living from 10 to 14 days or even less from the onset of the attack. Its habitat is certainly the secretions of the mucous membrane of the nose, mouth, and eyes, and the blood; and it is probable that the desquamating epidermis has no infectious power unless contaminated by these. It was found without infectivity by Anderson and Goldberger. In our experience a persisting discharge from an otitis media developing during the attack of measles does not continue to contain infectious material and to be a source of infection after 2 or 3 weeks at the most. We know of no definite studies made with regard to this. Inoculation experiments upon human beings, claimed to have been successful, were made with the blood by Home[1] and by Hektoen,[2] with the blood mixed with serum from the vesicles by Katona,[3] and with the nasal mucus by Mayr.[4] Some investigators have confirmed these results, while others have failed. Bauguess[5] accidentally produced measles in an infant by transfusion with the blood from the mother, who developed the disease shortly afterwards. Sellards[6] inoculated a number of men with blood from patients with measles with entire lack of success. There seems no reasonable doubt, however, that measles has been experimentally transmitted to man in exceptional cases.

Method of Transmission.—This may be either mediate or direct, the latter occurring in the vast majority of instances. The germ is certainly very virulent, and but a brief exposure is necessary. The disease can be communicated by way of the air through the mere presence in the sick-room, but that it can be carried any great distance by it is entirely unlikely. The experiments of Grancher[7] and others indicate that it cannot. Hoyne[8] claims, however, that cases have occurred in hospitals which can be explained only by the germs being carried by a draught of air from one end of a ward to another, or up a ventilating shaft, or even passing out of a window on one floor and entering one on a floor above. Similar experiences have been reported by others (Moro;[9] Jasinski and Progulski[10]). Many instances of transmission by fomites or by a third person are on record, but are certainly exceptional. The careful analysis made by von Jürgensen[11] throws doubt upon some of those oftenest quoted. Some more recent experiences in favor of mediate transmission have been published by Roch,[12] Rohmer,[13] Lanzarini[14] and others. The common experience, however, agrees with that of von Jürgensen. The usual absence of mediate transmission may be explained in part by the germ's short duration of life.

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[1] Medical Facts and Experiments, 1759.

[2] Jour. Infect. Dis., 1905, II, 238.

[3] Oesterreich. med. Wochenschr., 1842, 697.

[4] Zeitschr. d. k. k. Gesellsch. d. Aerzte zu Wien., 1852, VIII, 13.

[5] Amer. Jour. Dis. Child., 1924, XXVII, 256.

[6] Johns Hopk. Hosp. Bull., 1919, XXX, 257.

[7] Traité des mal. de l'enf., 2 Ed., I, 322.

[8] Arch. of Pediat., 1920, XXXVII, 606.

[9] Monatsschr. f. Kinderheilk., Orig., 1916, XIV, 4.

[10] Pedj. Polska, 1, 1922. Ref. Groer and Pirquet in Pfaundler and Schlossmann, Handb. der Kinderheilk., 1923, II, 187.

[11] Nothnagel's Encyclop. Amer. Edit., Measles, 247

[12] Arch. de méd. des enf., 1907, X, 292.

[13] Jahrb. f. Kinderheilk., 1912, LXXV, 78.

[14] La Pediat., 1907, XV, 366.

Yet it is evident that the unexpected development of measles in a child who has for some months been in a hospital-ward requires the explanation, either that the germ is carried by the air for long distances, that transmission by fomites or in some way by a third person may perhaps occur more often than we have reason to believe, or that it is very possible for some child recently admitted to be suffering from measles in an abortive and undiscoverable manner, and to be, in fact, the starting case of the outbreak. It is even possible that these abortive attacks may occur in individuals who have previously suffered from measles (Wagener).[1] The extension from one room of a hospital to another with an entirely different nursing staff makes the explanation of transmission by air the most likely one in such instances.

The germs probably enter the system by way of the respiratory tract. In the rare cases in the new-born placental transmission has taken place.

Period of Infectiousness.—It is probable that the danger exists during the last part of the stage of incubation, and certainly so from the onset of the invasion and during at least a portion of the period of eruption. The great frequency with which the disease is disseminated by schools and the difficulty of controlling its spread, proves the infectiousness of the early periods. The infectiousness lessens rapidly as the catarrhal symptoms disappear. Certainly the disease is communicated in but few instances during the stage of desquamation. Rooms which have been occupied by patients with measles can be used with safety by susceptible subjects very soon after the attack is over, according to some investigators within a few hours.

Pathological Anatomy.—There are few characteristic post-mortem lesions. According to Neumann[2] the principal changes in the skin consist of dilatation of the vessels, with a surrounding infiltration of small cells, situated in the upper portion of the cutis and extending into the Malpighian layer, together with an extensive infiltration around the hair-follicles and the sudoriparous and sebaceous glands. Similar changes are seen in the mucous membrane, a catarrhal inflammation with a small-celled infiltration occurring in the eyes and in the respiratory and alimentary tracts. The histology of the buccal eruption, as studied by Hlava[3] and by Flamini[4] consists in an interpapillary injection of the capillary vessels, and infiltration with round cells, thickening of the superficial layer of the epidermis and degenerative changes of the lower layer, forming a minute pustule.

The inflammatory process is followed by desquamation of the epithelium of both the skin and the mucous membrane. The lymphoid tissues throughout the body, including the lymphatic glands everywhere, the tonsils, spleen and the lymphatic follicles of the intestinal tract, exhibit decided cellular hyperplasia. Focal necrosis of the liver has been described (Freeman).[5] Denton[6] published careful histological studies on fatal cases of measles.

Symptoms.—ORDINARY TYPE. **Period of Incubation.**—This period, up to the first appearance of catarrhal symptoms, has been very accurately determined by Panum,[1] Lewy[2] and numerous other observers to be 10 or 11 days, or 14 days until the rash appears. Exceptionally it may be of slightly longer or shorter duration. In the majority of cases no symptoms occur. Meunier[3], however, insists that incubation is constantly attended by a very decided loss of weight beginning on the 4th or 5th day. Heubner,[4] Widowitz[5] and some others have occasionally seen a brief febrile reaction, sometimes with mild catarrhal symptoms, appearing very early in this period. The buccal eruption, presently to be described, is sometimes discovered before the catarrhal symptoms of the stage of invasion begin. Rolleston[6] maintained that ephemeral prodromal eruptions, such as urticaria or erythema, appear during the last part of the incubative period in nearly ½ of the cases studied. The characteristic changes of the blood (p. 389) begin to show themselves during the incubative period.

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[1] Monatsschr. f. Kinderheilk., Orig., 1916, XIII, 477.

[2] Med. Jahrb., 1882, 159.

[3] Časopis lékařů českých, 1906, 773. Ref. Centralbl. f. inn. Med., 1906, XXVII, 923.

[4] Riv. di clin. pediat., 1908, VI, 401.

[5] Arch. of Pediat., 1900, XVII, 81.

[6] Amer. Jour. Med. Sci., 1925, CLXIX, 531.

Period of Invasion.—Occasionally a convulsion or decided chilliness ushers in the attack, or fever develops suddenly; but as a rule the onset is gradual, the symptoms being indistinguishable from those of a severe general cold. The child is irritable, tired, chilly, and often peculiarly drowsy. As the disease progresses there are decided lachrymation, photophobia, redness of the conjunctiva, coryza, sneezing, thirst, and often a dry, hard, and sometimes distressing cough. Occasionally a few dry râles are audible in the chest. Epistaxis is not uncommon. The upper lip is excoriated by the nasal secretion, the appetite diminished, and the tongue coated, with the edges perhaps somewhat red and the papillæ here a little enlarged. Some soreness of the throat is complained of, and the pharynx, fauces and tonsils are moderately congested, the latter being slightly swollen. Vomiting occasionally occurs, and diarrhea is not infrequent. Headache is common and delirium may occur.

By the 2d or 3d day the characteristic eruption may be seen on the mucous membrane (enanthem), consisting of small, red macules dotted over the hard and the soft palate and resembling those which appear later upon the skin It is at its height when the cutaneous rash develops and may persist 3 or 4 days more. A condition of another sort, the so-called "buccal eruption" or "Koplik's spots," is found upon the mucous lining of the lips and cheeks, oftenest close to the junction of the latter with the upper jaw. It consists of minute bluish-white points each surrounded by a small slightly red areola. When the spots are numerous the areolæ coalesce, giving a redder tint to the whole lining of the cheeks and lips, over which are dotted the crowded spots.

Although the buccal eruption was previously described by Rinecker and by Reubold,[7] Flindt,[8] Monti,[9] Filatov,[10] and others, its diagnostic importance did not receive general attention until pointed out by Koplik[11] (Fig. 63). It can be seen to advantage only in bright daylight, preferably by carrying the child close to the window. Occasionally it may be

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[1] *Loc. cit.*
[2] Zeitschr. f. Kinderheilk., 1920, XXVI, 160.
[3] Gaz. hebdom., 1898, 1057.
[4] Lehrb. d. Kinderheilk., 1911, I, 285.
[5] Arch. f. Kinderheilk., 1923, LXXVII, 274.
[6] Brit. Med. Jour., 1905, Feb. 4, 233.
[7] Virchow's Archiv., 1854, VII, 76.
[8] Ref. v. Jürgensen, Nothnagel's Encyclop., Amer. Ed., 1902, 286.
[9] Jahrb. f. Kinderheilk., 1873, VI, 20.
[10] Dis. of Child. Amer. Transl., 1904, I, 97; II, 660.
[11] Arch. of Pediat., 1896, XIII, 918; Med. News, 1899, LXXIV, 673.

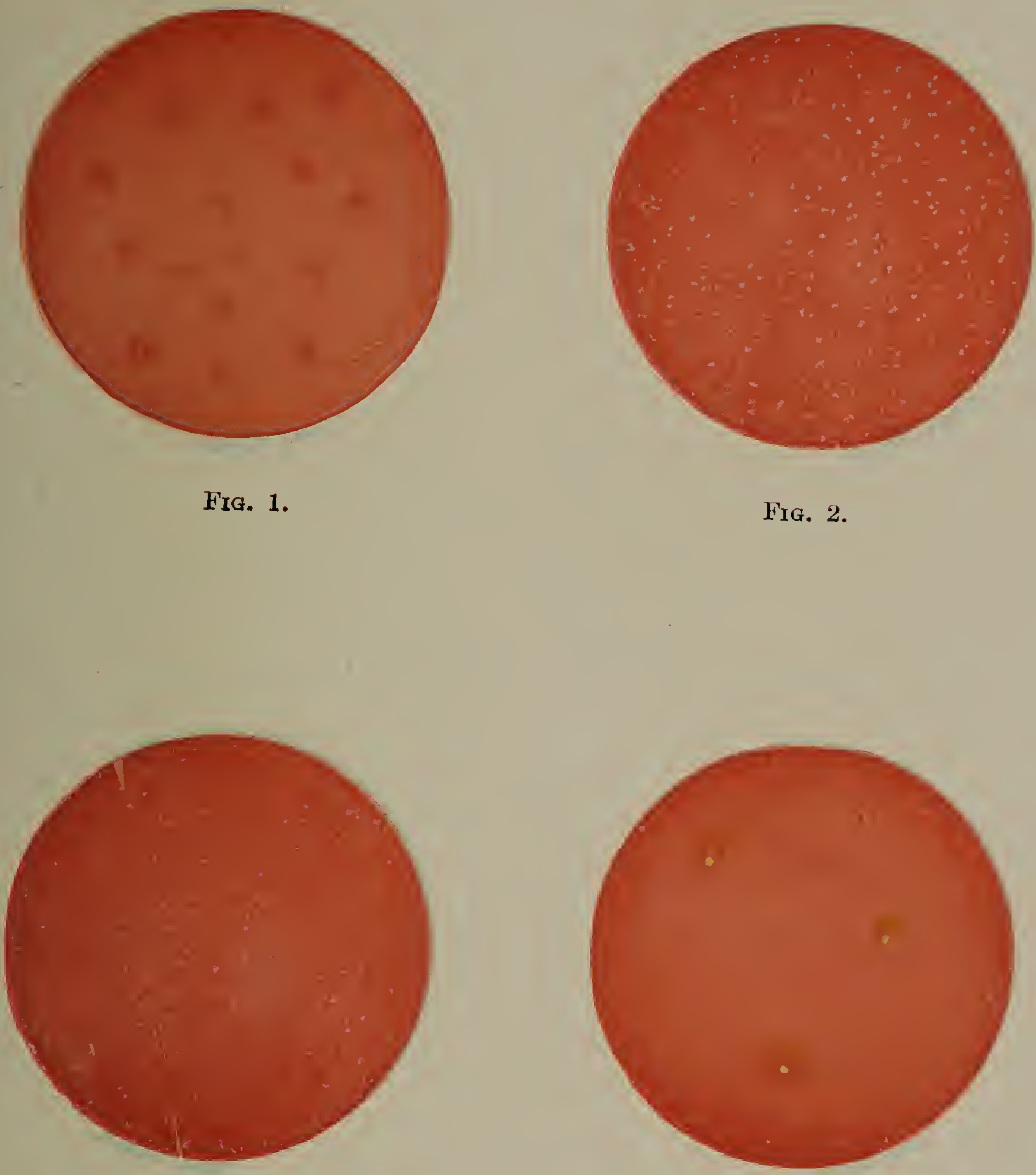

Fig. 1. Fig. 2.

Fig. 3. Fig. 4.

Fig. 63.—The Pathognomonic Sign of Measles (Koplik's Spots).

Fig. 1.—The discrete measles-spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

Fig. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale-pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

Fig. 3.—The appearance of the buccal or labial mucous membrane when the measles-spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthema on the skin is at this time generally fully developed.

Fig. 4.—Aphthous stomatitis apt to be mistaken for measles-spots. Mucous membrane normal in hue. Minute *yellow points* are surrounded by a red area. Always discrete.

(*Medical News*, 1899, lxxiv, 673.)

found as much as 5 days before the cutaneous rash develops (Cotter)[1] but is oftener seen 2 or 3 days before, and quite frequently is not discoverable until after this appears. The buccal eruption is almost pathognomonic of measles, and is present in from 80 to 90 per cent. of the cases. In the remaining, repeated, careful inspection will fail to reveal it. It disappears by the time, or even before, the cutaneous eruption is fully developed.

Prodromal cutaneous eruptions are not infrequently observed in this stage, if not seen earlier. Thus from a number of hours up to a day or more before the characteristic eruption is distinctly visible, there can sometimes be observed an alteration in the appearance of the skin of the face, suggesting a roughness, reddening or mottling situated, as it were, *beneath* the surface. In other cases there is a distinct localized blotchy erythema or an urticaria, or a more or less widely diffused scarlatiniform rash, or even a faint eruption which suggests the beginning of the ordinary rash. These prodromal eruptions are, in my experience, common. They usually disappear before the true exanthem of the disease becomes manifest.

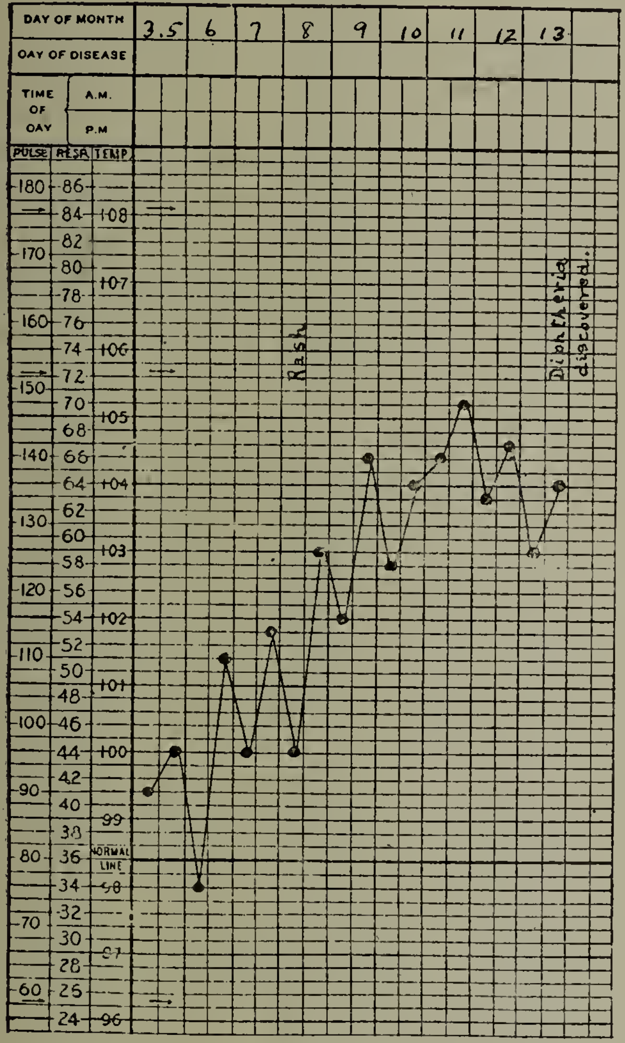

Fig. 64.

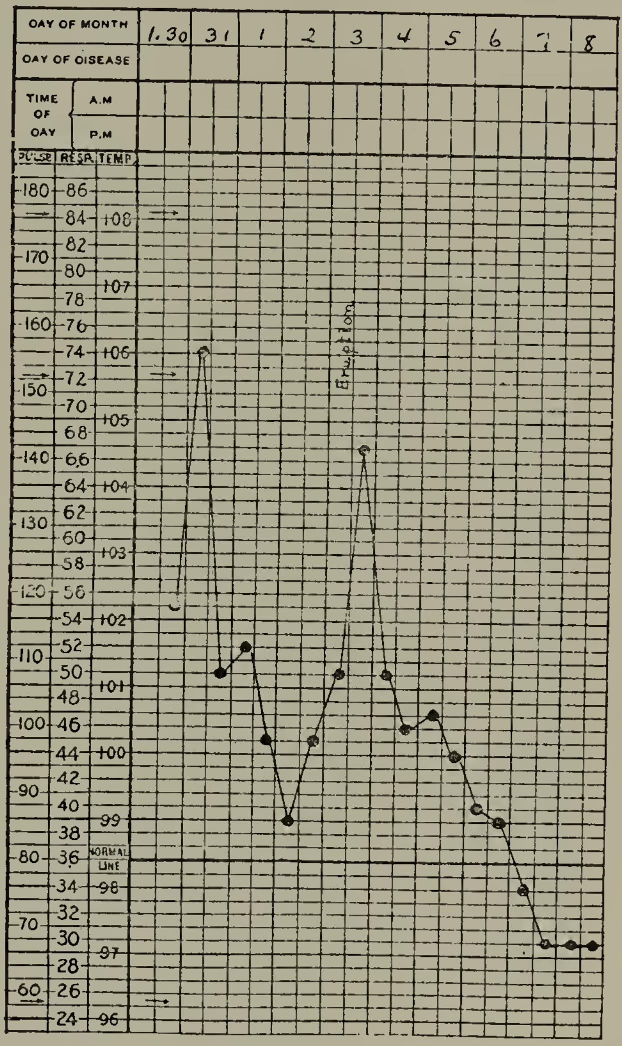

Fig. 65.

Fig. 64.—Measles without Pre-eruptive Fall of Temperature.

R. McN., 4 years old. Mar. 6, vomited; Mar. 8, well-marked catarrhal symptoms, buccal eruption, rash appearing on face and body; Mar. 10, attack severe; Mar. 13, diphtheria discovered.

Fig. 65.—Measles with Pre-eruptive Intermission in Temperature.

Lena M. Jan. 30, catarrhal symptoms began; Feb. 2, symptoms been very pronounced, fall of temperature to 99°; Feb. 3, abundant eruption, general symptoms improved, but temperature elevated; Feb. 8, desquamation.

All the catarrhal symptoms are well-marked by the 2d day of invasion, and increase steadily in severity through this stage. The temperature-curve during this period is subject to variations. Very frequently it increases steadily to 102° to 104°F. (38.9° to 40°C.), with ordinary morning remissions (Fig. 64). In very many cases, however, after a sharp initial rise there occurs about the 2d or 3d day a decided remission or even intermission of both evening and morning temperatures, followed by the redevelopment of fever before the eruption appears (Fig. 65). Occasionally the cutaneous eruption begins to develop before the temperature rises again (Fig. 66). This fall of temperature is entirely unattended by any amelioration in the other symptoms. It has been considered by many writers to be characteristic of the disease. Others maintain that the curve without intermission occurs as frequently as the other, and this has been my own experience. Bolognini[1] describes a very slight sensation of crepitation obtained by palpation of the abdomen during the stage of invasion. His observations are confirmed by Köppen.[2] The duration of the stage of invasion in typical cases is 3 or 4 days. Irregularity in its duration and course will be referred to later (p. 390).

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[1] Arch. of Pediat., 1900, XVII, 920.

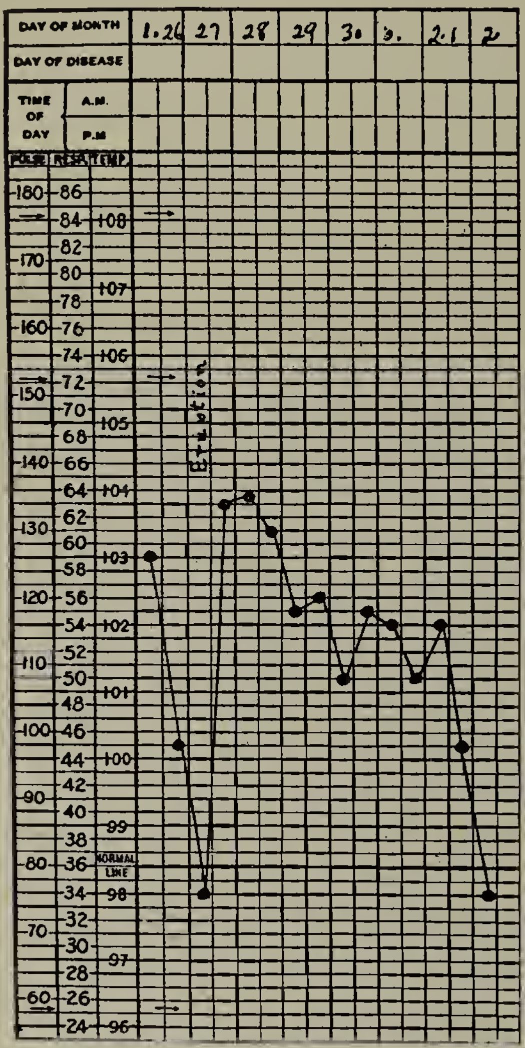

Fig. 66.—Measles with Pre-eruptive Fall of Temperature Continuing when Rash First Appeared.

Howard M., aged 7 years. Jan. 25, catarrhal symptoms and fever; Jan. 26, temperature falling; Jan. 27, abundant eruption on face, spreading to body, temperature still afebrile; Jan. 30, rash almost gone, temperature still elevated; Jan. 31, desquamation.

Period of Eruption.—*The Rash.*—The characteristic rash is found on the skin by the end of the 3d or the morning of the 4th day of the disease in ordinary cases; sometimes not until the 5th day. It develops first either upon the forehead, scalp, cheeks, temples, behind the ears, or about the mouth; the exact situation probably varying with the case. It then spreads with variable rapidity, but as a rule is not only more abundant by its 2d day in the regions where first seen, but has extended over the whole body, possibly excepting the legs and forearms, feet and hands, which may not be involved until its 3d day. Even the palms and soles finally exhibit it. The *individual spots* are at first of small pin-head size, pale-red, not elevated, round or irregular in shape, and discrete with the surrounding skin healthy. They rapidly enlarge up to the size of a large split pea, becoming distinctly elevated to sight and touch and of a darker-red color, commonly with a slightly bluish cast. There exists a decided tendency to grouping, a series of spots becoming confluent by their margins into irregular, short, straight or curved lines—the well-known "crescentic eruption" (Fig. 67). These groupings are separated by small white channels of healthy skin, the contrast producing the appearance characteristic of this disease. In some portions of the body, especially the face, back, buttocks and the inner surface of the thighs, the rash may become confluent in large areas, the intervening channels disappearing to a large extent. Even here, however, the peculiar uneven roughness and lack of uniformity in coloring are quite evident.

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[1] La Pediat., 1895, III, 110.

[2] Centralbl. f. inn. Med., 1898, XIX, 673.

The individual spots reach their fullest development in about 24 hours, and then begin to fade. Pressure by the finger will at first completely obliterate them for a moment; later a slight discoloration remains. Taken as a whole the rash is at its height on the 2d or 3d day of the eruptive period and then fades, beginning in the situation where it first appeared. It may consequently be diminishing on the face while not yet at its height upon the legs. All traces of the eruption have disappeared by the 4th or 5th day after the first appearance, except a pale-yellowish or brownish pigmentation which very commonly persists for a decidedly longer time.

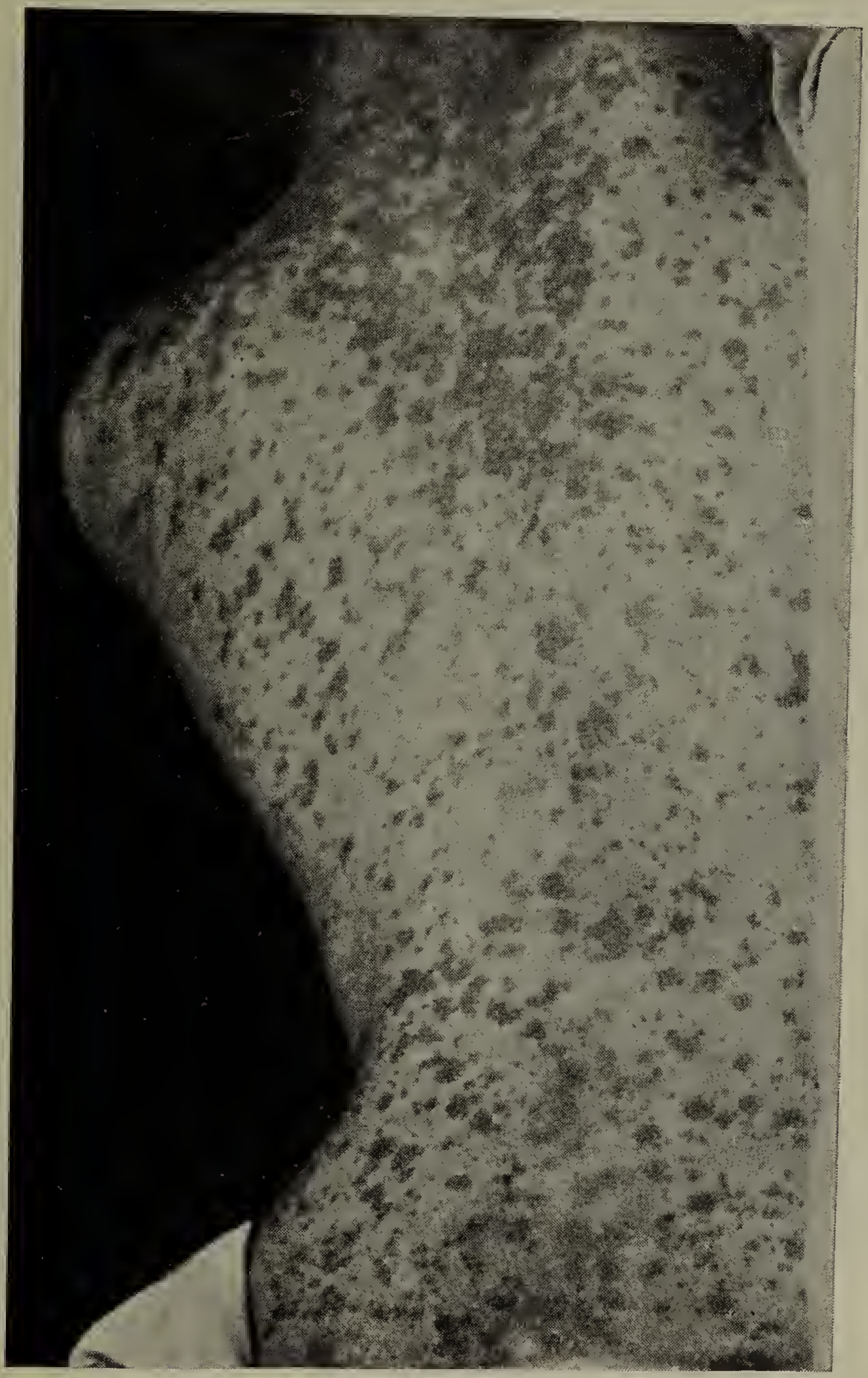

Fig. 67.—Eruption of Measles.

Boy of 12 years, showing the characteristic grouping exhibited by the eruption.

Other Symptoms of the Eruptive Stage.—The fever generally increases with the development of the rash and reaches its maximum with this upon the 2d or 3d day of the eruptive stage, with only slight morning remissions. There are, however, very many exceptions to this rule, and it is of frequent occurrence for the temperature to fall almost by crisis even upon the 2d day of this stage, while the rash is still at its height, or even before it has attained this. All the catarrhal symptoms meanwhile persist or increase in severity as the rash develops. On the 2d day of the rash there is present a very decided puffiness of the face, which, with the photophobia, conjunctivitis, severe coryza, obstructed nasal respiratiod, and excoriation of the upper lip give to the child a very characteristically stupid expression (Fig. 68). The eyes secrete freely, and the edges of the lids stick together during sleep. The voice is hoarse, cough is often distressing, and numerous râles can generally be heard in the chest. The tongue is coated, and in severe attacks may become dry or even denuded. The inflammation of the fauces and pharynx continues but is seldom very severe. Thirst is great, the appetite is lost, and diarrhea is a frequent and sometimes troublesome symptom. Vomiting is not common. There may be slight delirium during the height of the attack, or the drowsiness of the prodromal stage may persist. The superficial lymphatic glands throughout the body are swollen and often tender. As I[1] have pointed out elsewhere this enlargement is quite commonly so considerable that its well-recognized occurrence in rubella as well cannot be considered so diagnostic a symptom of the latter disease as is often supposed. The spleen is enlarged during the eruptive stage and becomes smaller as the rash subsides (Bleyer)[2]. There is often very

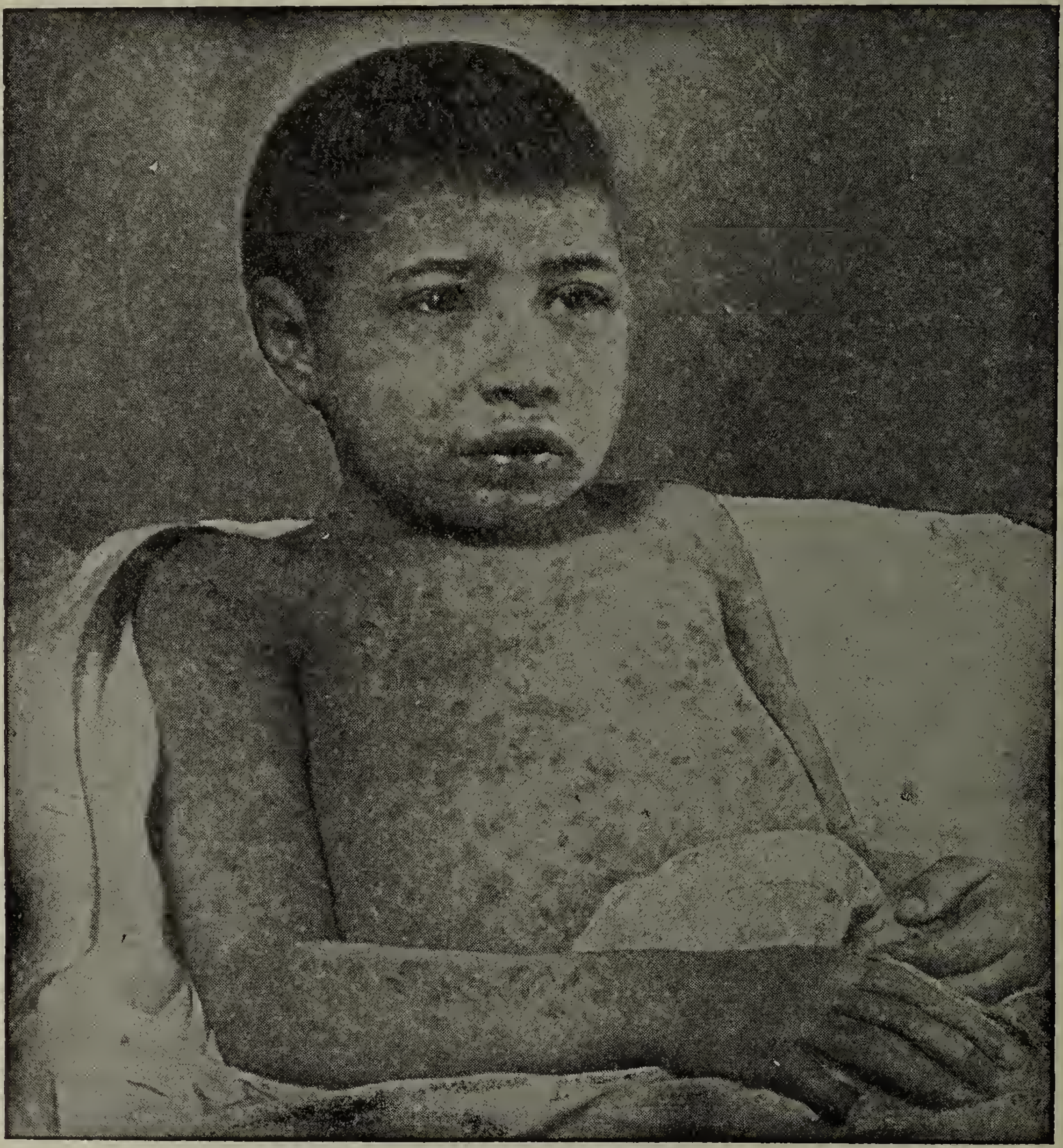

Fig. 68.—Facies in Measles.
Girl with measles, showing the peculiar heavy and swollen appearance of the face.

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[1] Univ. Med. Mag., 1892, IV, 634.
[2] Amer. Jour. Dis. Child., 1926, XXXI, 26.

annoying itching of the skin. Febrile albuminuria sometimes occurs; there is usually a marked diazoreaction; diacetic acid and propeptone may sometimes be found in the urine, and a moderate urobilinuria is present (Rach and Reuss).[1] The Pirquet tuberculin-reaction disappears during the eruptive stage in cases in which it was positive earlier.

The blood was studied by Renaud[2] and since then by Flesch and Schossberger,[3] Hecker,[4] Lucas[5] and others. There may be a transient lymphocytosis early in the incubation, but from 2 to 6 days before the first symptoms appear there occurs a leucopenia with a very characteristic relative diminution or even disappearance of the lymphocytes, the blood-picture showing a predominance of polymorphonuclear cells. This diminution of the lymphocytes shows itself several days before the development of the buccal eruption. During invasion the number of polymorphonuclear cells becomes diminished in uncomplicated cases both relatively and absolutely, and the blood-picture again becomes lymphocytic. A leucopenia, especially of the polymorphonuclear cells, with increase of the mononuclea, cells is very decided in, and quite characteristic of, the early eruptive stage. The eosinophiles are diminished in number. The normal number of leucocytes is soon attained as convalescence advances. Leucocytosis develops if complications are present. With the development of the eruption the number of blood-platelets diminishes and the bleeding-time shortens. An increase of platelets occurs with the fading of the rash (Schiff and Mátyas).[6]

When the rash begins to fade on the 2d or 3d day of the eruptive period, the temperature, if still elevated, falls rapidly by lysis, often reaching normal in 1 or 2 days after the fall begins. The catarrhal symptoms also improve rapidly, keeping pace with the temperature, although hoarseness, cough, and irritation of the eyes frequently last for several days longer. The average duration of fever is about 7 days in all, but often less. (See Figs. 65 and 66.) The frequency of respiration and pulse is in proportion to the elevation of temperature, unless complications appear. The rapidity of the disappearance of the symptoms is generally very striking.

Period of Desquamation.—The eruptive and desquamative stages cannot be very sharply demarcated. As a rule the stage of desquamation may be said to begin with the disappearance of the eruption, not including the pigmentation, and with the subsidence of other symptoms on the 7th or 8th day of the disease, although the actual scaling may not appear until 1 or several days later. The desquamation consists of very fine, branny scales. As a rule it is found first on the face and later elsewhere, following the order in which the eruption made its appearance. It continues a few days to a week, or occasionally longer. The amount of desquamation is usually in proportion to the intensity of the eruption; but although sometimes extensive and very noticeable, it is generally slight, most evident on the face, and in many cases cannot be detected at all. There are no symptoms characteristic of this stage, except in some cases a persistence of the catarrhal manifestations, especially the cough and the conjunctival irritation.

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[1] Zeitschr. f. Kinderheilk., Orig., 1911, II, 460.

[2] Thése de Lausanne, 1900. Ref. v. Jürgensen. Nothnagel's Encyclop. Amer. Med. Measles, 335.

[3] Jahrb. f. Kinderheilk., 1906, LXIV, 724.

[4] Zeitschr. f. Kinderheilk., Orig., 1911, II, 77.

[5] Amer. Jour. Dis. Child., 1914, VII, 149.

[6] Monatsschr. f. Kinderheilk., Orig., 1919, XV, 254.

Variations from the Ordinary Type.—Many variations from the type described may mark the attack either as a whole or in certain particulars.

The *duration of incubation* may be altered, but a stage of less than 8 days or more than 12 days is unusual. As short a duration as 5 days and as long as 3 weeks are on record. Occasionally symptoms are observed consisting of loss of appetite, slight fever, malaise, and perhaps slight indications of the catarrhal condition to follow. Exceptionally other symptoms appear during this period, but such are to be regarded as accidental or anomalous.

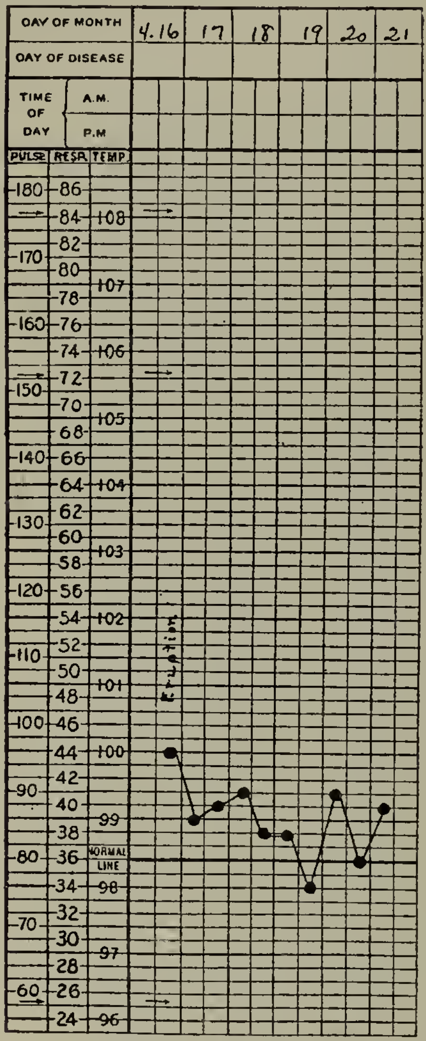

Fig. 69.

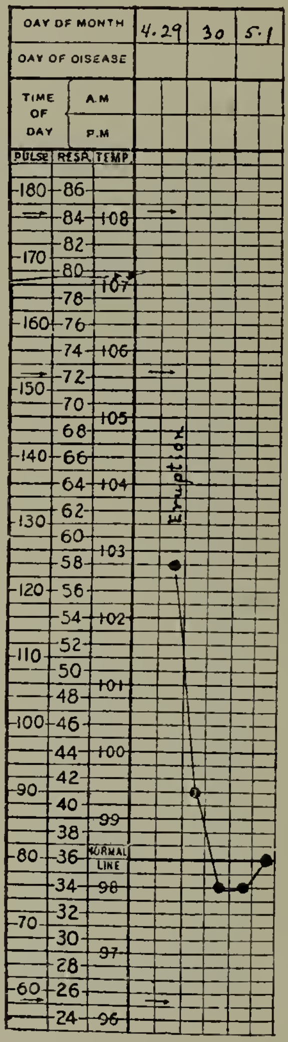

Fig. 70.

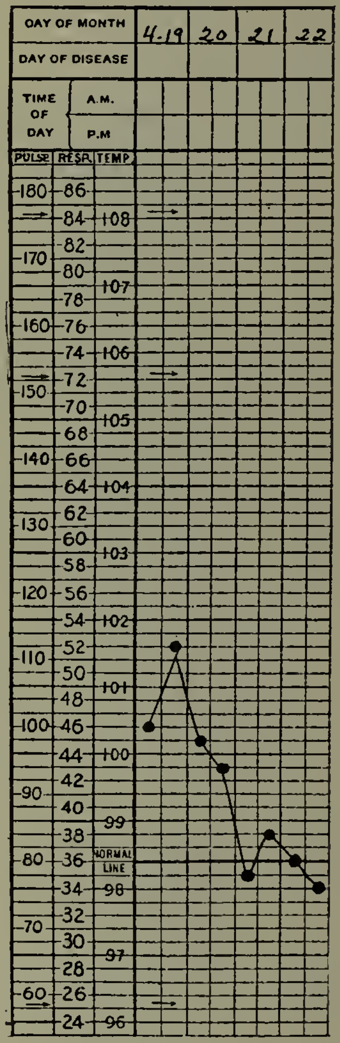

Fig. 71.

Fig. 69.—Rubeola Afebrilis.

Richard G., aged 8 years. Apr. 15, vomited, lachrymation; Apr. 16, vomited again, characteristic rash after a mustard-bath, mild catarrhal symptoms throughout attack, no fever during eruptive period, and no indication of it earlier.

Fig. 70.—Abortive Measles.

Frank A., aged 4 years. Apr. 29, for 3 days had coughing, sneezing and drowsiness, typical eruption appeared today on face, catarrhal symptoms continue; Apr. 30, rash spread yesterday, now fading; May 1, rash nearly disappeared.

Fig. 71.—Rubeola sine Catarrho.

Willie B., aged 10 years. Apr. 18, no symptoms noted; Apr. 19, no catarrhal symptoms except a very slight redness of the eyes, eruption developing; Apr. 20, eruption more abundant, no catarrhal symptoms whatever; Apr. 21, rash fading. House-epidemic.

The *stage of invasion* may be unusually protracted, extending perhaps 6 to 8 days Barthez[1] reports an instance of invasion continuing 16 days. It is, however, more prone to be abnormally short, lasting only 1 to 2 days, and sometimes there appears to be no period of invasion whatever. I have seen entire local epidemics of measles in institutions characterized by an absence or very slight development of prodromes. The

[1] Barthez and Sanné, Malad. des enf., 1891, III, 17·

combined length of incubation and invasion is fairly constantly 14 days. If, then, one of these periods is lengthened or shortened, the other must vary inversely with it. There are, of course, exceptions in which the total duration of the two periods is shorter or longer than the figure given. In other cases the initial symptoms may be unusually intense; stupor, convulsions, continuously high fever and severe diarrhea or vomiting being among these. The respiratory symptoms may be excessive, the secretion from the eyes and nose being profuse, and the breathing difficult, or croup may complicate the condition. Violent epistaxis may occur, or other complications may influence the character of this stage. In 1 instance under my own observation a well-marked lobar pneumonia represented the chief of the symptoms of invasion. This was followed at the proper time by the development of the eruption and of other evidences of measles, while the pneumonia meanwhile underwent rapid resolution. Bronchopneumonia is also reported as a prodromal symptom. These unusually severe initial symptoms may continue into the eruptive stage, or may ameliorate when the eruption appears.

The *eruptive stage* is also subject to numerous variations, giving distinct types to the attack. First is to be mentioned the **mild form.** In this the child is so little ill that it is not confined to bed, and suffers practically no discomfort. After a short invasion the catarrhal symptoms continue to be of very mild character and the fever is only slight, or even entirely absent (**Rubeola afebrilis**) (Fig. 69). The eruption is often pale-red, scanty and very little confluent, strongly suggesting rubella, or so poorly developed that diagnosis is very difficult.

An **abortive form** is characterized by a well-marked stage of invasion, but with an eruptive stage which, sometimes typical at the onset, sometimes poorly developed, rapidly disappears together with the attending symptoms (Fig. 70). **Measles without eruption** (**Rubeola sine eruptione**) has been described, catarrhal symptoms being present unattended by rash. Embden[1] reports 20 instances in one epidemic of 461 cases. So, too, cases are observed in which the characteristic rash occurs but without catarrhal symptoms (**Rubeola sine catarrho**) (Fig. 71). It is almost certain that the great majority of the cases apparently of this variety are instances of rubella or some disorder of the skin. Only in house-epidemics could the diagnosis of either of these two forms be made, and then only with great reserve.

The **severe forms** of measles, apart from the influence of complications, may be of several varieties, although no sharp line of distinction separates one from another. In one, which may be called the **prolonged type,** the fever lasts an unusually long time. This, as a rule, depends upon the persistence of catarrhal symptoms, and especially upon a more than ordinary degree of bronchitis. In other severe cases the virulence of the poison is very great. All the symptoms exhibit an unusual severity from the outset, the eruption being very intense, wide-spread, confluent, and of a dusky, blue-red color. In other instances the disease begins in the ordinary way, but soon assumes a severe type. In still others some of the symptoms are but little marked. This is especially true of the rash, which may be poorly-developed throughout the attack and frequently late in appearing, or which often shows a remarkable tendency to repeated disappearances, even when brought temporarily into evidence by hot baths ("Retrocession of the eruption"). It was long erroneously supposed that this "striking in of the rash" was the cause of the unfavorable symptoms. It is, however, only an evidence of the feebleness of the circulation. Many of these cases with retrocession assume what has been called the **typhoid form.** The rash is then at no time well-developed, there is great prostration, dry tongue, rapid and weak pulse, rapid respiration, high temperature, low delirium, restlessness, twitching, and perhaps repeated convulsions.

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[1] Inaug. Dissertat. Heidelberg, 1889.

The most severe type of the disease is the **malignant form.** The symptoms are even more severe than those described, the course of the disease rapid, and death may take place before the time is reached for the eruption to appear (Fig. 72). The temperature in some of these severe cases may reach 108° to 109°F. (42.2–42.7°C.). The **hemorrhagic type,** or "black measles" of the older writers, is another severe type, really a malignant variety, less often seen than formerly. It occurs only in subjects already much debilitated. The rash is pale and never well-developed. Hemorrhage stake place into the spots and elsewhere into the skin as well as into the muscles and from the mucous membrane. There is delirium and great prostration (Fig. 73).

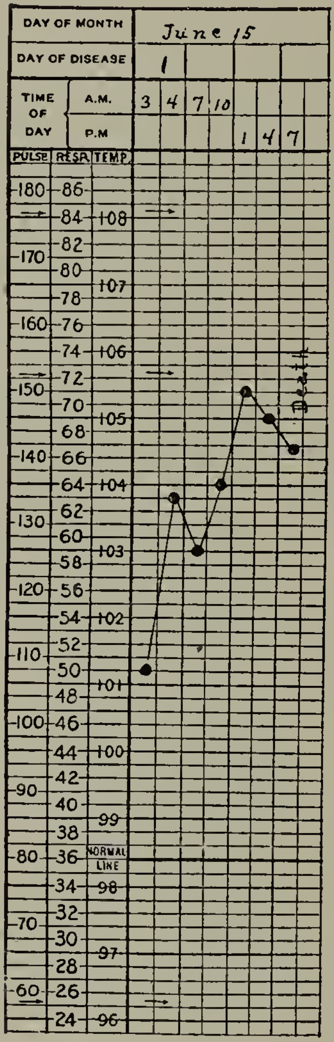

FIG. 72.

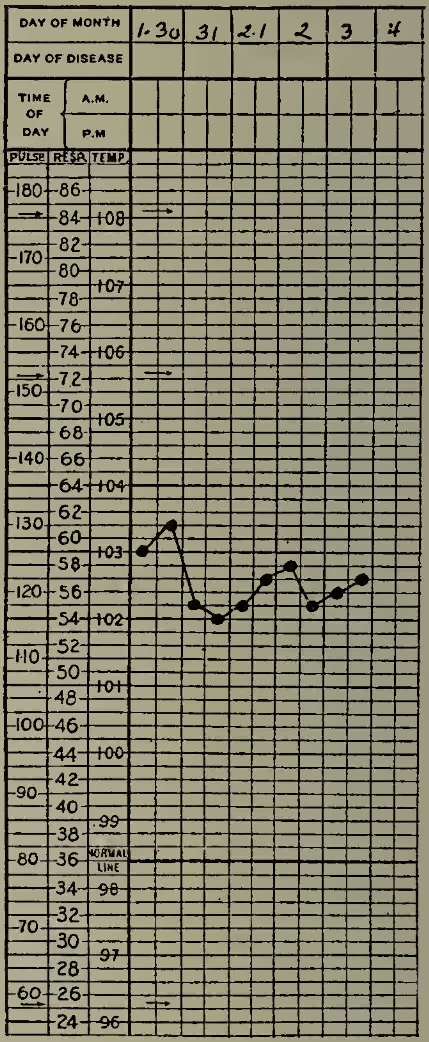

FIG. 73.

FIG. 72.—MALIGNANT MEASLES.

Robert McD., aged 16 months. June 14, vomiting, purging, fever, convulsions, said to have had fever several days, older brother convalescent from measles; June 15, faint macular rash after hot bath, convulsions and unconsciousness continued, hydrencephalic cry, death in evening of the 1st day of the eruption.

FIG. 73.—HEMORRHAGIC MEASLES.

Willie P., aged 5 years. Jan. 30, catarrhal symptoms noted; Jan. 31, severe chill, cyanosis, unconsciousness, well-developed rash followed hot bath; Feb. 3, been growing steadily worse, tongue dry, delirium; Feb. 4, eyes sunken, crowded petechiæ on abdomen, larger purpuric patches on legs. Death.

There are a number of **minor variations** from the ordinary type depending upon *characteristics of the eruption,* none of them possessing any special significance. Sometimes the rash appears upon the trunk before it does upon the face. Infrequently the macules are very small and so closely placed that the rash of scarlet fever is simulated. In some mild cases the rash develops only upon some one part of the body, especially the face and neck. In *Morbilli papulosi* the eruption consists of large, deep-red spots more papular than usual. *Morbilli miliares* exhibits minute vesicles upon the usual eruption. A *Morbilli bullosi* has also been described, although rarely seen, an eruption resembling pemphigus replacing or being combined with the ordinary rash of measles. Cases are described by Neff[1] and by Morton[2] and the literature reviewed. The term, *Ecchymotic measles* is well applied to the cases so frequently seen in which extravasation of blood or of blood-coloring matter has taken place into the rash, especially on the extremities. The deep-reddish-purple markings thus produced are uninfluenced by pressure, and remain distinct for days or even weeks after all other symptoms of the disease, except desquamation, have disappeared. To call this form "hemorrhagic measles" as is sometimes done, is a source of confusion, since there are none of the grave symptoms present which characterize the latter affection.

Complications and Sequels.—The younger the child the more frequent and more serious are the complications and sequels liable to be. After early childhood they are comparatively uncommon. Most important and oftenest seen are those connected with the *respiratory tract.* Exceptionally epistaxis may be so severe or so often repeated that it becomes an important complication. Chronic nasal discharge may be a sequel. Catarrhal laryngitis is always present to some extent and may at times be attended by so much mucous secretion, spasm or edema that severe laryngeal stenosis develops and even death may occur. When the stenosis depends on the first two factors it is intermittent; when on the last it is more or less constantly present. Stenosis is always a dangerous complication unless of very short duration. Ulcerative laryngitis is a not uncommon complication or sequel. Membranous laryngitis may be due to the presence of either a streptococcus or of the diphtheria-bacillus. (See p. 505 and Vol. II, p. 267.) The first occurs oftenest early in the attack, the second at a later period. The symptoms are much the same and a positive diagnosis can be made only by bacteriological examination. Gannelon[3] found membranous laryngitis in 14.4 per cent. of 1633 cases of measles. A subglottic laryngitis may occur and cause great disturbance of respiration, and even death, no membrane being discoverable.

Trachitis and bronchitis belong to the regular symptoms of the disease. The latter may sometimes become so severe that it prolongs the fever decidedly and may then be looked upon as a sequel.

Bronchopneumonia, frequently tuberculous, is very common especially in infancy and early childhood and is always most serious. It may develop at any time in the course of the disease, but oftenest during the eruptive stage or as a sequel after a short intermission. The statistics regarding the frequency of the occurrence of bronchopneumonia vary considerably. Von Jürgensen[4] found them ranging from 6 per cent. to

[1] Amer. Jour. Dis. Child., 1920, XIX, 469.
[2] Brit. Jour. Child. Dis., 1921, XVIII, 188.
[3] La rougeole a l'hospice des enfants assistées, Thèse de Paris, 1892.
[4] Nothnagel's Encyclop. Amer. Ed., 318.

16 per cent. of the reported cases of measles. Mixsell and Giddings,[1] who studied 3080 cases, found that pneumonia occurred in 26.8 per cent., all but a few being of the bronchopneumonic type. This is an unusually high incidence, depending probably on the fact that the patients were all hospital-cases, and that 68.65 per cent. were between birth and the age of 5 years.

Croupous pneumonia is a much less common complication, but is occasionally seen. Gangrene of the lung and pleurisy with effusion are observed exceptionally and bronchiectasis has been recorded as a sequel.

The *gastroenteric tract* likewise furnishes many complications and sequels. Some degree of catarrhal stomatitis is one of the regular symptoms of the disease. Aphthous and ulcerative stomatitis are frequently seen. Gangrenous stomatitis is rare, yet measles perhaps more than any other disease predisposes to it. Membranous pharyngitis is not uncommon, patches developing on the tonsils and adjacent parts. Like membranous laryngitis, with which it is often combined, it may be streptococcic or truly diphtheritic in nature. Diarrhea is not infrequently sufficiently severe to constitute a complication. Oftener it continues as a sequel, sometimes in a very chronic form. It may be simply catarrhal in nature or may depend upon ileocolitis. This latter is especially liable to develop in summer-time in debilitated subjects under 2 years of age. Le Lyonnais[2] collected 18 instances of appendicitis occurring as a sequel to measles.

Otitis is of frequent occurrence, varying with the epidemic. In a mild non-suppurative form it is very common in the early part of the eruptive stage. Then in young infants it may not be detected because of the failure to rupture externally. In a more severe form, with pain, suppuration and fever, it occurs oftener as a sequel about the end of the 2d week and it may result in mastoid involvement. Both ears are then usually affected, permanent injury may result, or meningitis may follow. Of 501 cases of chronic disease of the middle ear published by Downie[3] 26.1 per cent. owed their origin to measles. Yet as a rule chronic otitis follows much less often than after scarlet fever.

Inflammation of the eyes of various sorts is often observed with or after the attack. Catarrhal conjunctivitis is one of the symptoms of the disease and can be considered a complication only when unusually severe. It is frequently very persistent in poorly nourished children. Keratitis and iritis may occur as sequels and optic neuritis is occasionally seen, Griscom[4] having collected 23 cases in addition to 1 reported by him.

Circulatory affections are uncommon. Endocarditis, pericarditis and myocarditis are rarely seen. Thrombosis of the vessels in different parts of the body may exceptionally occur (Lutz).[5] In the extremities this may produce gangrene of the limb. *Swelling of the thyroid gland* has been reported. Of *genito-urinary* complications nephritis is generally considered unusual. I have, however, seen it not infrequently either with the attack or later. Acute degenerative lesions of the kidney develop in malignant cases. Pyelitis may occur. Ulcerous vulvitis and gangrene of the vulva are very rare sequels, to which, however, measles especially predisposes.

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[1] South Med. Jour., 1923, XVI, 90.
[2] Thèse de Paris, 1913–14, No. 109. Ref. Brit. Jour. Child. Dis., 1914, XI, 234
[3] Brit. Med. Jour., 1894, II, 1163.
[4] Ann. of Ophthalm., 1912, XXI, 42.
[5] Berl. klin. Wochenschr., 1913, L, 1566.

Affections of the bones and skin have been recorded as complications, among them gangrene, herpes zoster, herpes labialis, urticaria, erythema, and furunculosis. Osteomyelitis and necrosis have been recorded. Arthritis is a very uncommon sequel. Generalized cutaneous emphysema has been reported, or cutaneous tuberculosis rapidly develops. Purpura hæmorrhagica is a rare sequel, studied by Kelly.[1]

Nervous affections are unusual. Serous meningitis occurs occasionally as a sequel to otitis, or without this; or that of a tuberculous nature appears as a result of a complicating tuberculosis. A dull apathetic condition is exceptionally seen, lasting for weeks after the disease is over. I have observed it in 1 instance. In 2000 cases of imbecility Beach[2] found 11 which dated from an attack of measles. Chorea and epilepsy have been reported as sequels and tetany has been occasionally observed during the attack. Convulsions sometimes develop during the attack and then constitute a very unfavorable indication. Paralysis of various forms may follow measles. It may be cerebral, myelopathic or peripheral in nature. Bénard[3] reports a fatal case of bulbar paralysis occurring as a sequel. The subject of paralysis after measles has been reviewed by Allyn,[4] Brückner,[5] and others. Encephalitis exceptionally develops as a sequel. We[6] have known this to occur in a number of instances. The subject has been briefly reviewed by Horwitt.[7]

Other *infectious diseases* may exist in conjunction with measles, or as sequels or predecessors to it. One of the most common and unfavorable combinations is that of measles and diphtheria, the latter being prone to develop during the convalescent stage of the former. In 3400 cases in the Medical Asylums Board's Hospitals (Rolleston)[8] 2.10 per cent. developed diphtheria. Scarlet fever and measles often occur together, or one immediately after the other. The combination of varicella, erysipelas, vaccinia, grippe, or typhoid fever with measles is occasionally seen, or the immediate following or preceding of the latter by one of the others. Epidemics of pertussis are particularly liable to precede or follow or to prevail at the same time with epidemics of measles. Measles, too, is certainly very prone to be followed closely by evidences of tuberculosis or, if the latter disease has already manifested itself, to increase its further development. Very many of the cases of bronchopneumonia attending measles are tuberculous in nature, and osseous or glandular tuberculosis is frequently a sequel.

Relapse.—This is encountered usually with great rarity, although Leach[9] observed it 4 times in an epidemic of 262 cases. Goldberger[10] reviews the literature upon this subject and reports 3 personal cases. Macciotta[11] has observed it 8 times. Undoubtedly many reported instances are errors in diagnosis. It takes place in from the 2d to the 4th week or sometimes later, after the attack is apparently over but while the original infection is still present in the system. It consists in the return of some or all of the characteristic symptoms of the disease. The mere re-development of some one symptom during the attack, as, for instance, the reappearance of the rash, does not constitute a relapse. As a rule the relapse is less severe and of shorter duration than the first attack.

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[1] Brit. Jour. Child. Dis., 1922, XIX, 86.
[2] Brit. Med. Jour., 1895, II, 707.
[3] Bull. et mém. soc. méd. d. hôp. de Paris, 1921, XLV, 184
[4] Med. News, 1891, LIX, 617.
[5] Jahrb. f. Kinderheilk., 1902, LVI, 725.
[6] Amer. Jour. Dis. Child., 1920, XX, 84.
[7] Arch. of Pediat., 1924, XLI, 476.
[8] Brit. Jour. Child. Dis., 1915, XII, 21.
[9] Lancet, 1905, II, 1837.
[10] Amer. Jour. Dis. Child., 1925, XXX, 55.
[11] Il Policlinico; sez. prat., 1925, XXXII, 116.

Recurrence.—In spite of the wide-spread belief among the laity to the contrary, second attacks of measles are of very great rarity. This is the opinion of the majority of authorities. On most occasions the so-called recurrences are instances of errors in diagnosis, which circumstances make particularly easy in this disease. Maiselis[1] collected only 21 cases from medical literature, while he found 154 of scarlet fever. In 1100 cases of the disease Widowitz[2] observed no instance of a second attack. Berinsohn[3] records recurrence in 5 per cent. of 80 infants; in 2.2 per cent. of 314 children between 1 and 3 years of age; in 1.2 per cent. of 415 children between 4 and 7 years of age, and in none above this age. This incidence seems unusually high.

Prognosis.—The prognosis of measles is generally good, the mortality averaging from 3 to 6 per cent., often less, but sometimes reaching much higher figures. During 5 years there occurred in Philadelphia 50,715 cases, with a mortality of 1.6 per cent. (Graham).[4] The number of cases of measles is, however, so great that the number of deaths from it is large. Comby[5] gives 20,518 fatal cases in Paris during 19 years and McCollom[6] states that the deaths during 5 years in London per 10,000 of the population were 3 or 4 times more numerous from measles than from scarlet fever. Veeder[7] estimates that between 9000 and 10,000 deaths from measles take place annually in the United States. The greatest number of fatalities is observed in the 2d week of the attack.

Many factors influence the mortality. That of the *epidemic* is one of the most noteworthy of these, the disease being much more fatal in some years than in others. At times under unfavorable conditions the death-rate has exceeded 30 per cent. of the cases, while at other times it does not reach 2 per cent. In 1914 in 170,004 cases of measles occurring in portions of the United States the mortality was 1.73 per cent. (Wilson).[8] *Age*, also, exercises a powerful influence. In general the younger the patient attacked during infancy and childhood the graver the prognosis, and the number of fatal cases is very much diminished after the 5th year. The disease appears to be milder and the mortality less in the first 6 months than in the succeeding months of the 1st year. Variot[9] found a mortality of 12.31 per cent. among 601 cases of the disease in children. In the 1st year the mortality was 32.72 per cent. and in the 2d year 29 per cent. The greatest actual number of deaths occurred in the 2d year, there being fewer cases of the disease in the 1st. Of 367,602 deaths from measles reported in England and Wales during 40 years 335,874 were in children under 5 years of age (Williams).[10] Henoch's[11] statistics for the Charité Hospital gave a mortality of 55.6 per cent. in the first 2 years of life and 9.3 per cent. for from 3 to 11 years. In some epidemics, however, the mortality has been notably high among adults. This was the case in the Faroes' epidemic of 1845 (Panum),[1] and Kilbourne[2] reported a severe epidemic of 600 cases occurring in barracks with a mortality of 5 per cent.

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[1] Virchow's Archiv, 1894, CXXXVII, 468.

[2] Wien. klin. Wochenschr., 1909, XXII, 1596.

[3] Nederlandsch. Tijdschr. f. Geneesk., 1924, II, 1864, Ref. Jour. Amer. Med. Assoc., 1924, LXXXIII, 1630.

[4] Jour. Amer. Med. Assoc., 1917, LXVII, 1272.

[5] Traité des mal. de l'enf., 2d ed., I, 347.

[6] Bost. Med. and Surg. Jour., 1903, CXLVIII, 31.

[7] Arch. of Pediat., 1917, XXXIV, 321.

[8] Arch. of Pediat., 1916, XXXIII, 261.

[9] Bull. de la soc. de pédiat., 1904, No. 1.

[10] 20th Cent. Pract. of Med., XIV, 120.

[11] Ref. Williams, *loc. cit.*, 121.

Debilitating influences, such as want, exposure, crowding, imperfect hygiene in general, and neglect of treatment, increase the death-rate greatly. It is probably such factors which have caused the high mortality in foundling asylums and other institutions for infants and children, and among soldiers in camps. The proportion of fatal cases among the poor in institutions is always much higher than in private practice among the better classes. Of 1575 children treated during 5 years in the Hospice des Enfants-Assistés in Paris, 728 died; *i.e.* 46.22 per cent. (Comby).[3] Rolleston[4] reported a mortality of 12.6 per cent. in 3400 cases in the Metropolitan Asylums Board's Hospitals in 1913, most of the children being of the poorer classes.

Complications and sequels are far oftener the cause of death than is the disease itself. So, too, the occurrence of measles as an affection secondary to other diseases adds greatly to the danger. Bronchopneumonia, tuberculous or otherwise, perhaps occasions more deaths than any other complicating affection, but diphtheria and diarrhea are the factors in many instances. Of 157 cases of the combination of measles and diphtheria published by Blakely and Burrows[5] 34 per cent. died. Even after the disease seems completely over, death often results from the development of fatal sequels, particularly tuberculosis, to which disease measles especially predisposes or which, when already present, measles is likely to bring into activity.

Certain *unfavorable symptoms* may be referred to. A high temperature during invasion indicates that the attack will probably be a severe one. A poorly developed eruption or one which retrocedes readily, combined with marked general symptoms, is of unfavorable import. High fever persisting while the other symptoms are disappearing suggests the presence of some complication. The development of unusual hoarseness may denote the existence of severe laryngitis.

Diagnosis.—The diagnosis of measles, although generally easy in the eruptive period, is sometimes attended by great difficulty. It rests principally upon the long prodromal stage with the attending fever and catarrhal symptoms, and the development later of the characteristic eruption. In the stage of invasion the resemblance of the catarrhal symptoms to those of a severe cold is very close. The fever is perhaps unduly high for the latter condition, and the symptoms in general too severe; yet positive diagnosis at this stage would be impossible were it not for the presence of the spots upon the palate, and especially of the characteristic buccal eruption. Very exceptionally the latter has been reported present in pertussis and follicular tonsillitis (Michelazzi),[6] and in rubella (Widowitz;[7] Muller[8]). Yet since few observers claim to have seen it in any other disease than measles, the presence of the buccal eruption is a very valuable diagnostic sign. Its absence, however, is not proof that the disease is not measles.

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[1] *Loc. cit.*, 292.

[2] Milit. Surgeon, 1912, XXXI, 294.

[3] Traité des mal. de l'enf., 2d ed., I, 348.

[4] Brit. Jour. Child. Dis., 1915, XII, 21.

[5] Bost. Med. and Surg. Jour., 1901, CXLV, 89.

[6] Gaz. degli. osp. e delle clin., 1904, XXV, 35.

[7] Wien. klin. Wochenschr., 1899, XII, 919.

[8] Münch. med. Wochenschr., 1904, LI, 98.

The diminution of the lymphocytes is of diagnostic value during incubation, the relative increase of polymorphonuclear cells then distinguishing measles from the first stage of pertussis, in which there is an increase of the lymphocytes (Renaud).[1] The neutrophilic leucopenia and the diminution of the number of the eosinophiles are a diagnostic aid in the eruptive stage, being exactly the opposite of the condition seen in scarlet fever.

Rubella is the disease which resembles measles most closely. The chief points of distinction are the shortness and mildness or absence of prodromes in rubella; the slight degree of catarrhal symptoms; and the more rapid development of the eruption, its absence of grouping, and its more multiform, paler and fugacious character. The degree of glandular enlargement is of little aid in diagnosis. (See Rubella.) Yet in some atypical cases of rubella the rash is characteristically morbilliform in character, and the catarrhal symptoms are marked, while in some instances of measles the symptoms, including the eruption, are poorly developed and strongly suggest rubella. In sporadic cases of measles the diagnosis is consequently often impossible unless the buccal eruption is discovered.

Scarlet fever can cause difficulty only in atypical cases. Such cases sometimes exhibit a very blotchy rash somewhat resembling measles, while, on the other hand, the rash of measles may at times be so confluent that scarlet fever is suggested. Careful examination will, however, show decided differences in the two eruptions. That of measles is always slightly uneven to the touch and areas or channels of healthy white skin can always be found in some localities. The chin and the region about the mouth are free from eruption in scarlet fever and always involved in measles. There is moreover in scarlet fever the more sudden onset, decided sore throat, often initial vomiting, and the absence of catarrhal symptoms. The desquamation in the two affections is entirely different in character. The differences in the blood-picture have been referred to.

Grippe of the respiratory type may closely resemble the initial stage of measles. There is, however, less photophobia. The development of the characteristic rash in measles, or, even before this, of the buccal eruption, will settle the diagnosis. *Typhoid fever* sometimes exhibits a rash so abundant that that of measles is strongly suggested. I have occasionally seen such cases. (See p. 446, Fig. 93.) The diseases have, however, no other symptoms in common. The rash of *typhus fever* may be much like that of measles and catarrhal symptoms attend the prodromal stage. Other symptoms, however, differ entirely. Severe and rapidly fatal cases of *cerebrospinal fever* may resemble malignant measles. Both possess the sudden development of threatening cerebral symptoms, while ill-defined eruption with a hemorrhagic tendency may be present in either. I have known of several instances where the diagnosis between these two diseases could not be determined with certainty. The eruption of *variola* may at first resemble measles with an unusually papular rash. The error fortunately is usually that of considering measles to be small-pox. The diagnosis is generally soon apparent. The initial stage of variola is totally different, being marked by headache, vomiting and pain in the back, without catarrhal symptoms, while the rash is more papular and shot-like than that of measles. It is possible, too, for the prodromal rash of variola to resemble the eruption of measles to some extent. *Vaccinia* occasionally exhibits a morbilliform erythema. It has, however, no other symptoms suggesting measles. *Varicella* can scarcely be a source of confusion. The eruption could simulate that of measles only at the onset. The roseola of *syphilis* may resemble the rash of measles closely. Other symptoms, however, are lacking.

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[1] Thése de Lausanne, 1900.

Various *eruptions,* not symptoms of infectious fevers, may have a close likeness to that of measles. Notable of these are some of the erythemata, especially the medicamentous rashes, among these being those produced by antipyrine, copaiba, luminal, cubebs and chloral. That following the administration of diphtheria-antitoxin or other serum occasionally awakens suspicion. In none of these are other rubeolous symptoms present. They may, however, lead to errors in diagnosis. This is especially true if coryza happens to coexist. Urticaria and erythema multiforme may exhibit at times a very morbilliform eruption. The absence of other symptoms and the general history of the attack are usually conclusive.

The fact that the Pirquet tuberculin-reaction disappears during the eruptive stage of measles (p. 389) may aid in the diagnosis of some of the disorders mentioned, if it is known that the reaction was positive before the disease developed.

Treatment. Prophylaxis. Quarantine.—Although measles is generally a mild disease, yet the inability to predict the outcome in any given case necessitates the employment of all prophylactic measures possible. Especially are young children and those in a debilitated condition to be protected from it. Unfortunately the ease and frequency with which it is communicated before its existence is suspected render these measures frequently of no avail. Other children of the family who have been in contact with an affected child during the state of invasion have probably already contracted the disease. Still, as it may chance that infection may not yet have occurred, isolation should be established immediately, and maintained until all danger of infecting others is over. The appearance of the hypolymphocytosis during incubation can be made use of effectively in epidemics to enforce prompt isolation of the patient. In the absence of entire certainty regarding the duration of infectiousness quarantine should continue for at least 2 weeks from the onset. Other non-immune children who have been exposed should be kept from intercourse with susceptible playmates for 14 days in order to permit the usual period of incubation to elapse. It must be recognized, however, that the improbability of the disease being transmitted by a third person renders immune children of no danger to others, even if they have been associating with the patients. If the germ, as already stated, does not live longer than 2 weeks from the onset of the attack, disinfection of the room after the disease is not imperative, provided it is thoroughly cleansed and aired. There is no necessity, too, of destroying books, toys and the like. However, disinfection can do no harm, and is an additional precautionary measure. So, too, it is wise to allow the room to be unoccupied by non-immune persons for a week or more after the patient is removed from it. Other susceptible children of the family need not necessarily be sent from the house. They are not likely to contract the disease after isolation is established.

Prophylactic Injection of Convalescent's Serum.—Convalescent's serum in the prophylaxis of measles has been extensively employed and has proved of the greatest value. Its use is limited because serum from convalescents is not always available, and also by the fact that the immunity is only temporary, lasting from 4 to 6 weeks according to von Torday[1] and others. It is especially indicated in checking the spread of the disease in institutions, and at times it is practicable in private families for use in subnormal children or those suffering from other diseases. Although for the treatment of measles already developed convalescent serum was employed in 1895 by Weisbecker,[2] for purposes of immunization it appears to have been first used by Cenci[3] in 1901. Several years later it was given by Nicolle and Conseil[4] and subsequently by Richardson and Connor.[5] It was Degkwitz,[6] however, who stimulated its employment in an extensive and practical way. Many articles have appeared on the subject, and an excellent review is given by Zingher.[7]

The serum is obtained preferably from several donors, free from tuberculosis and syphilis, about 7 to 14 days after defervescence from measles; tested for sterility; and preserved with tricresol or by desiccation. It should be used within a few weeks, but may be active up to 4 months, and the dried serum perhaps longer. Three and a half c.c. (0.12 fl. oz.) are injected intramuscularly before the end of the 4th day after exposure. If given as late as the 6th day the dose should be doubled. Equally good results may be obtained by using citrated or uncitrated whole blood in twice the amounts recommended for serum (Zingher).[8] Convalescent's serum or whole blood used in the manner described will result in protection in a large percentage of children. If given as late as the 7th day after exposure, even though larger doses are employed, complete passive immunity usually will not follow but a modified form of the disease may occur, which probably causes active and permanent immunity. In healthy strong children this modified form with permanent immunity may be sought deliberately as recommended by Debré and Ravina[9] and by Zingher,[10] by the injection of 2.5 c.c. (0.08 fl. oz) of convalescent's serum within from 5 to 10 days after exposure. Regan[11] recommends larger doses, up to 8 or 10 c.c. (.27 or .34 fl. oz.). Experience with the method has now been so extensive and so favorable that there is no possible doubt regarding the efficacy of the treatment. The injection of serum from individuals who have had measles some years previously seems to afford some protection if given in double the dose of convalescents serum (Rietschel;[12] Salomon;[13] (Zingher[14]). Horse-serum and diphtheria-antitoxin have prophylactic qualities against measles, according to Galli,[15] but our own experiences with them lead us to doubt their efficacy. It is evident that they could possess no specific power.

[1] Zeitschr. f. Kinderheilk., 1921, XXIX, 148
[2] Zeitschr. f. klin. Med., 1896, XXX, 312.
[3] Riv. di clin. pediat., 1907, V, 1017.
[4] Bull. et mém. soc. méd. d. hôp. de Paris, 1918, XLII, 337.
[5] Jour. Amer. Med. Assoc., 1919, LXXII, 1046.
[6] Zeitschr. f. Kinderheilk., 1920, XXV, 134; XXVII, 171.
[7] Jour. Amer. Med. Assoc., 1924, LXXXII, 1180.
[8] *Loc. cit.*
[9] Bull. et. mém. soc. méd. d. hôp. de Paris., 1923, XLVII, 226.
[10] *Loc. cit.*
[11] Jour. Amer. Med. Assoc., 1924, LXXXIII, 1763.
[12] Zeitschr. f. Kinderheilk., 1921, XXIX, 127.
[13] Deutsch. med. Wochenschr., 1923, XLIX, 1151.
[14] *Loc. cit.*
[15] La Pediat., 1922, XXX, 898.

Preventive inoculation had been tried by Home,[1] Thomson[2] and others, but without convincing success. The experience of Herrman[3] was somewhat more encouraging. The nasal mucous membrane of infants at the end of the 5th month of life was inoculated with the specially prepared nasal secretion of patients beginning an attack of measles. Many of them appeared to develop the disease in a greatly modified form, which it is believed may confer permanent immunity. The results were good, but on the whole the injection of convalescent's serum seems preferable. Tunnicliff and Hoyne[4] have been able to protect against measles by injections of the blood-serum of a goat which had been inoculated with the green-producing streptococcus previously described (p. 380).

Treatment of the Attack.—In average uncomplicated cases very little treatment of any kind is required and, in any event, is purely symptomatic. The selection and management of the sick-room, and the method of conducting quarantine and nursing are described in the introductory remarks under Infectious Diseases (p. 345). There is particular need of an abundance of fresh air without exposure in this disease, since the tendency to the development of bronchopneumonia is certainly checked in this way. The air should be somewhat moist, and the temperature of the room an average one. There exist, it is true, great differences of opinion regarding the effect of cold air in this affection, but no one would advocate exposure which may cause chilling of the extremities and cyanosis. The eyes should be carefully guarded from undue exposure to light; but without making the room nearly dark as is often done. The sensations of the patient are the best guide in this respect. These can be determined in young subjects by careful observation. Shielding the eyes by a screen is often a great relief. The patient should be confined to bed while fever lasts and should be lightly covered. The head must not be kept too low, as this often tends to increase the amount of cough. The diet should be light, milk being one of the most serviceable foods. Such cereals as oatmeal are to be avoided on account of the possibility of exciting the diarrhea to which the disease predisposes. Precautions must be taken that the patients do not become debilitated from lack of sufficient nourishment.

The patient should receive ablution daily, exposure being carefully avoided. Daily examinations of the mouth, nose and throat ought to be made in order to discover the onset of any complication here, and of the ears frequently.

Further treatment of some of the individual symptoms may be considered more in detail.

Fever in ordinary cases may be combated by such simple febrifuges as potassium citrate or spirits of nitrous ether, in some cases with the addition of small amounts of tincture of aconite root. A warm tub-bath is often of great benefit. If the temperature is unusually high and is unrelieved by these measures, antipyrine or phenacetin may be employed, small repeated doses being given rather than larger single ones. The mere occurrence of a temperature of 104°F. (40°C.) at the height of the disease, unattended by any unfavorable symptoms, does not demand energetic treatment. The continued application of an ice-cap to the head is often serviceable. Occasionally in obstinate and threatening hyperpyrexia the cool pack or cool tubbing is of value, unless general cyanosis or coldness of the extremities shows the existence of cardiac weakness. It must be remembered that children often bear the application of cold water or ice badly.

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[1] *Loc. cit.*

[2] Glasgow Med. Jour., 1890, XXXIII, 420.

[3] Arch. of Pediat., 1915, XXXII, 503; 1922, XXXIX, 607

[4] Jour. Infect. Dis., 1926, XXVIII, 48.

Nervous symptoms, such as headache, unusual restlessness, grinding of the teeth, stupor, impending convulsions and the like, are benefited by some bromide salt, and, still more, by warm tub-baths, phenacetin, or antipyrine given as described. Lowenburg and Schaller[1] report a case of hemorrhagic measles with encephalitis which recovered after vigorous treatment with convalescent serum given intravenously, intramuscularly and by intraspinous injection.

Constipation may be relieved by enemata. Purgatives should, as a rule, be avoided on account of their tendency to act too freely in this disease.

Diarrhea generally requires no medication in ordinary cases in healthy subjects. If at all severe, or if the child is debilitated, it must be promptly checked with bismuth, sulphuric acid, or tannic acid preparations with or without opium.

Vomiting seldom needs other treatment than temporary rest of the stomach with careful selection of the diet.

Irritation of the eyes demands the protection from light referred to. In addition there should be frequent douching with a tepid solution of boric acid. Rubbing the edges of the lids with petrolatum prevents their adhesion during sleep. Cold wet cloths applied to the eyes and changed every few minutes are serviceable when inflammation is severe. Protection and treatment of the eyes must be continued during convalescence until the irritation has disappeared.

Inflammation of the throat and nose may well be treated by spraying with an alkaline antiseptic, such as liquor sodii boratis comp. (Dobell's solution) diluted, or with a mentholated oily spray (menthol 2 grains, liquid petrolatum 1 oz.) (0.13; 30). The systematic employment of this procedure probably lessens the chances of the development of diphtheria, as well as of pneumonia through the extension of germs to the lungs. The distressing *cough* requires small repeated doses of deodorized tincture of opium, or, in older children, of morphine or codeine, enough being given to produce some result. When *bronchitis* is severe, the chest being full of small mucous râles and dyspnea present, the application of mustard-poultices, or even of dry cups may be helpful. Frequently repeated warm tub-baths are often beneficial, and belladonna is also useful in this connection. The patient should be propped up well in bed, as this renders respiration easier. *Pneumonia* should be guarded against by the careful avoidance of all exposure, especially after fever has disappeared, the supply of an abundance of warm, fresh, moist air, the rigorous confinement of the child to bed, the control of bronchitis, and the use of a nasal and pharyngeal spray. It is also advisable not to keep subjects with measles and bronchopneumonia in the same room with other patients suffering from measles alone, as the pulmonary condition tends to spread to the latter. In fact in hospital practise large numbers of cases of measles should never be together. Although the employment of the box-system does not as surely stop the extension of the disease in unaffected children as is the case in some other disorders, it certainly does limit the spread of complicating pneumonias from one measles-patient to another.

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[1] Arch. of Pediat., 1926, XLIII, 73.

Cardiac or general debility calls for the use of alcoholic or other stimulants. The hot mustard tub-bath is of especial service in the cases where retrocession of the rash shows the weakness of the heart. The development of *otitis* may be hindered by the use of the antiseptic spraying of the nose and throat referred to, combined with the constant wearing of a flannel cap over the ears.

Itching of the skin is relieved by the frequent application of a powder consisting of camphor 1 dr. (3.9); zincum oxidum ½ oz. (15.5); amylum ½ oz. (15.5) or of a 2 per cent. carbolized petrolatum. In the case of young children a 1 per cent. thymolated petrolatum is safer.

Nore detailed treatment of the various complications referred to, as well as of others not mentioned in this connection, will be found described in discussing these diseases. It may be remarked, however, that the frequency with which diphtheria associates itself with measles in institutions, and with which germs resembling the Klebs-Loeffler bacillus are found on the mucous membrane of cases of measles, even though without suspicious symptoms, justifies the preventative inoculation with antidiphtheritic serum which has been recommended.

During *convalescence* the diet may be increased in variety but confinement to bed should continue, except in the mildest cases, for at least 10 days from the beginning of the stage of invasion; certainly for several days after fever has disappeared; and to the room for 2½ to 3 weeks. The eyes often require careful protection for some time, and the bronchitis which frequently persists may need appropriate treatment. Before leaving the sick-room a disinfectant bath may be given (p. 271), but it is probable that this is entirely superfluous since the infection has usually disappeared before the desquamative period. Owing to the sensitiveness of the mucous membranes the first outing should be in good weather and the patient carefully protected. Tonics are often required at this period, among the most useful being iron, cod liver oil and strychnine. If health is not rapidly regained, a complete change of air to a warm, dry region is advisable, owing to the tendency to the development of tuberculosis as a sequel.

CHAPTER IV

RUBELLA

(German Measles)

The disease has very numerous synonyms. The term Rubella was first used by Veale[1] and appears to be that most suited to it. "Rötheln," a title formerly often applied to it, is a foreign word, and there is no need for its employment in English-speaking countries.

For many years the disease had been confounded with measles and with scarlet fever, but its independence is now fully recognized. It is probably the mildest of the commoner acute infectious fevers. The first clear description of its symptoms as we now know them was given by Heim[2] in Germany and by Maton[3] in England. Much earlier than this accounts were published of an affection claimed to be distinct from measles, yet clearly much more severe than, and different materially in many respects from, rubella as seen at the present day.

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[1] Edinb. Med. Jour., 1866, 404.

[2] Hufeland's Journal der pract. Arzneykunde, 1812, XXXIV, St. III, 60.

[3] Med. Transac. Col. of Phys., Lond., 1815, V, 149.

Etiology. Predisposing Causes.—Among these *age* is important. The disease is most common between the ages of 5 and 15 years, yet adults are frequently attacked, and F. Seitz[1] reports an instance in a woman of 73 years. Among 664 cases collected by J. Seitz[2] 45 (7 per cent.) were adults. Whether adults possess a relatively lesser degree of susceptibility is uncertain, although it has seemed to me probable. Satisfactory data are lacking on this point. Infants, especially under the age of 6 months, are much less frequently attacked.

Sex, race, and climate are not etiological factors. *Epidemic influence* is particularly marked, the disease being unusual, even in large cities, except in certain years when extensive epidemics occur. Season is not without influence, most epidemics appearing in winter and spring. Concerning the *individual susceptibility* there is a difference of opinion. Some regard the disease as only mildly contagious, many persons seeming immune. My own experience is that it is decidedly contagious, although less so than measles, and that a large proportion of children exposed will contract it. Older subjects, as stated, probably possess a greater degree of immunity.

Exciting Cause.—This is undoubtedly a germ, but its nature is entirely unknown. Sindoni and Ritossa[3] describe a gram-positive diplococcus found in the blood. *Transmission* is probably by way of the secretion from the eyes and the oral and respiratory mucous membranes, and possibly in the scales of the epidermis, although the latter is doubtful. Indirect infection through a third person, clothing, and the like, is perhaps possible, but certainly very unusual. Transmission by the air probably occurs as with measles, but regarding this there appear to be no data. Hess[4] attempted to transmit the disease to apes without success.

The *period of greatest infectiousness* is unknown. The disease is, without doubt, infectious during the stages of eruption and of invasion, and it seems likely during the latter part of incubation, but probably ceases to be so with the disappearance of the rash. Definite knowledge is lacking regarding the infectiousness during desquamation. It is doubtful whether it exists.

Pathological Anatomy.—There are no lesions characteristic of the disease. The eruption appears to depend upon a capillary hyperemia of the upper layers of the corium with slight inflammatory exudate (Thomas).[5] More exact studies have not been made.

Symptoms. ORDINARY COURSE. **Period of Incubation.**—One of the features of the disease is the variability of this period. The average range would appear to be from 1 to 3 weeks, although I[6] have seen it as short as 5 days. There are no symptoms characteristic of this stage, other than in some cases some enlargement of the posterior auricular, occipital, and superficial cervical glands.

Period of Invasion.—In the great majority of cases prodromal symptoms are either absent or so insignificant that they are overlooked.

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[1] Ref. Edwards, Keating's Cyclop. Dis. of Ch., 1889, I, 688.
[2] Correspondbl. f. Schweiz., Aerzte., 1890, XX, 369.
[3] La Pediat., 1925, XXXIII, 57.
[4] Arch. of Int. Med., 1914, XIII, 913.
[5] Ziemssen's Handb. spec. Path. u. Therap., B. II, H. II, 128.
[6] N. Y. Med. Rec., 1887, XXXII, 11; 37.

They usually consist of slight cough, sneezing, mild congestion of the mucous membrane of the nose and eyes, drowsiness, malaise, enlargement of the superficial cervical glands and sometimes slight fever. These last generally not more than 12 to 24 hours or less.

Period of Eruption. *Rash.*—Commonly without warning the child on waking in the morning is found to be covered by the characteristic rash. This appears, as a rule, first upon the face and spreads very rapidly over the body, covering it within a few hours or a day, and involving sometimes the soles and the palms. It consists of irregularly shaped, pale-rose spots, slightly elevated both to sight and to touch, and varying in size from an ordinary pin-head up to a split-pea. The spots are more or less closely placed, but not arranged in groups as in measles. They are for the most part discrete, but often show decided confluence on the face and on regions pressed upon and kept warm in bed, such as the nates and the flexor surfaces of the thighs. On the trunk the rash is usually paler than elsewhere and of a slightly brownish-red color. Very commonly the eruption passes rapidly like a wave over the body, having almost faded from the face, neck, and trunk by the time the full development is reached on the extremities 12 to 24 hours later; the acme on any one part lasting only from a few hours to half a day. This has been considered a characteristic of the disease. Nearly, or fully as often, however, in my experience, the eruption reaches its fullest development everywhere on the 2d day, after which it fades with great rapidity. The total duration of the rash upon the body equals 2 to 4 days, although it often lasts a much shorter time.

Other Symptoms of the Eruptive Stage.—Appearing with the eruption, or continuing in an accentuated form from the stage of invasion, when this is discoverable, are the trifling symptoms of the eruptive stage. They consist of moderate redness of the eyes, occasionally sneezing, sore throat, and slight hoarseness and cough. Decided coryza is absent. Often there are no catarrhal symptoms whatever, except the moderate affection of the throat, which is one of the most characteristic and constant manifestations. Sometimes this is not complained of, but on inspection the mucous membrane of the pharynx exhibits a diffuse redness, and the tonsils are generally swollen. In a considerable number of cases an eruption of yellowish-red or brownish-red spots (enanthem), of pin-head size, is visible over the soft palate, the uvula, and the mucous lining of the cheeks. This appears simultaneously with the rash upon the skin and lasts about half a day. Widowitz[1] claims to have found the buccal eruption of measles in 10 out of 135 cases of rubella (7.41 per cent.), and Müller[2] has also seen it repeatedly. The experience of observers in general does not corroborate this statement. I have seen 1 instance in which I believed it to be present. Occasionally, the cough and sore throat continue longer than the cutaneous eruption. Elevation of temperature is generally slight or absent. It reaches its maximum on the 1st or 2d day of the eruptive stage, and either falls suddenly while the rash is still at its height or diminishes more slowly as this fades. The fever seldom exceeds 101° to 102°F. (38.3° to 38.9°C.). Not infrequently a subfebrile temperature persists for some days after the rash has disappeared. The tongue is clean or slightly coated; never "strawberry" as in scarlet fever. Enlargement of the peripheral glands throughout the body, and especially of the superficial cervical and posterior auricular glands, is nearly always present and very characteristic, and often continues after the rash has disappeared. It is not, however, as diagnostic as once supposed, since it may occur in measles as well. Itching and edema are cutaneous manifestations occasionally seen, and roughness of the skin resembling cutis anserina is common. The bowel-movements and urine are unaffected, except for a slight febrile albuminuria in some instances or a diazo reaction often seen (Nagahara).[1] Vomiting is unusual. The studies of the blood, although not giving entirely uniform results, agree more or less in describing a leucopenia with an increase of lymphocytes during the eruptive stage. There is no increase in the number of eosinophiles. Hildebrandt and Thomas[2] found an increase in the mononuclear cells and a diminution of the neutrophiles most marked from the 4th to the 6th day. Spieler[3] observed an increase in the neutrophiles during incubation but a diminution of these and an increase of the lymphocytes in the stage of eruption, the condition in this respect resembling that seen in measles. Kaneko[4] likewise found lymphocytosis and leucopenia during the eruptive stage. Hess[5] noted definite increase of the number of lymphocytes before the eruptive stage. Very characteristic is the considerable number of plasma-cells present (Benzing;[6] Bensa[7]).

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[1] Wien. klin. Wochenschr., 1899 XII, 919.

[2] Münch. med. Wochenschr., 1904, LI, 98.

The duration and character of the general symptoms are, as a rule, proportionate to the persistence and intensity of the eruption, but to this there are numerous exceptions.

Period of Desquamation.—Spots of a faint brownish or yellowish color are often left after the eruption has faded. These persist 2 or 3 days. A very slight, branny desquamation resembling that of measles is common but by no means always present. It appears shortly after the disappearance of the eruption and continues 1 to 3 days.

Variations from the Ordinary Form.—The symptoms just described are what might be called typical. One of the chief characteristics of rubella, however, is its tendency to variations, many of which occur so frequently that they cannot be called unusual. On the other hand the severe epidemics described by the older writers and by a few more modern ones are to be considered as entirely anomalous.

The variable length of *incubation* is a normal and characteristic element of the disease. Epistaxis and sore throat have been recorded as occasionally seen in this period (Squire).[8] The *invasion* is sometimes unusually prolonged. I have known it to last 48 hours, and periods as long as 6 or 7 days have been reported. All such prolongations are decidedly exceptional. The prodromal symptoms may rarely be unusually severe. Among those detailed are decided coryza, vomiting, convulsions, delirium, bleeding from the eyes, ears or nose, edema of the face, dizziness, fainting, severe headache, rigors, croup, and various cutaneous eruptions.

The *eruption* is extremely prone to vary, not only in different epidemics, but in different cases in the same epidemic, and even in different parts of the body in one person. In fact, this multiform character is

[1] Jour. Orient. Med., 1923, I, 115.
[2] Zeitschr. f. klin. Med., 1906, LIX, 444.
[3] Wien. med. Wochenschr., 1915, LXV, 919.
[4] Jour. Orient. Med., 1923, I, 112.
[5] *Loc. cit.*, 913.
[6] Zeitschr. f. Kinderheilk., Orig., 1920, XVI, 12.
[7] Rivista di clin. pediat., 1922, XX, 513.
[8] Quain's Dict. of Med., Amer. Ed., 1885, 1382.

one of the greatest characteristics of the disease. Two types are especially noticeable, which are best designated as *Rubella morbilliforme* and *Rubella scarlatiniforme*. Both must be considered as entirely normal forms.

In **Rubella morbiliforme** the spots are fully the size of a split-pea and deeper colored than usual, exhibiting the tint of measles and being often characteristically grouped as in that disease. Such a rash cannot be distinguished from that of measles.

In **Rubella scarlatiniforme**, on the other hand, the eruption, at first macular and discrete, becomes by the 2d day widely confluent and is not perceptibly elevated. Careful examination will usually reveal a few macules in the general redness, especially on the brows, wrists and fingers. Nevertheless, in many cases a diagnosis from scarlatina, based upon the eruption, is entirely impossible, especially if the case was not seen at the onset. Filatow[1] believes the scarlatiniform type to be a distinct disease and applies to it the older German title "Rubeola scarlatinosa." I have, however, in institution-epidemics of rubella, observed some children with an eruption exactly simulating measles, and others

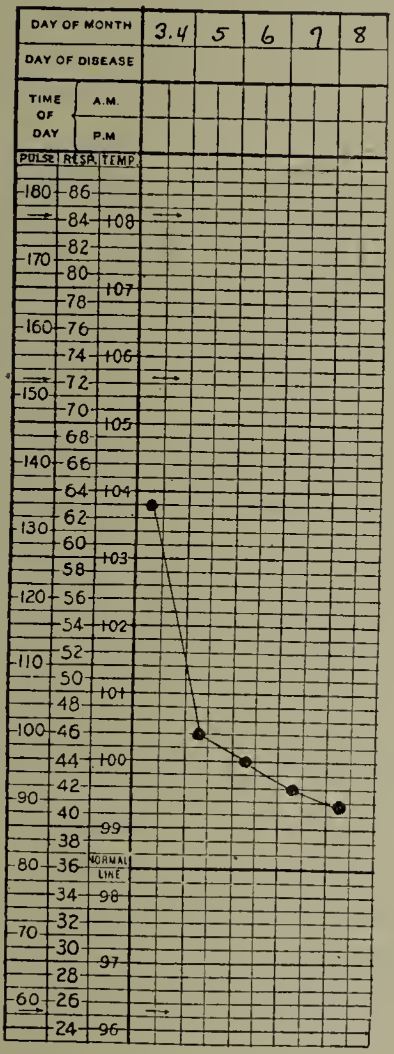

FIG. 74.

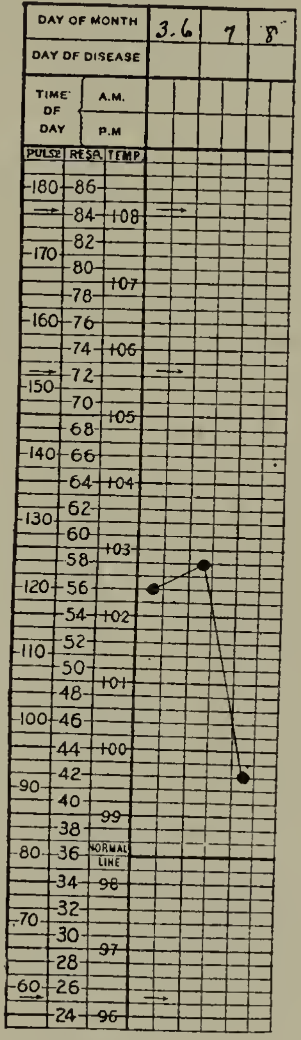

FIG. 75.

FIG. 74.—RUBELLA MORBILLIFORME.

Katie G., aged 5 years. Mar. 4, lachrymation, injection of eyes, drowsiness, twitching of limbs, red throat, numerous discrete pea-sized macules on face; Mar. 5, rash fading from face, abundant on body, deep-red, pea-sized, grouped as in measles and indistinguishable from that eruption, child brighter; Mar. 6, rash fading rapidly; Mar. 7, branny desquamation beginning.

FIG. 75.—RUBELLA SCARLATINIFORME.

Bertha L., aged 8 years. Mar. 6, eyes injected, throat red, abundant nearly uniform eruption over body suggesting scarlatina, but with macules visible in many places; Mar. 7, rash indistinguishable from scarlet fever except at wrists and ankles, throat red; Mar. 8, fading rapidly, macular character visible on legs, throat still quite red; Mar. 9, rash practically gone, branny desquamation beginning.

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[1] Arch. f. Kinderheilk., 1886, VII, 241.

with the most typical rubella scarlatiniforme. All gradations may be found between the two extremes. Sometimes the rash is scarlatiniform in some regions of the body and morbilliform in others. In general, the more elevated the rash, the greater the tendency to the morbilliform type; the less elevated, the more disposition to confluence. The 2 illustrative cases (Figs. 74 and 75), both rather severe, illustrate these two forms of the disease. The children were inmates of the same institution and were ill at the same time, in company with many typical cases of the malady.

Other peculiarities of the rash are occasionally seen. Vesicles, a purpuric eruption, a marbled appearance, and a sensation as of shot beneath the surface have been reported. I have seen the rash first appear as annular red spots. The sequence of its appearance may vary also, the trunk or the arms being first involved in some cases. Sometimes the eruption is limited to a certain region, especially the face and neck, lasting but a few hours only. It is probable also that the disease may rarely occur without eruption. At times the rash nearly disappears, to reappear after a brief interval, and in other cases there seems to be a true retrocession of the rash, with general symptoms more severe than usual.

Considerable variation may occur in the symptoms of the eruptive stage other than the rash. The catarrhal manifestations are in rare instances as marked as in measles. This is noticed oftenest in the rubeoloid type. Decided hoarseness is sometimes present and cough may occasionally be harassing. The tonsils may be sufficiently swollen to make deglutition difficult or impossible, and sometimes the follicles are engorged. Exceptionally the temperature may reach 103°F. (39.4°C.) or over, or vomiting may be severe. Delirium and convulsions have been observed.

The *desquamation* presents nothing varying from the type, except that in some cases it is usually abundant and prolonged.

Complications and Sequels.—These occur only rarely. Those oftenest seen are in connection with the respiratory tract. Severe *bronchitis* is sometimes witnessed, and *pneumonia* may occur as a complication or sequel. I have seen it follow in 2 instances. *Croup* and *pleurisy* have been reported. *Stomatitis* is an occasional complication. *Intestinal catarrh* is rare, although in a very anomalous severe epidemic reported by Cuomo[1] it was witnessed frequently. A secondary *sore throat* occurring rarely as a sequel has been recorded by several observers, and *albuminuria* and *nephritis* have been described as frequent in some entirely anomalous epidemics (Edwards),[2] but ordinarily are most unusual. *Arthritis* was seen by Wichman[3] in 18 out of 75 cases, and fatal purpura hæmorrhagica by Stratford.[4]

Other *infectious diseases* may occasionally be associated with rubella as complications or sequels. Among those recorded are typhoid fever, erysipelas, varicella, diphtheria and mumps. Among other complications and sequels reported, generally in anomalous cases, are thyroid enlargement, conjunctivitis, keratitis, otitis, endocarditis, icterus, urticaria, temporary paralysis, serous meningitis, and abscesses in different parts of the body.

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[1] Giorn. internaz. d. scien. med., 1884, VI, 529.
[2] Amer. Jour. Med. Sci., 1884, LXXXVIII, 448
[3] Hospitalstidende, 1898, VI, 921
[4] Lancet, 1911, II, 156

Relapse and Recurrence.—*Relapse* is very unusual. Isolated cases are recorded in medical literature. It occurred 3 times in 150 cases which I[1] have previously reported. It is oftenest seen from 1 to 3 weeks after the beginning of the first attack.

Recurrence is rare, one attack fully protecting from another. In 363 cases of rubella Widowitz[2] saw recurrence in but 1 instance.

Prognosis.—This is almost invariably good; certainly so unless the disease has assumed an entirely anomalous form. Certain epidemics have been reported, especially in earlier years, where the mortality reached from 5 to 9 per cent. All such occurrences are, however, very unusual. Death has generally depended upon complications, but occasionally upon the depressing effect of the disease itself. I have never seen a fatal case, although I have occasionally seen individuals decidedly ill with the disease.

Diagnosis.—The most important diagnostic signs are the variable duration of incubation, the short or absent prodromes, the slight degree of catarrhal symptoms and of fever, the presence of sore throat and enlarged superficial cervical, occipital and posterior auricular glands, and the characteristic discrete eruption. The combination of the last with insignificant attendant symptoms is very suggestive of the disease. The importance of a correct diagnosis is great, since an error may be sufficient to spread measles or scarlet fever through a house or a school. Yet owing to the variability of the symptoms this is seldom easy and, in sporadic cases, often impossible. There is no disease of which it is more true that the diagnosis must be based upon the study of the entire complex of symptoms.

Mild cases of *measles* resemble rubella very closely. The longer duration and greater severity of the prodromal symptoms in measles and the buccal eruption will be valuable diagnostic aids. Schick[3] considers a positive tuberculin-test evidence of rubella as against measles. A negative reaction is without value. It is, however, the distinguishing of mild *scarlatina* from the scarlatiniform type of rubella which is especially difficult. A longer period of incubation, the absence of vomiting, and the presence of slight catarrhal symptoms with but moderate sore throat suggest rubella; but the most important diagnostic symptoms are the presence of macules somewhere in the scarlatiniform rash, and especially upon the face, and the absence of any approach to the strawberry tongue. The Schultz-Charlton obliteration test (see p. 371) with normal serum may also be of service in doubtful cases, as this is never positive in rubella (Scheffer).[4] Later the differences in the desquamation may make the diagnosis clear. When these differential symptoms fail to render a decision reasonably certain, the only safe plan, with regard to other members of the household, is to make the provisional diagnosis of scarlet fever. When an epidemic of rubella is prevailing the problem is a much simpler one.

Treatment. Prophylaxis.—The decided contagiousness of rubella and the fact that it is communicated to others even before it is recognized, render prophylaxis exceedingly difficult. In view of this, and of the usual harmlessness and short duration of the affection, it is questionable whether isolation is worth while, if only the diagnosis has been made with certainty. The inconvenience attending separation of the sick children seems scarcely warranted by the insignificant nature of the disease. If it is determined to institute quarantine, the same course should be followed as in measles and probably for an equal length of time.

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[1] *Loc. cit.*

[2] Wien. klin. Wochenschr., 1909, XXII, 1596.

[3] Ergebn. d. inn. Med. u. Kinderheilk., 1910, V, 302

[4] Zeitschr. f. Kinderheilk., 1923, XXXV, 239.

Treatment of the Attack.—This is purely symptomatic and is, indeed, seldom needed. The patient should be confined to bed while fever lasts and given a light diet. Of course should the individual case or the prevailing epidemic be unusually severe, more energetic measures are required, both prophylactic and after the attack develops.

CHAPTER V

THE FOURTH DISEASE, INFECTIONS ERYTHEMA AND ROSEOLA INFANTUM

THE FOURTH DISEASE

(Filatow-Dukes Disease)

In 1900 Dukes[1] published the report of 3 series of cases which, he believed, showed the existence of a fourth eruptive fever in addition to measles, scarlet fever and rubella. He maintained that the affection generally believed to be the scarlatiniform type of rubella was in reality a distinct disorder. This was practically the view advanced by Filatow[2] in 1885, and which had earlier been suggested by Thomas.[3] Zahorsky[4] has observed 2 small outbreaks. The only reason for maintaining the independence of the affection was that it apparently did not protect from scarlatina. The arguments advanced by Dukes did not seem to me[5] at all convincing, there being no proofs given that the disease was not rubella in two epidemics and mild scarlet fever in the third. This has been practically the position maintained by Williams,[6] Caiger,[7] Pleasants,[8] Poynton,[9] and most of those who have given the matter close attention. Many physicians have accepted the new disease, but as yet no cases have been reported which prove convincingly that the affection protects from neither measles nor scarlatina, and this will be necessary before it can properly be given recognition. The question is consequently still unsettled.

ERYTHEMA INFECTIOSUM

This disease, although previously described by other observers, was first claimed to be an independent infectious disorder by Escherich[10] in 1896. Its position as a distinct affection still remains to be established, yet it certainly appears in no way to protect from scarlatina, measles or

[1] Lancet, 1900, II, 89.
[2] Arch. f. Kinderheilk., 1886, VII, 241.
[3] Ziemssen's Cyclop. Pract. Med. Amer., Ed., 1875, II.
[4] Amer. Jour. Dis. Child., 1924, XXVIII, 261.
[5] Phila. Med. Jour., 1902, April 12.
[6] Brit. Med. Jour., 1901, II, 1797.
[7] Brit. Med. Jour., 1901, II, 590.
[8] Phila. Med. Jour., 1902, May, 24, 938.
[9] Brit. Med. Jour., 1901, II, 594.
[10] Transac. 11th Internat. Med. Cong., 1896.

rubella. Excellent reviews of the subject are given by Escherich,[1] Shaw,[2] Tobler[3] and Stooss,[4] the last three accompanied by colored plates. Epidemics have also been described by Michalowicz,[5] Heisler[6] and Herrick.[7]

It occurs epidemically especially in spring and summer (Tobler, in winter) and attacks children generally between 4 and 12 years of age. Adults also contract it, but it is uncommon in infancy. It is only moderately infectious. The nature of the germ is unknown.

Symptoms.—The period of incubation, as far as yet determined, varies from 6 to 14 days. Initial symptoms are generally absent or consist only of malaise, discomfort, catarrhal manifestations and sore throat. The rash appears first on the face in the form of a rose-red efflorescence, occupying especially the cheeks, symmetrical in distribution, disappearing momentarily on pressure, and suggesting erysipelas. The skin is swollen and hot, and the edges of the affected area are generally slightly raised and sharply defined in an irregular line. There is no itching. The forehead and chin exhibit only discrete small patches of bluish-red color, and the temples, bridge of the nose and the oral circle are uninvolved. On the 2d day the eruption appears on the body, especially the gluteal region where it is always well-developed, and on the outer surface of the arms and legs, but only to a limited extent on the trunk. In these regions it is less intensely colored than on the cheeks and resembles rather the spots on the forehead. It is morbilliform in character, but more macular than papular, with some confluence on the extensor surfaces of the arms, while on the flexor surfaces of the arms and legs it is always less intense. The hands and feet are the last parts to be involved. In many regions on the extremities the rash has a map-like appearance or is crescentically grouped or forms annular spots with paler centers, especially while fading. This annular feature is particularly characteristic. On the trunk there are scattered discrete spots, pea-size or larger, sometimes grouped as described; or there may be no eruption whatever.

The rash fades from the face in 4 or 5 days, and disappears entirely from the body in 6 to 10 days from its first appearance. No pigmentation or desquamation follows. The mucous membranes exhibit no eruption.

There are few symptoms attending the eruption, the lymphatic glands are not swollen, there are no catarrhal manifestations, the urine is normal, slight fever is sometimes present, and the child generally feels perfectly well. The blood shows a normal or slightly diminished number of leucocytes for the first few days, but then develops a moderate leucocytosis with some increase of the eosinophiles (Stoos).[8]

In the matter of **diagnosis,** the disease is most likely to be confounded with rubella, although it is in reality quite unlike it. In the latter disorder the rash spreads uniformly over the body, is well-developed in the oral circle and on the forehead and exhibits either discrete small macules or a wide-spread confluence over the cutaneous surface. The lymphatic glands are enlarged. None of this is true of infectious erythema. Measles may resemble it closely, yet superficially, in the appearance of

[1] Monatsschr. f. Kinderheilk., 1904, III, 285.
[2] Amer. Jour. Med. Sci., 1905, CXXIX, 16.
[3] Ergebn. d. inn. Med. u. Kinderheilk., 1915, XIV, 70.
[4] Pfaundler and Schlossmann, Handb. d. Kinderheilk., 1923, 3 Aufl., II B, 235
[5] Przegl. pedj., 1909, IV–V. Ref. Jahrb. f. Kinderheilk., 1910, LXXI, 235.
[6] Münch. med. Wochenschr., 1914, LXI, 1684.
[7] Amer. Jour. Dis. Child., 1926, XXXI, 486.
[8] Pfaundler and Schlossmann, Handb. d. Kinderheilk., 1923, II, 239.

the eruption on the extremities. The involvement of the mucous membrane in this disease, the presence of buccal or palatal eruption, the distribution of the rash, and the fever and catarrhal symptoms serve to distinguish it. Scarlet fever has but slight resemblance. The general symptoms, the appearance of the throat and the distribution of the rash are characteristic in this disease. The medicamentous and other erythemata have little in common with infectious erythema, and the history, course and distribution of their eruptions aid in distinguishing them.

The **prognosis** is entirely favorable, there are no complications and sequels, and **treatment** is not required.

ROSEOLA INFANTUM

(Exanthema Subitum)

This eruptive fever, the infectiousness of which seems doubtful, has been described by a number of investigators (Levy;[1] Veeder and Hemplemann;[2] Westcott;[3] Greenthal;[4] Park and Michael;[5] Tso;[6] Ruh and Garvin;[7] Conrad;[8] Shima;[9] Glanzmann[10]). The first account of it was given by Zahorsky[11] who named it Roseola infantum. Veeder and Hemplemann called it Exanthem subitum, and Westcott Pseudo-rubella An excellent review is given by Heiman.[12]

Its nature is not certainly determined, but there is reason to believe that it is an independent disorder, since in a few of the reported cases the child had previously had rubella, measles and scarlet fever, or did so later. Infectiousness must be slight, since in none of the published cases has there been more than 1 case in a family. We have, however, observed an instance in which a second child in a family was attacked 5 days after the development of the disease in the first patient.

Etiology.—The cause is unknown. The majority of the cases occur in late spring. The *age* ranges from 6 months to 3 years, or occasionally up to 14 years. The first 2 years of life show the majority of the cases.

Symptoms.—The onset is abrupt with high fever, which continues for 3 days, generally without other symptoms except malaise. Occasionally the onset is attended by vomiting, and rarely by convulsions. Sometimes there is looseness of the bowels; but the appetite is good and there are no catarrhal symptoms other than, very exceptionally, congestion of the throat. Fever continues until the 4th day, or occasionally the 3rd or 5th, and then falls by crisis, and contemporaneously the eruption appears. This is generally seen first on the neck and spreads rapidly to the trunk, upper arms and thighs. The face and the limbs below the elbows and knees are little involved. Its height is reached in a few hours and it has disappeared by the end of 48 hours. The rash consists of abundant, pale, rose-red, macular or sometimes elevated, scattered macules from 1 to 5 mm. (.04 to .20 in.) in diameter, irregular in out-

[1] Jour. Amer. Med. Assoc., 1921, LXXVII, 1785.
[2] Jour. Amer. Med. Assoc., 1921, LXXVII, 1787.
[3] Amer. Jour. Med. Sci., 1921, CLXII, 367.
[4] Amer. Jour. Dis. Child., 1922, XXIII, 63.
[5] Amer. Jour. Dis. Child., 1922, XXIII, 521.
[6] China Med. Jour., 1922, XXXVI, 130.
[7] Arch. of Pediat., 1923, XL, 151.
[8] Virg. Med. Month., 1923, XLIX, 705.
[9] Jour. Oriental Med., 1923, I, 61, Ref. Jour. Amer. Med. Assoc., 1923, LXXXI, 422.
[10] Schweiz. med. Wochenschr., 1924, LIV, 589.
[11] Pediatrics, 1910, XXII, 60.
[12] Arch. of Pediat., 1925, XLII, 447.

line or less often round, and only occasionally confluent. A crescentic grouping is never present. A whitish areola may surround the lesions. Itching and desquamation are absent. There are no subjective symptoms whatever during this stage, the patient apparently feeling perfectly well. The blood, examined both before and during the eruptive period, according to Veeder and Hemplemann and confirmed by Glanzmann, shows a very decided leucopenia, sometimes as low as 3500, with a relatively high lymphocytosis. A decrease in the eosinophiles was noted by Shima.

The **prognosis** is entirely favorable.

Diagnosis.—The chief diagnostic features are the sudden onset, the comparatively long febrile period, the absence of catarrhal or other symptoms, the blood-findings, and the character of the eruption as described. *Scarlet fever* is distinguished by the angina, strawberry-tongue, leucocytosis, early appearance of the eruption, and desquamation. *Rubella* shows little if any elevation of temperature, early development of the rash, red throat, and enlargement of the lymphatic glands. *Measles* is promptly differentiated by the marked catarrhal symptoms and the presence of the Koplik spots.

Treatment other than symptomatic is not required.

CHAPTER VI

VARIOLA

(Small-pox)

Small-pox is now seen but seldom in children where the practice of vaccination is prevalent, yet some discussion of it is requisite on account of the danger of confounding its modified form (varioloid) with varicella.

It has existed since earliest times in Asia and since at least the 7th century in Europe. The best early description of it was by Rhazes[1] in the first part of the 10th century. It appeared in America in the 16th century.

Etiology. Predisposing Causes.—Race, climate and locality exert no influence, the disease being spread over all parts of the earth. Sex and age, too, do not affect its occurrence. The infant may be born suffering from the disease, or having already passed through an attack, or it may develop it soon after birth as easily as at any later period of life. The individual susceptibility is very great, and the majority of unprotected persons exposed contract the disorder. Yet an immunity, temporary or permanent, is found occasionally to exist. The affection is particularly prone to appear in large epidemics, which, at least in temperate climates, reach their maximum during the cold months of the year.

Exciting Cause.—The disease is one of the most contagious known, yet the nature of the germ has never been positively determined. A protozoan (the cytoryctes variolæ) has been described by Guarnieri;[2]

[1] De variolis et morbilis, Lond., 1756.

[2] Centralbl. f. Bakt., 1894, XVI, 299.

Councilman, Magrath, and Brinckerhoff;[1] and others; but it is claimed by Proescher[2] and others that these bodies are only proteid end-products. Very minute organisms have been found which seemed to be concerned in the causation of the disease, but it is doubtful whether they are filterable (MacCallum).[3]

Transmission.—The transmission of the germ by inoculation was long made use of in "variolation." It is communicated also by direct contact, and, as generally believed, to an unusual degree through the surrounding air, from a distance of several hundred yards. This is, however, denied by many. It is readily carried by clothing, insects and the like, or by a third, healthy person. The infection certainly resides in the pustules and the crusts. It does not appear to be contained in the mucous secretions unless contaminated by pustular discharge. The blood, however, has been proven to be infectious and it is also evident that the disease can in some way readily be communicated before the eruption appears. Such a thing as a healthy small-pox carrier does not exist, in the sense in which the term is used for diphtheria and typhoid fever. Danger of infection by the patient ceases by the time the crusts have fallen and the skin has become smooth. The *tenacity of life* of the germ is very great and may continue even for some years. The poison is generally received by way of the respiratory tract and sometimes by the digestive.

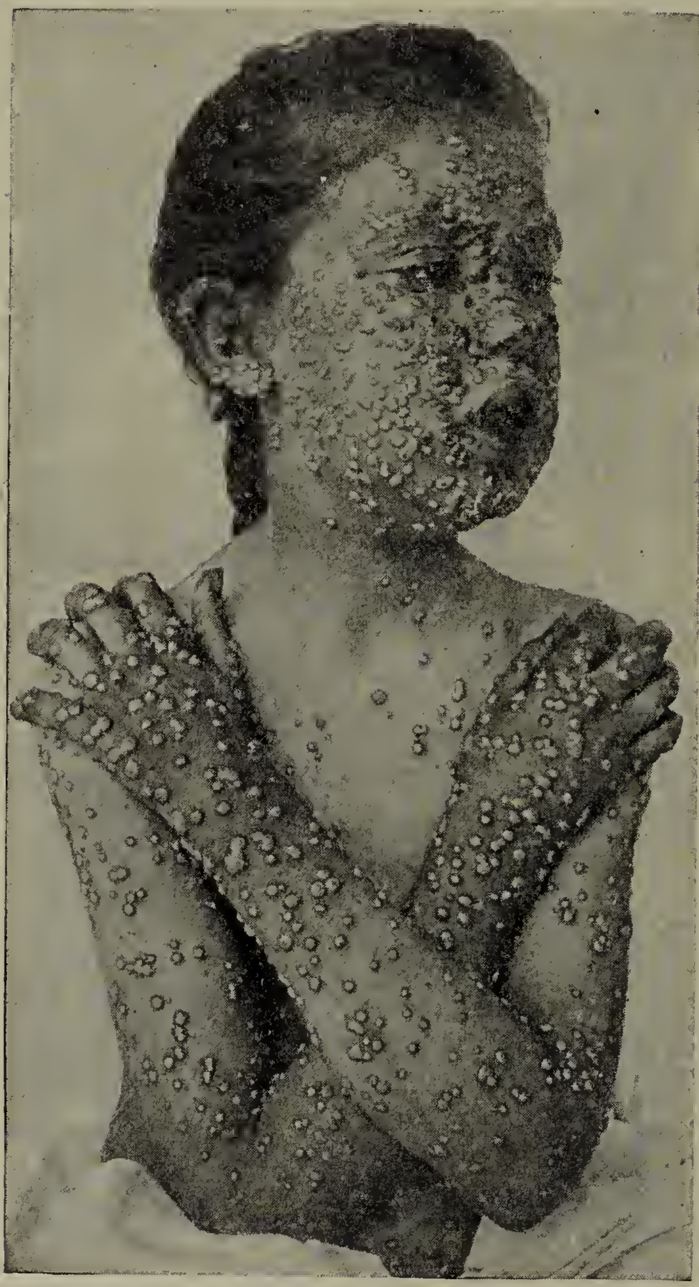

Fig. 76.—Discrete Small-pox.

Occurring in an unvaccinated girl. 8th day of eruption. (*Welch and Schamberg, Acute Contagious Diseases*, 1905, *Pl. XV*, *opp. p.* 177.)

Pathological Anatomy.—The earliest step in the formation of the pock is a circumscribed hyperemia followed by a necrosis of the epidermal cells, beginning in the Malpighian layer. The cells now fuse into a reticular framework, as seen later in the vesicle, and transudation of serum takes place into the lesion, with a proliferation of cells in the periphery. The combination of these changes forms the *papule*. Continued transudation of fluid produces the *vesicle*. Most investigators believe that umbilication results from the holding down of the central portion of the surface of the vesicle by the reticular bands beneath it, while the cellular proliferation of the periphery raises this portion higher. The increasing tension later tears away the reticulum and the umbilication disappears. Effusion of blood may take place into the lesion. Leucocytes in constantly increasing number enter the vesicle and a *pustule* results. They may penetrate too, the tissues forming its base, until the whole thickness of the skin is involved. If this occurs, *scarring* follows.

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[1] Jour. Med. Research, 1903, IX, 372; 1904, XI, 12.
[2] New York Med. Jour., 1913, XCVII, 741.
[3] Medicine, 1926, V, 59.

Analogous changes take place in the mucous membrane of the mouth, throat, eyes, nose, larynx, trachea, bronchi, esophagus, rectum, vagina, ureter, and urethra. Sometimes a pseudo-membrane covers the ulcers produced. Petechiæ may be present on the mucous membrane as well as on the skin.

Hemorrhage may be discovered in the serous membranes, the various organs, the muscles, and the medullary cavity of the bones. This is especially true of the hemorrhagic type of the disease. Hypostatic pulmonary congestion and bronchopneumonia are common post-mortem lesions. In cases which have passed into the suppurative stage, parenchymatous degeneration of the liver, heart and kidneys is found, and the spleen and lymphatic glands are enlarged.

Symptoms.—The symptoms vary to such a degree with the type of the disease, that it is more convenient first to describe the ordinary form (Variola vera) and then the modifications which may arise.

(*A*) VARIOLA VERA. DISCRETE SMALL-POX (Fig. 76).—In this, the usual form of small-pox, the lesions are discrete throughout, or confluent on the face and hands only.

Period of Incubation.—This is usually 12 to 14 days, although it may vary from 5 up to 20 or more. As a rule no symptoms are present, but occasionally the last days of this period are marked by malaise, dullness, headache, and loss of appetite.

Period of Invasion.—The onset is generally sudden, with repeated vomiting, prostration, headache, pain in the back, limbs, and epigastrium, loss of appetite, coated tongue, thirst, high fever and rapid pulse. Convulsions often usher in the attack in early life. A chill may occur in older children. Diarrhea may replace the constipation present in adults. The temperature rises very rapidly to 103° or 104°F. (39.4° or 40°C.) or more. There is restlessness, either sleeplessness or drowsiness, and often delirium.

On the 2d day the pain in the back and head and the rapid pulse persist, and occasionally the convulsions, vomiting and abdominal pains also; and the temperature continues unaltered or even rises. Not infrequently, but less often in children, a *prodromal rash* is observed, which is scarlatiniform or morbilliform in type. The scarlatiniform rash is generally limited to the inner surface of the thighs, the lower part of the abdomen, the axillæ, and the sides of the chest and, according to Tsurumi and Isono,[1] the outer side of the upper arms; but sometimes covers the entire surface and in color suggests erysipelas. The morbilliform eruption is irregularly distributed, either in limited areas or more widely spread. The prodromal rash may disappear before the characteristic eruption of the disease develops, or may last for a time after its appearance. Petechiæ are sometimes seen in cases destined to be hemorrhagic. The duration of the period of invasion is from 2 to 3 days. The general symptoms of the 2d day continue unabated until the eruption begins to develop.

Period of Eruption. *The Rash.*—On the 3d or sometimes the 4th day from the onset of the initial symptoms the characteristic eruption begins to show itself. The *individual lesion* consists at first of a red macule which gradually becomes more prominent and is transformed into a hard, elevated papule by the 2d day (the 4th of the disease). It now gives the often-described sensation as of a shot beneath the skin. On the 3d day of the eruption (the 5th of the disease), a minute vesicle appears in the centre and grows slowly to the 5th or 6th day, when it is as large as a good-sized split pea, very firm, of a mother-of-pearl color, circular, slightly flattened on top, often with a central umbilication, and with a narrow red areola. Pricking with a needle allows a small quantity of serum to exude, but the vesicle does not collapse; that is to say, it is multilocular.

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[1] Jour. Infect. Dis., 1921, XXIX, 109

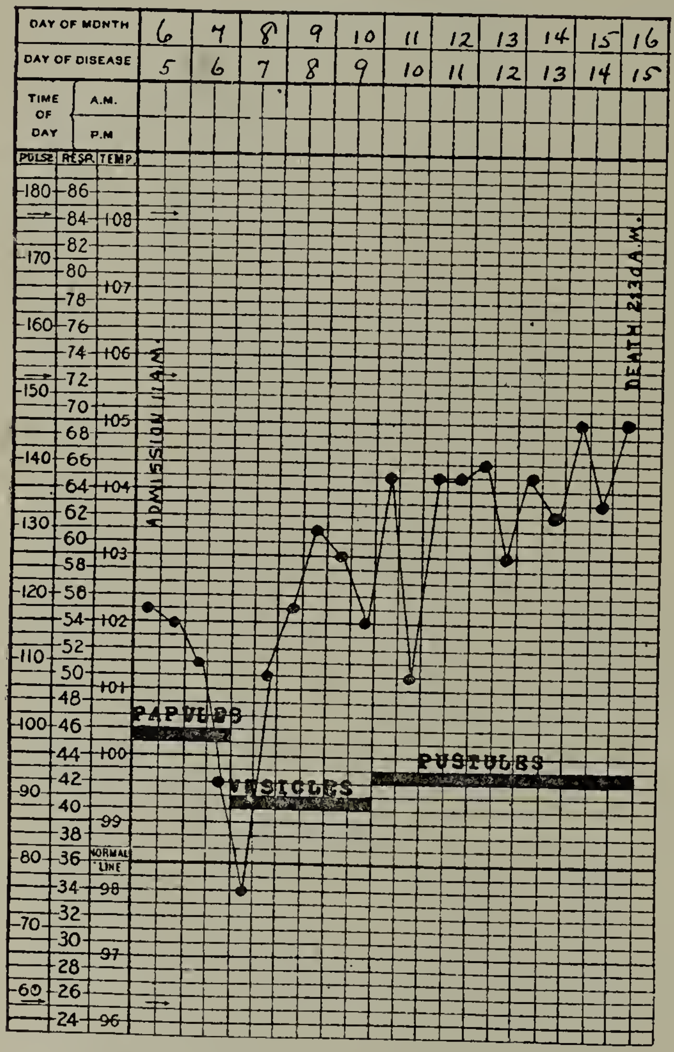

Fig 77.—Discrete Small-pox with Typical Course.

Emily E., aged 5 years. Never vaccinated. Mar. 2, nausea, headache, aching in legs and back, fever; Mar. 5, amelioration in symptoms, papular rash developing on face; Mar. 7, decided fall of temperature, vesicles appearing; Mar. 10, rise of temperature with pustular stage. From a patient in the Philadelphia Hospital for Contagious Diseases. *Courtesy of Dr. B. Franklin Royer.*

By the 6th day of the eruption (the 8th of the disease) suppuration begins and proceeds rapidly, and by the 8th or 9th day is at its height, the vesicle having become a yellow, entirely opaque pustule, of globular form without umbilication, and surrounded by a very distinct areola with much swollen skin about it. The pustule may rupture in a day or more or may remain unruptured, while a secondary umbilication develops.

The *regions first attacked* are generally the face and wrists whence the rash extends to the head, hands, and arms, and in 24 hours to the trunk and then to the lower extremities, occupying about 3 days before it reaches its fullest extent. By this time the entire surface, but especially the face and head, is well covered, the rash having become vesicular in the region first attacked, while still macular or papular elsewhere. The parts of the body constantly exposed to the air show the greatest number of lesions, the trunk being nearly always least involved. In adults the distal portions of the limbs are more affected than the proximal, but this is less well-marked in children. There are more lesions above than below the umbilicus, and the flanks are usually clear. The pit of the axilla is uninvolved. The lesions are separated from each other by skin that is normal except for the swelling, which is especially marked on the face, hands and feet. In some cases the pocks appear everywhere simultaneously; in others first on some other regions than the usual ones. They may number some hundreds in an average case. Those upon the palms and soles consist at first of macules surrounded by an indurated area. They become flattened, deep-seated vesicles, but do not pass through a papular stage.

Simultaneously with the cutaneous eruption, or a little earlier, lesions may be found on the mucous membrane of the mouth, pharynx, nose, and sometimes the larynx, vagina, rectum and eyes. These run a course similar to those upon the skin, except that the vesicles soon rupture and leave ulcerated surfaces.

Symptoms Attending the Eruption.—With the appearance of the rash upon the skin, or shortly after or before it, there is usually a sudden or gradual fall of temperature to normal or nearly so, and the constitutional symptoms improve greatly (Figs. 77 and 80). When suppuration begins on the 6th day of the rash, the temperature gradually rises again with morning remissions. This is called the "secondary" or "suppurative" fever. Then follows increase of temperature until about the 11th or 12th day of the eruption when it falls by lysis in favorable cases, the elevation reached being generally less than in the stage of invasion. With the onset of the secondary fever the pulse and respiration again become rapid, the heart often weak, and prostration decided. There is severe pain, swelling, tension, and itching of the face, neck and extremities. The eyes may be closed and nasal respiration obstructed; the face so swollen that the patient is unrecognizable. Restlessness, sleeplessness, and delirium are common. The throat is sore and swallowing difficult, thirst excessive, the tongue parched, the breath foul, the teeth and gums covered with sordes, diarrhea may occur, and conjunctivitis and cough are common. The urine often contains albumin and casts. The discharging pus from ruptured lesions produces a very offensive odor. The pain which the swelling of the skin occasions at this period is often intense and every movement or even the pressure of the bed or the bed-clothes may be the cause of great suffering.

The blood in small-pox exhibits a diminution of hemoglobin early in the disease. The red blood-corpuscles tend to form irregular clumps instead of rouleaux. Magrath, Brinckerhoff and Bancroft[1] confirm the observations of previous investigators, that a varying degree of leucocytosis, especially of the mononuclear cells, is present during the eruptive stage. Hoffman,[2] in a careful study of the blood in 300 cases, found a slight leucopenia during the prodromal period; but with the eruption there is a rapid increase in the white cells, averaging 17,000 from the 9th to the 14th day. The increase is especially in the lymphocytes, which reach from 45 to 65 per cent. The eosinophiles are increased to from 3 to 10 per cent. in the first 3 weeks, and then increase to from 15 to 20 per cent. A few myelocytes are present. The normal conditions begin to be approached in the 4th week but are not entirely attained until after 6 weeks.

Period of Desquamation.—Drying begins about the 11th or 12th day of the eruption, generally first in the lesions which were first to appear. By the 14th or 15th day, in average cases, this process is well under way, and the crusts begin to fall. Many, however, are very adherent and do not separate until during the 4th week or later, unless removed forcibly. Following the separation a branny desquamtaion occurs. On the hands and feet the entire skin may be shed in the form of moulds. After desquamation is over there remains a reddish pigmentation, later becoming brownish. The normal color is regained only after weeks or months.

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[1] Jour. Med. Research, 1904, XI, 247.

[2] Jour. Trop. Med. and Hyg., 1923, XXVI, 217. New York Med. Jour., 1923, CXVIII, 616.

With the beginning of desiccation the swelling and pain in the skin lessen, the areolæ about the pustules grow smaller, the temperature begins to fall and the symptoms in general improve. The itching of the skin is now frequently intense. The hair often falls out temporarily. Feebleness of body and of mind may persist for several weeks and disappear only slowly. Any scarring left by the disease does not assume the characteristic white appearance for 3 or 4 months.

The principal well-defined modifications of the type of the disease are (*B*) *Confluent small-pox,* (*C*) *Hemorrhagic small-pox,* (*D*) *Mild small-pox,* (*E*) *Varioloid,* or *small-pox modified by vaccination.*

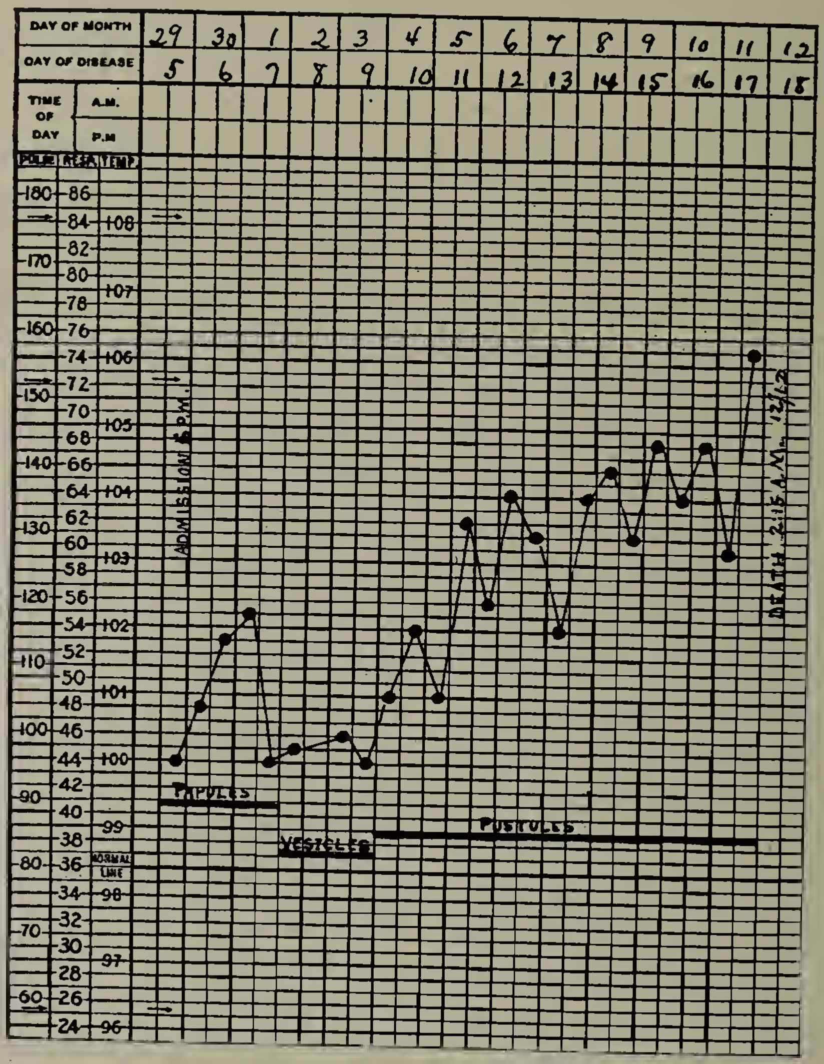

FIG. 78.—CONFLUENT SMALL-POX.

Adult. Never vaccinated. June 25, very intense headache and backache, with dizziness, vomiting, chills, and high fever; June 28, been very ill and in great pain, wide-spread papular eruption appeared this date and spread rapidly; July 1, vesiculation began, lasted 3 days, became confluent on face, legs, wrists, and forearms; July 4, pustulation began, great pain and high fever which lasted until death. From a patient in the Philadelphia Hospital for Contagious Diseases. *Courtesy of Dr. B. Franklin Royer.*

(*B*) CONFLUENT SMALL-POX.—This form is characterized by the tendency of the lesions to fuse, generally with an increase in severity of all the constitutional symptoms. The prodromes are the ordinary ones, but always severe. The eruption appears at the usual time or often somewhat earlier, and spreads with great rapidity. The lesions are closely crowded, and the swelling and redness of the skin come on early. Sometimes the face assumes a general redness at the beginning of the eruptive period instead of exhibiting the ordinary macules. In the suppurative stage many of the pustules fuse into irregularly shaped suppurating areas, especially on the face and extremities, but on the trunk they continue discrete. The entire face may be covered by one enormous pustule. The involvement of the throat is usually extensive.

The subsidence of symptoms and the fall of temperature seen as the eruption is developing are less than in discrete small-pox. Then, with the onset of suppuration or before it, all the general symptoms are manifested in great force. Vomiting and retching may be continuous, the cervical lymphatic glands are much enlarged and the temperature is high with little remission (Fig. 78). The symptoms in general are those of intense toxemia. The crusts which form later are slow in being shed.

(*C*) HEMORRHAGIC SMALL-POX.—This is the malignant form of the disease. The incubation is often very short. The hemorrhagic tendency may develop either in the prodromal or in the eruptive stage. In the former, called **purpura variolosa** or "black small-pox," the symptoms are violent and a purpuric rash appears, at first petechial and later ecchymotic. Much of the surface of the body may exhibit an almost uniform purplish color. Hemorrhage from various mucous membranes may occur. Generally the true eruption of small-pox does not develop; or, if the patient live until the time of the eruptive period, is represented by a few papules only. There may be delirium or stupor, but often the mind remains clear throughout. This is the most malignant form of the disease.

In the second variety of hemorrhagic small-pox, called **variola pustulosa hæmorrhagica,** the earlier stages of the disease are severe, but there is nothing characteristic until the vesicles or pustules begin to form. The hemorrhagic tendency then develops either slowly or rapidly. Bleeding takes place into the lesions and, in the still severer cases, into the surrounding tissues and from the mucous membranes. The mind usually remains clear.

(*D*) MILD SMALL-POX.—Individual cases, or even epidemics, occur in which, although there has been no protection by vaccination, the course of the disease is extremely moderate. The lesions are few in number or entirely absent (**Variola sine eruptione**), and the constitutional symptoms mild or insignificant. Occasionally the prodromes are of the usual severity, but the development of the eruption is abortive, and the whole course of the disease greatly curtailed, the condition being entirely similar to varioloid.

(*E*) VARIOLOID; MODIFIED SMALL-POX.—This is the form observed in persons partially protected by vaccination. It is characterized by its mildness and the comparatively small number and rudimentary nature of the lesions. The disease, however, does not differ in nature from ordinary small-pox and is as capable of producing the unmodified form in unprotected persons. The stage of invasion is more variable than in discrete small-pox, ranging often from 1 or 2 days up to 4 or 5 days. The prodromal symptoms may be the ordinary ones of small-pox or may be insignificant and even overlooked. A prodromal rash is frequent, especially of the rubeoloid form. The distribution of the lesions is very variable. Sometimes only about half a dozen may be found, or even exceptionally none at all. They are often confined to the face and hands. In other cases they may be quite numerous or may even be confluent or partially so. As a rule they develop first on the face and spread rapidly. Their first appearance is that of red macules, suggesting the roseola of typhoid fever. These quickly change into papules and then some of them into vesicles, while others never pass the papular stage. Many of the vesicles do not suppurate but rapidly dry into crusts; others become purulent, but are superficial, soon dry, and rarely leave scars. The entire crop of lesions also develops more rapidly than in unmodified small-pox and is present within 12 hours from the first evidence of eruption. There are no large areolæ and no severe swelling of the skin. With the first appearance of the eruption the initial fever falls rapidly by crisis and is seldom again elevated, the suppurative fever being absent and all symptoms disappearing. The whole process is much shorter than in variola vera and the desiccation begins earlier and is soon completed (Fig. 79).

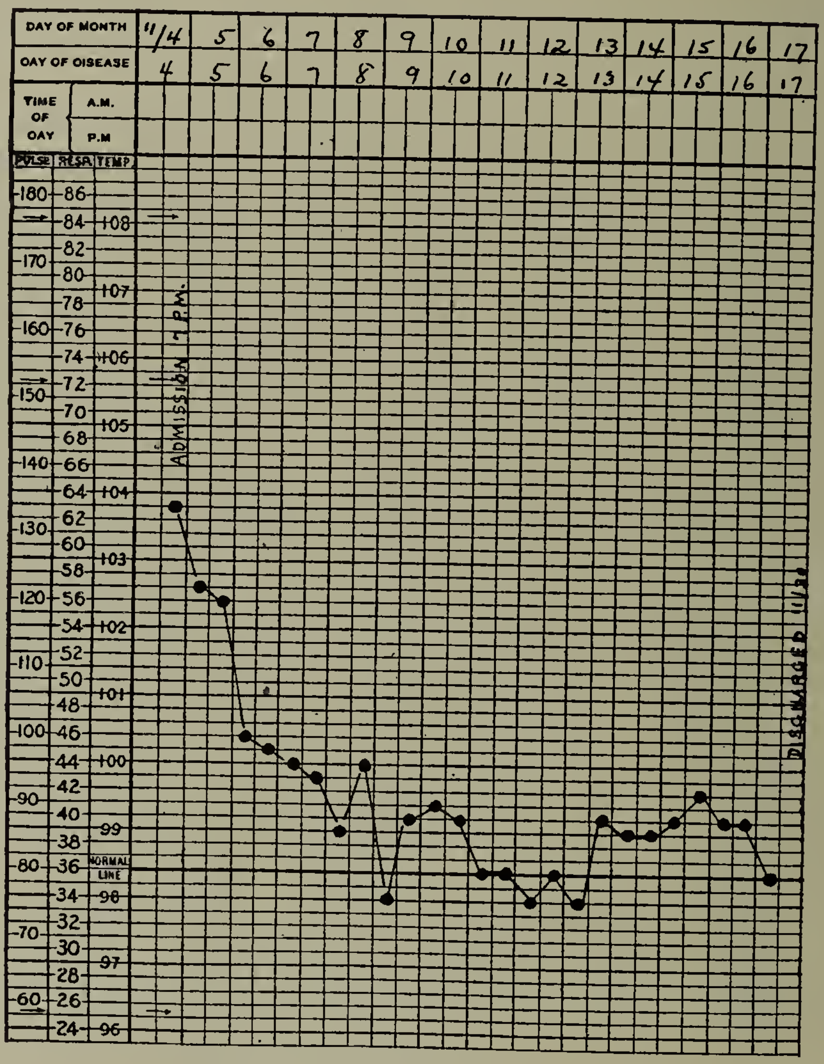

FIG. 79.—VARIOLOID.

Adult. Vaccinated in youth. Nov. 1, headache and backache, high fever, nausea, dizziness; Nov. 3, symptoms continued until this date, when generalized papular eruption developed; Nov. 4, vesicles developed, but nearly all dried in this stage; a few pustules without secondary fever. From a patient in the Philadelphia Hospital for Contagious Diseases. *Courtesy of Dr. B. Franklin Royer.*

Complications and Sequels.—Affections of the respiratory apparatus are among the most frequent complications of variola. Laryngitis is common, and in severe attacks involvement of the cartilages may occur, or edema of the glottis cause death. Bronchitis is invariable in severe attacks of small-pox and bronchopneumonia is almost always present in fatal cases. Lobar pneumonia is uncommon, but pleurisy, especially of the purulent form, is frequent. A pseudodiphtheritic inflammation of the throat is not uncommon. Cardiac complications are rare, myocarditis occurring oftener than other forms. Otitis is observed occasionally and conjunctivitis, keratitis, and iritis not infrequently, sometimes followed by more or less permanent impairment of sight. Albuminuria is common, but nephritis is rare. Multiple cutaneous abscesses are of frequent occurrence as sequels (Fig. 80). Bedsores or gangrene may develop, and suppurative adenitis is not infrequent. Osteomyelitis may be a sequel, and a special necrosive, non-suppurative form has been described, oftenest occurring in childhood, which frequently destroys the epiphyses (Brown and Brown).[1] Among nervous disorders neuritis is an occasional sequel and myelitis, encephalitis and meningitis may occur as complications.

Small-pox may coexist with, follow or precede other acute infectious diseases but this appears to be very exceptional. Erysipelas may develop as a sequel during convalescence.

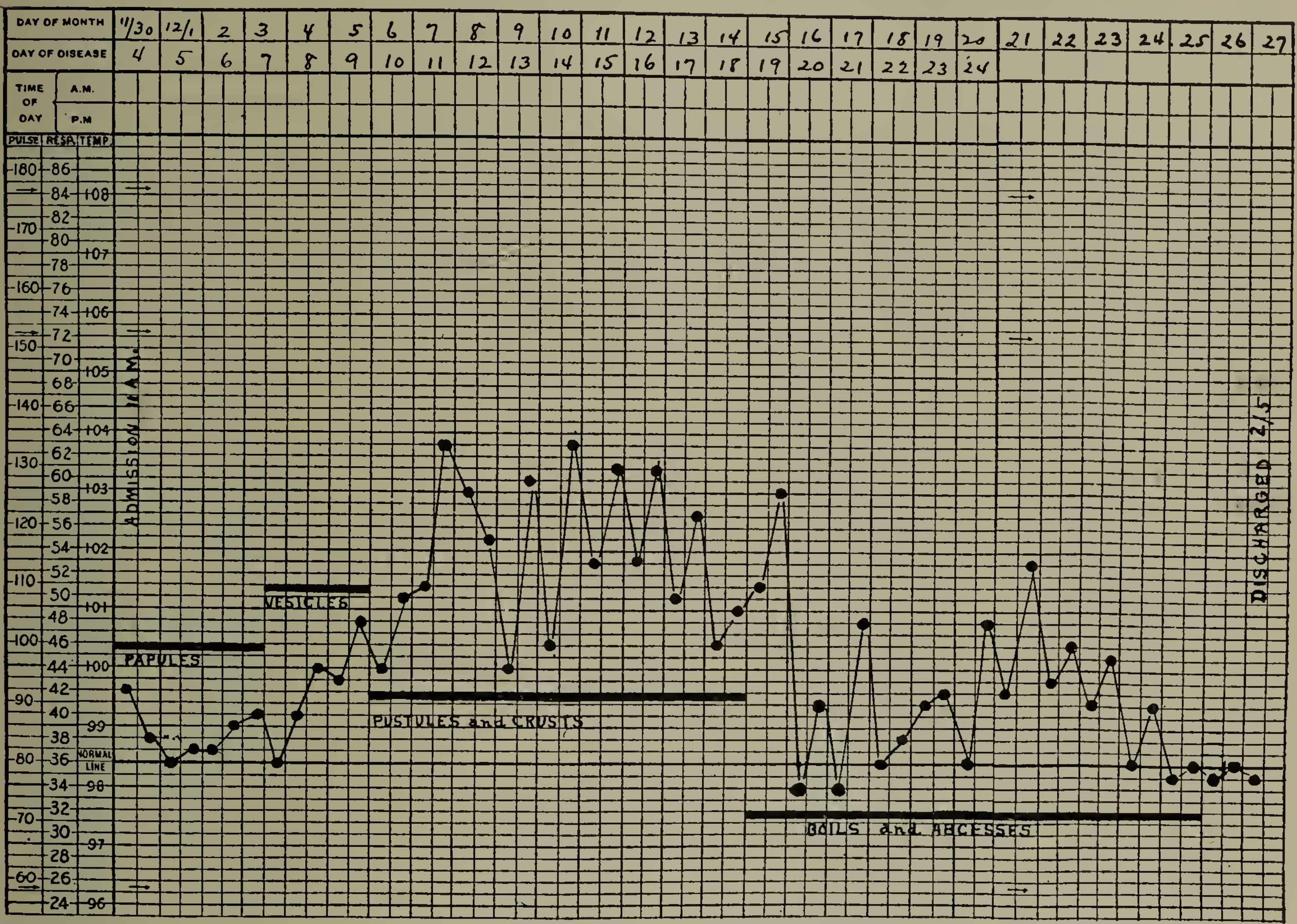

Fig. 80.—Discrete Small-pox Followed by Boils and Abscesses, Prolonging the Temperature.

Adult. Usual prodromes began Nov. 27 and patient was severely ill for 3 days; papular eruption on Nov. 30, with rapid subsidence of all symptoms; boils and abscesses appeared on Dec. 16. From a patient in the Philadelphia Hospital for Contagious Diseases. *Courtesy of Dr. B. Franklin Royer.*

Relapse and Recurrence.—*Relapse; i.e.* a second attack developing before the poison is out of the system is rarely observed. *Recurrence* of small-pox is sometimes seen, but is very uncommon. As a rule one attack confers a life-long immunity, and a second occurrence is usually mild. Vaccination gives an immunity which may be permanent or only temporary. (See Vaccination, p. 430.)

Prognosis.—The mortality of unmodified small-pox is high, varying from 25 to 45 per cent. or more. The character of the epidemic is one of the important factors. In some outbreaks the virulence is but slight, and few cases die. Various causes influence it. In infancy and early childhood the number of deaths is very large. Statistics of the Hospital for Contagious Diseases of Philadelphia (Welch and Schamberg)[2] gave a mortality of 61.66 per cent. in subjects under 1 year, as compared with a total mortality of 31.79 per cent. A mild initial stage generally presages a mild attack. Severe initial symptoms, however, do not necessarily indicate that the attack as a whole will be severe. Death may occur in any stage of the disease. In many cases the initial symptoms are so grave that children die before the eruptive stage develops. Danger again arises during the suppurative period, and it is at this time, about the 11th or 12th day of the disease, that the majority of deaths take place. A wide-spread, intense, scarlatiniform prodromal eruption is an unfavorable symptom, often indicating the onset of hemorrhagic small-pox. The morbilliform prodromal rash is rather a good omen The presence of petechiæ during invasion is generally, but not necessarily, an unfavorable indication. A fall of temperature occurring promptly with the development of the rash promises a mild attack, while unusually high fever during the period of eruption is unfavorable and may signify that a fatal ending is immediately impending. The sooner the suppurative fever begins to fall the better the prognosis and the less danger there is of threatening complications developing. Severe nervous symptoms during the eruptive period are an unfavorable indication, as is an abundant eruption on the mucous membrane of the mouth and throat. The danger of the disease is usually in direct proportion to the number of pocks and the amount of suppuration. Confluent small-pox gives a very high mortality, especially under 5 years of age. The great majority of hemorrhagic cases die. The prognosis in modified small-pox is nearly always good, depending upon the degree of protection which vaccination has afforded.

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[1] Jour. Amer. Med. Assoc., 1923, LXXXI, 1414.

[2] Acute Contagious Diseases, 1905, 275.

Diagnosis.—In well-developed small-pox the diagnosis can scarcely fail to be made. Early in the disease, however, or later in very mild cases, and especially in the modified form, it may be very difficult. The characteristic diagnostic symptoms are the severe pain in the head and back in the initial stage, the sudden drop of temperature about the beginning of the eruptive period, and the well-marked papular stage through which the eruption passes. With great frequency a diagnosis of *grippe* is made at the onset. Early in its development the rash has often been mistaken for *typhoid fever.* At this period, too, it may strongly resemble *measles,* and errors in diagnosis have frequently been made. The absence of the catarrhal symptoms of measles, however, and of the Koplik spots aid in excluding this. The morbilliform prodromal eruption of small-pox may likewise suggest measles. This eruption, however, is not at all elevated as is the rash of measles. The scarlatiniform prodromal rash may lead to the suspicion of *scarlet fever.* The absence of initial sore throat, and the localization of the eruption elsewhere than first on the face and chest, serve to exclude the latter disease. Further, the prodromal rashes are not common in children. I have seen a *generalized vaccinal eruption* occasion confusion. The development of the rash at the same time with the vaccine vesicle and the absence of all the characteristic initial symptoms of small-pox render the diagnosis generally easy. A differential point given by Stalnaker[1] is that in generalized vaccinia there is never the involvement of the mucous membranes which is so constantly observed in small-pox. Difficulties will necessarily be present in cases in which the vaccination-pocks of vaccinia occur in combination with those of small-pox (Fig. 81). The *pustular syphiloderm* also may be mistaken for small-pox. The slower onset and course, the appearance of the eruption in successive crops, the history, and the presence of other evidences of syphilis aid in recognizing this affection. *Varicella* occurring in a severe form is the disease causing the greatest diagnostic difficulty. This is especially true in the exceptional cases where the stage of invasion of varicella has been unusually long or well-marked. So, too, mild varioloid may closely simulate ordinary varicella. In varicella, however there is a rapid development and drying of the vesicles, and in small-pox, even in the modified form, the invariable presence of a papular stage and slower desiccation. In varicella, again, is the occurrence, side by side, of lesions in all stages of development, and in variola the existence of but a single crop in any one locality. The rash of varicella is most abundant on the trunk, especially on the back; that of variola on the face and the distal parts of the extremities. The lower leucocyte count of varicella is a distinguishing feature. These differences make the diagnosis generally clear. Yet in spite of them mistakes are frequent and easily made.

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[1] U. S. Naval Med. Bull., 1922, XVI, 290.

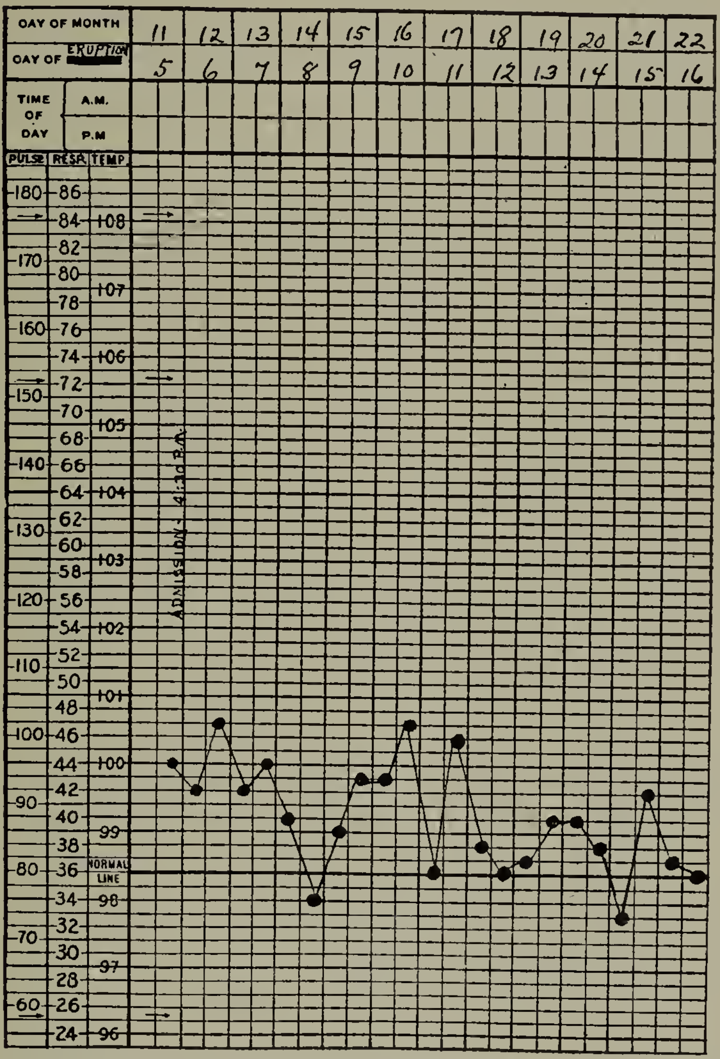

FIG. 81.—MODIFIED SMALL-POX AND VACCINATION OCCURRING TOGETHER.

Philip C., aged 7 months. Exposed to small-pox from Oct 24 to Nov. 1. Vaccinated Nov. 1; began to take actively by Nov. 4; eruption of small-pox appeared Nov. 7, but dried in vesicular stage. Fever probably due in part to active vaccination. From a patient in the Philadelphia Hospital for Contagious Diseases. *Courtesy of Dr. B. Franklin Royer.*

There are a number of names given to mild small-pox in different countries, among them such titles as Alastrim, Amaas, Kaffir-pox, Cuban Itch, Glass-pox and Milk-pox. Some have considered these to be distinct diseases, but most authorities deny this (Painton;[1] Hoffman;[2] Hill[3]).

Treatment. Prophylaxis. Quarantine.—Owing to the intense infectiousness of the disease, any suspected case should be isolated absolutely at once. The extreme diffusibility of the poison renders quarantine in private houses very difficult, and treatment in special hospitals is to be highly recommended. When this is impossible the patient should be confined to his room in the method recommended for infectious diseases in general (p. 345), and all non-immune persons should leave the house. These must necessarily be kept under surveillance and away from other persons until a time equalling that of the period of incubation is past. Everyone exposed should be promptly vaccinated, since by its more rapid development vaccination may modify or even prevent the action of the small-pox infection (Fig. 81). The isolation of the patient must continue until every vestige of the disease has disappeared. This takes longest on the thick epidermis of the palms and soles. The time is necessarily very variable but averages 4 to 6 weeks from the onset of the disease. The patient, the room, and every article which has been exposed should then receive the most thorough disinfection. The patient should be given a bath of 1:10,000 corrosive sublimate solution and the room be thoroughly fumigated with formaldehyde. Attention to detail cannot be too minute.

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[1] Brit. Med. Jour., 1923, XI, 1080.
[2] *Loc. cit.*
[3] Canad. Med. Assoc. Jour., 1924, XIV, 44.

Treatment of the Attack.—This is necessarily purely symptomatic. The patient should be kept scrupulously clean. The bed-clothing should be light and changed often, the room well-ventilated and moderately cool, the food light but nourishing and administered frequently. Water should be given freely. The aching of the initial stage may require opiates, and the fever may need to be controlled by suitable hydrotherapy. The free use of cardiac stimulants is indicated if there is any evidence of weakness. Such sedatives as the bromides and chloral may be required, if great nervous excitement exists.

Many methods have been recommended for the treatment of the cutaneous eruption. None possesses undoubted power to check the tendency to pitting, although the cutting off of the sun's chemical rays by the exposure to red light only has had much said in its favor. Welch and Schamberg[1] believe some good has been accomplished by the employment of tincture of iodine. It may be diluted if necessary. Painting the lesions with a strong solution of potassium permanganate has also been recommended. Much can be done to alleviate the tension and itching of the skin by the application of cold, wet dressings changed frequently, or of glycerin and water, or oily substances. The employment of a mask to keep these in place is often serviceable. Except for young children the oily dressings may contain carbolic acid. Ichthyol (5 to 10 per cent.) has been highly recommended for local use. It is important to keep the crusts always moist with aqueous or oily substances, and frequent warm bathing is useful in favoring their softening and removal as well as earlier for the relief of the cutaneous discomfort. If irritation, such as by scratching, and secondary infection can be avoided little scarring may result.

The local conditions of the mouth, throat and nose require astringent and cleansing gargles, washes and sprays. The holding of ice in the mouth or the employment of orthoform or cocaine locally often gives great relief. The eyes must be cleansed several times a day with boric acid or other mild antiseptic solutions. During the suppurative stage the importance of abundant nourishment is very great and free stimulation is often required. The same methods, with general tonic treatment, are frequently needed during convalescence. Modified small-pox usually requires little treatment of any sort, especially after the initial stage is past.

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[1] *Loc. cit.*, 311.

CHAPTER VII

VACCINIA. VACCINATION

Etiology and Pathology.—Vaccinia, or cow-pox, is an infectious eruptive disease oftenest seen upon the udders of cows, although the horse is occasionally subject to it. Its identity with small-pox has been disputed, but it seems most probable that it is this disease modified by its occurrence in cattle. The inoculation of the human subject with the vaccine virus is called *vaccination.* Although it had long been a popular belief that the contracting of cow-pox from animals prevented small-pox in man, and although the inoculation with the vaccine virus had been practised previously, yet the careful testing of the actual protectitive power and the urging of the procedure upon the medical profession is to be credited to Jenner.[1]

The microörganism of vaccinia is not definitely known. Various bacteria have been discovered in the lymph, and certain bodies apparently protozoa, and entitled "cytoryctes," have been found in the serum of the vesicle by Guarnieri[2] and others. Paschen[3] claims, however, that these are only end-products, and that the germ is much more minute and still undertermined. This is sustained by Prowazek and Miyaji.[4] Whatever its nature, it must be the same as that of variola.

Vaccination was originally performed with matter taken from a cow-pox sore occurring upon a human being. Shortly afterward the virus obtained directly from the cow was employed. Later both bovine and humanized lymph were extensively used, but of recent years the former has entirely supplanted the latter. This has removed the possible danger of transmitting disease, especially syphilis, by the use of humanized lymph. The lymph may be dried upon ivory points or quills, but is preferably, and now usually, mixed with glycerin and preserved in glass tubes; the so-called "glycerinated lymph." Glycerin destroys all pyogenic bacteria if thorough ripening has been allowed. Noguchi[5] has devised a method of inoculation of the testicles of rabbits and young bulls with a virus free from bacteria, and of making cultures from the inoculated organ. In this way virus absolutely free from foreign germs can be produced.

Age for Vaccination.—Regarding the age when vaccination is best performed there is some difference of opinion. That which I prefer is about 3 or 4 months. By this time the vigorous infant will probably have overcome any earlier digestive difficulties. After this age the constitutional effect of vaccination is liable to be greater. Should small-pox be prevalent vaccination should be done in the 1st month. Other circumstances, too, influence the decision, and an occasion should be selected when the infant is in good general condition and free from any irritation of the skin and when the weather is not very hot.

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[1] An Inquiry into the Causes and Effects of the Variolæ vaccinæ, etc., London, 1798.

[2] Centralbl. f. Bakt., 1894, XVI, 299.

[3] Münch. med. Wochenschr., 1906, LIII, 2391.

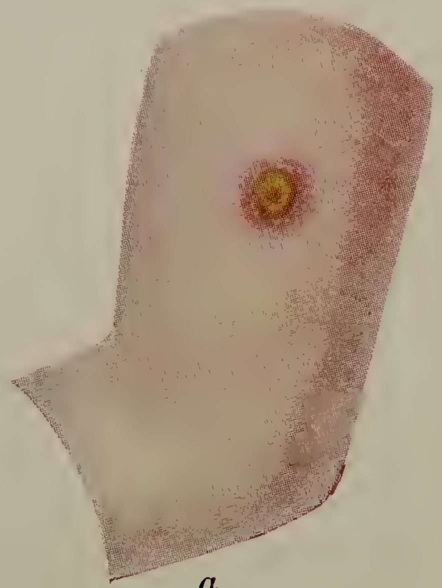
[4] Centralbl. f. Bakt. und Parasitenk., 1914–15, LXXV, 144.

[5] Arch. of Pediat., 1915, XXXII, 698, Orig.

Method.—It has been largely recommended to vaccinate in two or three different spots about an inch apart, on the ground that the greater the number of lesions the less will be the subsequent danger of contracting small-pox. In America scarifying in a single spot is more commonly employed. (See Protective Power of Vaccination, p. 430.) The *locality* usually chosen is the outer surface of the upper arm at about the insertion of the deltoid muscle, or, in the case of female infants, the outer surface of the leg shortly below the knee. The skin should be vigorously cleansed with soap and water and then with alcohol, and the hands of the operator disinfected. A needle previously sterilized by being boiled, or by holding the point in an alcohol flame, should be used to make on the skin four or five scratches parallel and close together, and then an equal number crossing them. They should be only deep enough to scarify the epidermis and expose the blood-vessels, but not to draw blood, lest the entrance of the virus be prevented by it. The whole surface of scarification should not exceed ¼ inch (0.64 cm.). By compressing the small rubber bulb which is commonly sold with the glass tube the lymph flows out upon the wound, and may then be gently but thoroughly worked into the scratches. In place of a needle a scarifier may be employed, as used for the cutaneous tuberculin-test. Very complete and satisfactory outfits are now furnished in which the glass tube terminates in a sharp point with which the scarification may be done. The point is then broken off and the upper end of the tube as well, the rubber bulb fitted and the virus expelled. Other satisfactory outfits contain a sterilized needle preserved until needed in a capillary glass tube. The wound should be allowed to dry for 15 or 20 minutes and then be covered with sterilized absorbent cotton bound upon it with a gauze bandage, and made firm with a few strips of rubber adhesive plaster. The cotton should be left constantly in position throughout the whole course of the development of the pock, unless the vesicle ruptures and moistens it too greatly, or an unusual degree of suppuration takes place. In this case the application of fresh cotton is needed. During the vaccination-period the infant should be sponged but not placed in the tub, in order that the cotton shall not become wet. Vaccination-shields are not to be recommended.

A modification of the procedure described has been highly recommended. It is called the multiple puncture or acupuncture method. The skin is cleansed with ether, a small drop of the virus placed on it in the position desired, and then about 6 punctures made through the drop, holding the needle almost parallel with the skin. The puncture should be confined to an area of not over ⅛ inch (0.32 cm.), and should not be deep enough to draw blood. The drop of vaccine virus is now immediately wiped off. No after dressing of any sort is required. The vesicle and subsequent crusting and scar are very small. It has been advised by several writers to vaccinate by means of intradermal injections, since in this way the scarring is absent or lessened. These various methods possess the advantage of avoidance of infection; the disadvantage that, leaving no scar, there is no evidence later that vaccination has been successful.

Course of Normal Vaccination (Fig. 82).—The development and the histological structure of the vaccine-lesion is practically identical with that of small-pox. (See p. 414.) Except for the scratch-marks the skin shows no alteration until the 3d or sometimes the 4th or 5th day. This closes what may be called the *period of incubation.* A small, faintly-red macule now appears and marks the beginning of the *period of eruption.*



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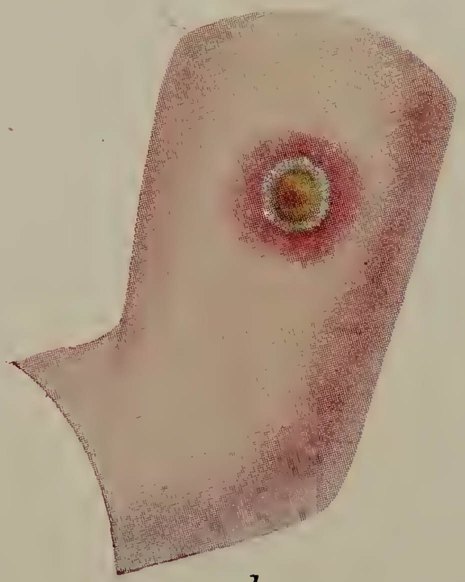

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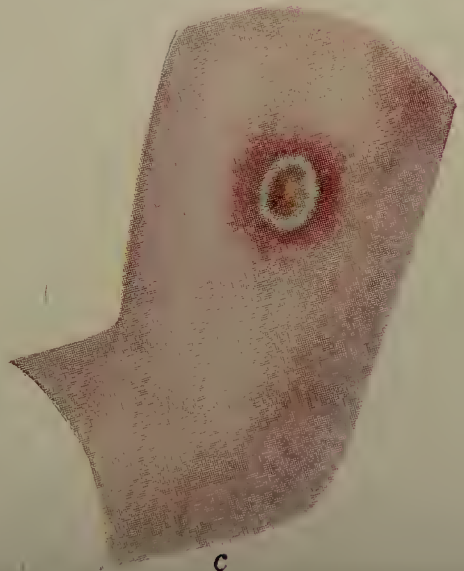

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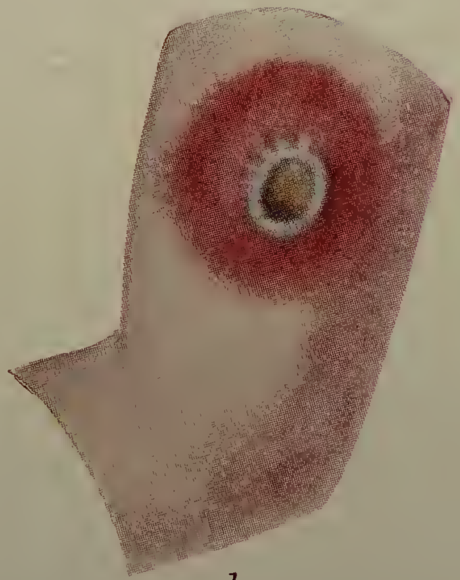

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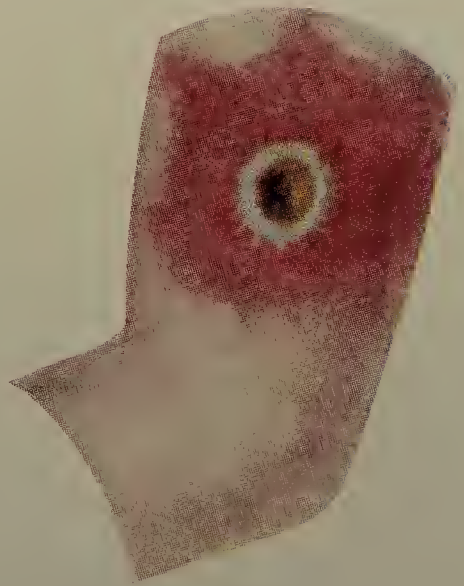

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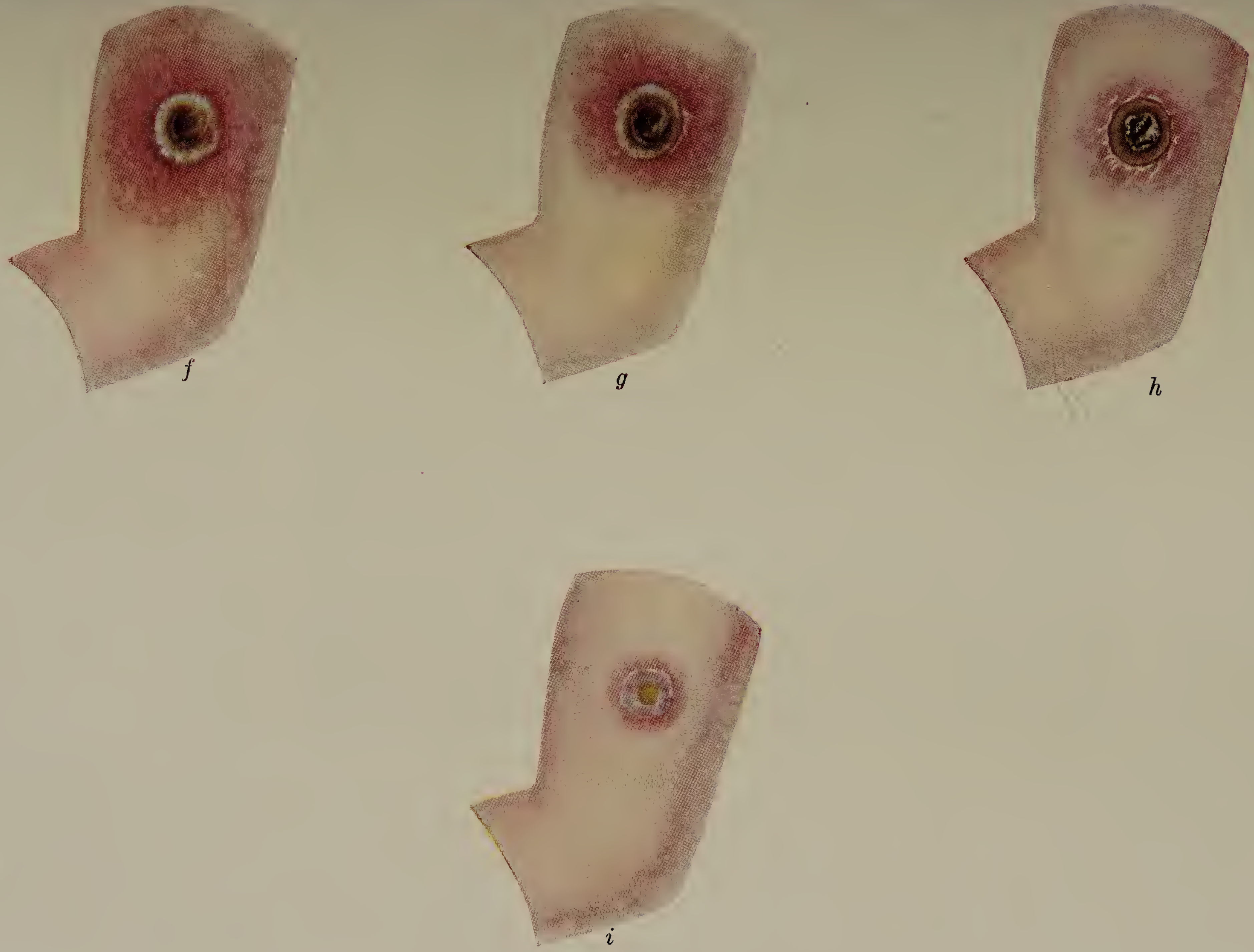

FIG. 82.—COURSE OF NORMAL VACCINATION.

(*a*) 5th day; (*b*) 6th day; (*c*) 7th day; (*d*) 8th day; (*e*) 9th day; (*f*) 10th day; (*g*) 11th day; (*h*) 15th day; (*i*) 25th day.

Infiltration quickly changes this into a papule surrounded by a narrow red areola. By the 5th day of the vaccination a small vesicle appears in the papule, and by the 6th day this covers the scarified area and is filled with transparent lymph. The vessel is multilocular in structure and umbilicated, resembling that of small-pox. From the 7th to the 9th day it reaches its maximum size, is of a pearly-grey color and ½ inch (1.3 cm.) or less in diameter. The areola about it then commences to extend in width and the lymph to become cloudy through the production of pus-cells in it. The vesicle is now tense and yellow, the umbilication disappears, the areola grows still more pronounced, and by the 9th day the underlying and surrounding tissues are red and swollen. On the 10th or 11th day desiccation begins and the surrounding swelling and redness diminish rapidly. The pock is now flaccid and depressed, and rapidly dries into a dark crust. This crust is often fully formed by the 15th day, although it does not fall off, unless forcibly removed, until about the end of the 3d week or later, when it leaves a reddish scar which afterwards becomes white, depressed, and characteristically pitted (Fig. 83).

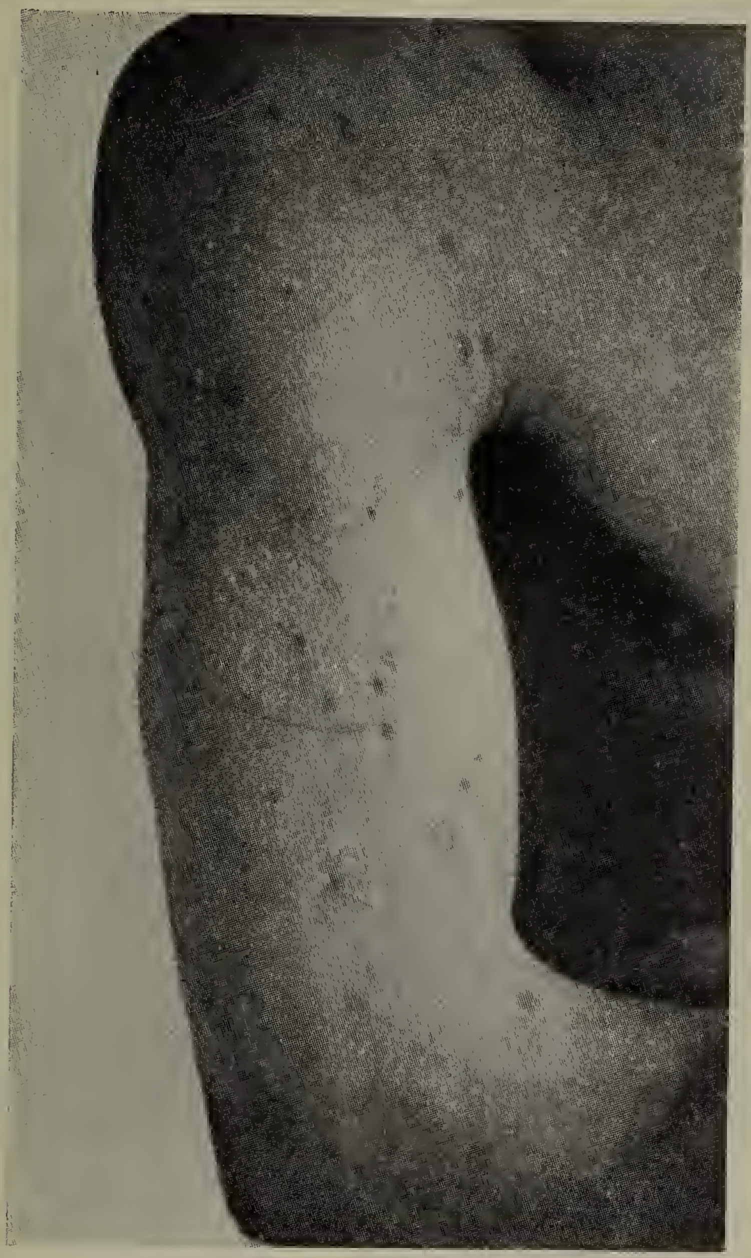

FIG. 83.—SCARS OF VACCINATION.

Four good scars from an infantile vaccination, showing pitting and depression beneath surrounding skin. Patient contracted in adult life a mild, modified smallpox for which he was treated at the Philadelphia Hospital for Contagious Diseases. Stains of smallpox-lesions visible in the photograph. (*Welch and Schamberg, Acute Contagious Diseases*, 1905, *Pl. II.*)

Certain *symptoms* attend the vaccinal eruption, but less often in the early months of life than later. Some itching and tension may occur when the papule is developing and still more when the pustule is at its height. About the 5th or 6th day after vaccination there is in many cases slight fever of a remittent type, which gradually increases, reaching its maximum of 102° to 104°F. (38.9 to 40°C.) on the 8th or 9th day with the full development of the pustule and then falling quickly or gradually (Fig. 84). There is fretfulness, restlessness, loss of appetite, malaise and occasionally vomiting. The axillary or the inguinal glands may be swollen and tender when the pustule is at its height. A decided leucocytosis is present (Sobotka).[1] According to Kato[2] there is a slight primary leucocytosis beginning about the 3rd day after inoculation and lasting about 4 days. Later, about the 12th day, a leucocytosis begins which continues from 7 to 10 days.

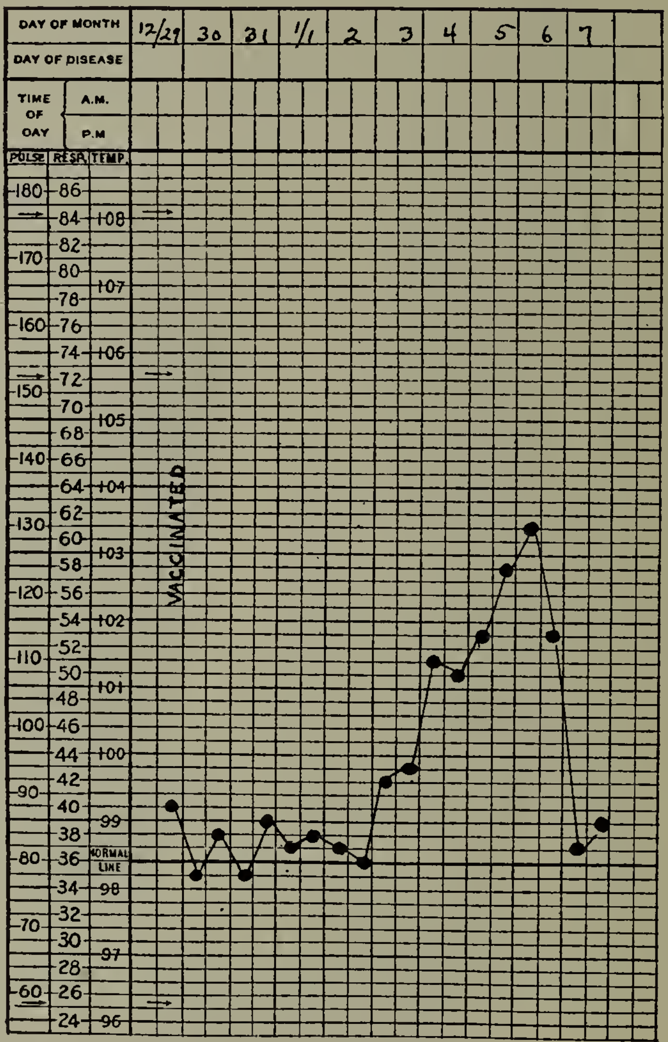

FIG. 84.—VACCINATION.

Marie B., aged 21 months. Dec. 29, vaccinated; Jan. 3, vomited; Jan. 4, lesion well-developed; Jan. 5, arm very sore, axillary glands swollen; Jan. 6, fretful and restless during night, arm unchanged; Jan. 7, arm much less swollen and child bright.

Irregularities in the Course of Vaccination.—Not infrequently the incubation is unduly prolonged, the lesion not making its appearance under a week or even more after vaccination. Less often incubation is shortened and the vesicle is visible by the 3d day. The fever is in some cases decidedly high. The local process may be severe, the lesion becoming unusually large, and not infrequently having several small secondary vesicles developing in the surrounding tissue. These generally run an abortive course.

In rare cases a **spontaneous generalized vaccinia** is witnessed (Fig. 85). In this condition secondary vesicles are found on various parts of the body. They first appear at the time of the maturation of the original pock, or shortly before or after this, and then continue to occur in successive crops, maturing rapidly, the whole process extending occasionally over several weeks, and consequently exhibiting at the same time different stages of development. Generally they leave no scars, but to this there are exceptions. In some cases the lesions strongly suggest variola.

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[1] Zeitschr. f. Heilk., 1893, XIV, 349.

[2] Amer. Jour. Dis. Child., 1924, XXVIII, 711.

A **vaccinia generalized by autoinoculation** likewise may occur, the child reinoculating itself in various places by scratching or in other ways. The spots are usually very few in number. If eczema, for instance, is present the region involved may become infected from the vaccine-pustule and a secondary large confluent lesion develop. The lesions have been seen in the mouth, throat, eye, tongue, nose and elsewhere. The title "generalized vaccinia" is better reserved for the spontaneous form.

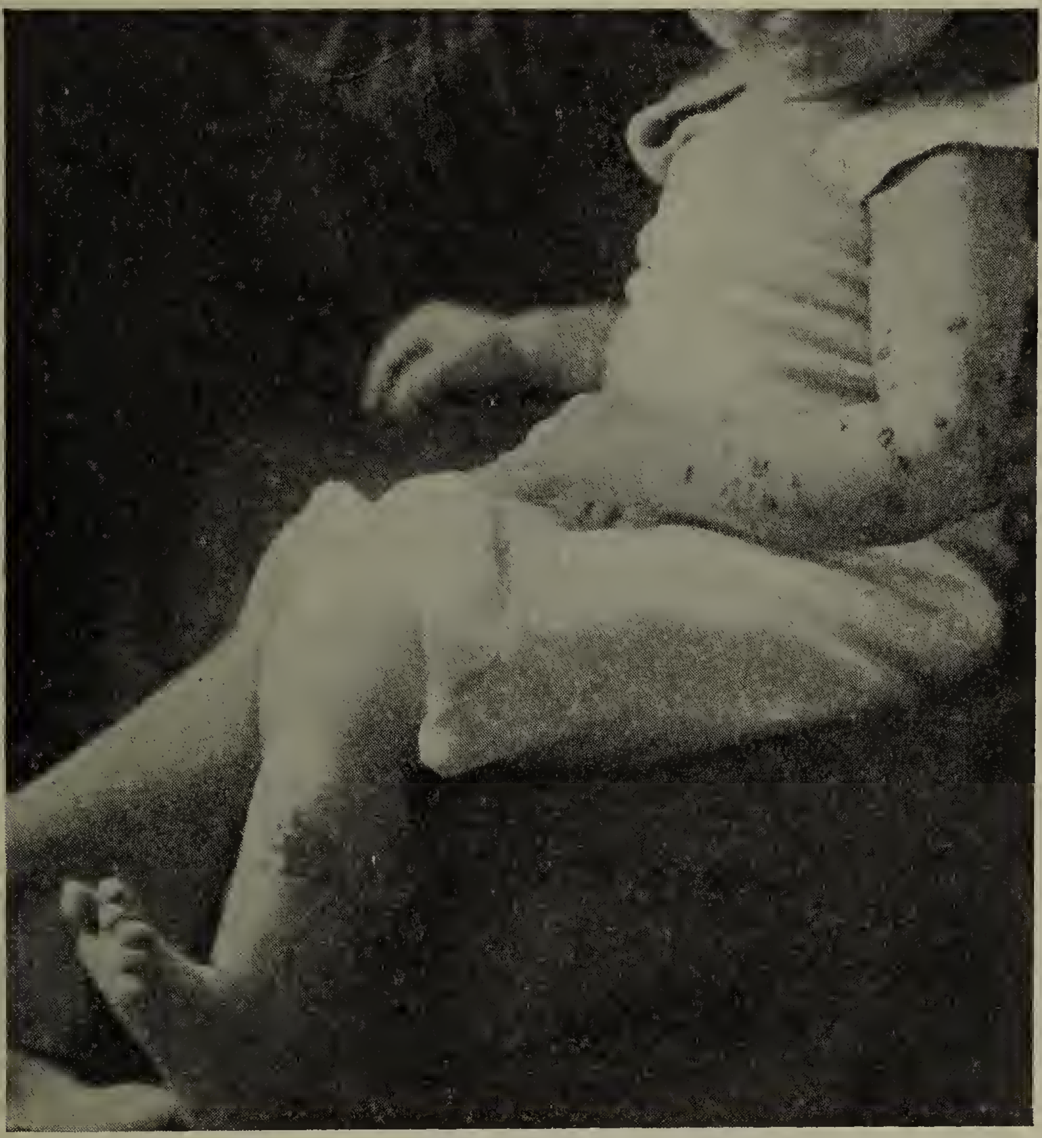

FIG. 85.—SPONTANEOUS GENERALIZED VACCINIA.
Primary vaccination below the knee.

Generalized vaccinia is fortunately rare, inasmuch as it may affect the entire system severely and even cause death. Huddleston[1] was able to collect from medical literature but 50 reported cases of generalized vaccinia, most of them of the spontaneous form, 7 of which terminated fatally. Fatal cases have since been reported by others (d'Astros;[2] Hegler[3]).

Spurious Vaccination.—Particularly in subjects vaccinated not for the first time a lesion may appear which is not evidence of a genuine vaccinal infection. In some such cases a vesicle develops promptly and rapidly produces pus and crusts, the latter by the 7th or 8th day.

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[1] Med. News, 1901, LXXIX, 370.
[2] Marseille méd., 1912, XLIX, 149.
[3] Dermatolog. Wochenschr., 1914, LVIII, Ergänzungsh., 29.

The attendant itching is severe. In some instances merely a papule forms, or a very small vesicle upon this, no scar resulting. In others a projecting, dark-red, slightly granular "raspberry excrescence" may develop instead of the normal vesicle. In these conditions it is probable that little, if any, protective power results, and revaccination should be practised.

Revaccination and Insusceptibility to Vaccination.—As with all infectious diseases certain individuals are temporarily or permanently immune. Repeated vaccinations during infancy may fail to take. In all such cases further attempts should be made at intervals, as there is no certainty that the insusceptibility will be lasting.

Protective Power of Vaccination.—Nothing except the widespread employment of vaccination can account for the great diminution in the number of cases of small-pox. Not only do general statistics throughout the civilized world prove this beyond cavil, but the instances of severe local outbreaks of small-pox in regions where vaccination had been neglected show both the importance of the procedure and that the virulence of small-pox has by no means lessened with the passing years. In countries where vaccination has been made compulsory its value is particularly marked. The accompanying illustration (Fig. 86) shows graphically the result. After the introduction of compulsory vaccination into Prussia in 1874, the average annual death-rate from small-pox fell to 1.91 per 100,000 of the population, whereas from 1816 to 1870 it had ranged from 7.32 to 62.0 per 100,000. In Austria without such regulation the contemporaneous death-rate from it was unaffected (Welch and Shamberg).[1] In the Philippine Islands where small-pox had

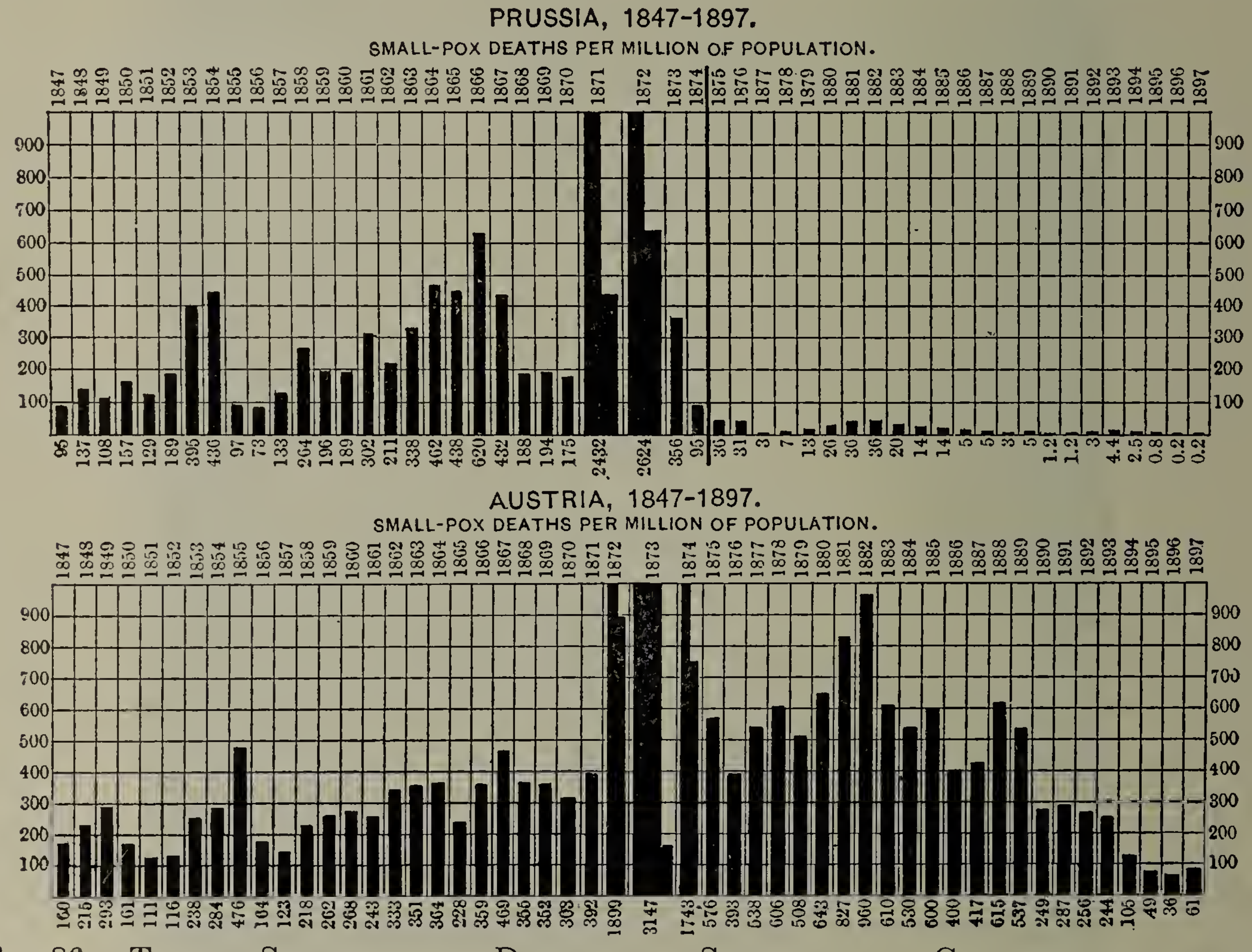

Fig. 86.—Tables Showing the Decline of Small-pox in Germany after the Enaction of Compulsory Vaccination in 1874.

Smallpox mortality is compared with that of Austria. (*Welch and Schamberg, Acute Contagious Diseases*, 1905, 123.)

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[1] *Loc. cit.*, 123.

previously been rampant, with an average annual mortality of at least 6000 cases in the six provinces near Manila, not a single vaccinated person died after systematic vaccination was established in 1907 (Heiser and Oleson)[1] and the efficacy of vaccination in the Philippine Islands is again attested by the report of Padua.[2]

The need for the establishment of a vaccination-law is illustrated by the fact that 18,811 cases of small-pox with 242 deaths occurred in 678 cities of the United States and Canada during the first 6 months of 1924.[3] This subject is well reviewed by White.[4]

The *duration of the protective power* varies. With some persons it is complete and permanent, while with others it gradually lessens, and modified or even typical small-pox is acquired if exposure occurs, the severity of this depending to some extent upon the time which has elapsed since vaccination was performed. The naming of an average duration of protective power is impossible. Welch and Schamberg[5] estimate that absolute protection by vaccination in infancy is found in only about 25 per cent. of the cases It is safe to maintain that revaccination should always be performed at the age of from 5 to 7 years, again at puberty, and again at the beginning of adult life. Certainly, too, if small-pox becomes prevalent, all those who have been undoubtedly exposed should be revaccinated at once, and everyone who has not had a successful vaccination within 5 years should submit to it. If the attempt at revaccination fails it should be repeated. If this also fails, we cannot, of course, be certain that immunity exists, but it is reasonable to assume it.

Although it is manifestly not to be expected that successful revaccination in a partially protected person will produce a lesion exactly similar to that of a primary vaccination, yet it should certainly possess some general resemblance to the original. The character and size of the primary scar has some bearing upon the *degree of protection* offered. The statistics of the Hospital for Contagious Diseases of Philadelphia, from 1871 to 1903 (Welch and Schamberg),[6] give a mortality from small-pox of 6.55 per cent. in patients with one good scar from vaccination in infancy, 14.39 per cent. in those with one fair scar and 24.83 per cent. in those with one poor scar. The degree of protection has been generally believed to be in proportion to the number of scars; hence the custom prevalent in Europe of vaccinating in several places. The statistics of the Philadelphia Hospital for Contagious Diseases do not entirely support this view, the quality rather than the number of the marks appearing to exert the greatest influence.

Complications and Sequels.—An unusual degree of inflammation may occur in the sore, depending upon trauma or a depreciated state of health, or oftener upon infection by pyogenic germs. Deep ulceration may follow, the entire thickness of the skin at the site of the lesion being destroyed, or a diffuse cellulitis or even gangrene developing. Such an occurrence is nearly always due to an infection of the wound at the time of vaccination or later; rarely to any trouble with the virus itself. An attack of eczema may sometimes be produced by vaccination. Urticaria is a not infrequent complication, and a rubeoloid erythema is still oftener seen, developing during the maturation of the pock. This may be localized or may cover much of the body. Other cutaneous disorders have occasionally been reported as complications or sequels, among them impetigo, lichen, ecthyma, furunculosis, psoriasis, pemphigus and miliaria. In rare cases the pocks and the surrounding tissue may become hemorrhagic. Schwartz[1] reports a rare case of post-vaccinal purpura. Exceptionally a general sepsis may follow vaccination, arising from the infection of the wound. An interesting small epidemic of this is described by Brouardel.[2] Erysipelas was formerly a common and much dreaded complication, but with improved aseptic methods it has become very rare. Lotz[3] found but 2 deaths reported from it in 1,252,554 vaccinations. Syphilis has rarely, but undoubtedly, been communicated by vaccination. The employment of bovine lymph entirely avoids the chance of this. The transmission of tuberculosis is perhaps possible with human lymph, but the danger with glycerinated lymph from calves does not exist. Tetanus has undoubtedly been given by vaccination, the germs either entering accidentally an ill-cared-for wound, or being contained in an infected lymph. Although McFarland[4] collected 95 cases from medical literature, the occurrence of this disease as a complication is relatively very rare. Proper supervision of the production of the lymph used will avoid it entirely, so far as the contamination of the lymph is concerned. There is, it is true, always a possibility of its causation by the entrance of germs from the air into the vaccination-wound; but much less, with proper care, than exists in the case of any slight wound which has been left exposed to the air.

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[1] U. S. Publ. Health Rep., 1911, XXVI, pt. 1, 277.
[2] Jour. Amer. Med. Assoc., 1923, LXXX, 1361.
[3] Jour. Amer. Med. Assoc., 1924, LXXXIII, 1344.
[4] Amer. Jour. Dis. Child., 1924, XXVIII, 735.
[5] *Loc. cit.*, 115.
[6] *Loc. cit.*, 57.

Mortality.—The mortality from vaccination is a negligible figure if proper precautions are taken. According to Kübler[5] there were 113 deaths in approximately 32,000,000 vaccinations (0.000035 per cent.) done in Germany during the 13 years from 1885 to 1897, and many of these were directly traceable to neglect.

Treatment.—The treatment of the vaccination lesion has already been described. Should fever develop a mild febrifuge is advisable. In subjects beyond the 1st year of life the vaccinated limb should be used with moderation. If an unusual degree of inflammation occurs the dressing must be removed and the wound treated on general surgical principles.

CHAPTER VIII

VARICELLA

(Chicken-pox)

Although described much earlier, varicella was first differentiated clearly from variola by Fuller[6] in 1730 and by Heberden[7] in 1767. Even after a hundred years there were still some who considered it only a form of variola; but at the present time the question is definitely settled, the independence being based on the appearance of distinct epidemics and its entire failure to protect from small-pox or to be prevented by vaccination.

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[1] Amer. Jour. Dis. Child., 1925, XXX, 856.
[2] Twentieth Century Pract. of Med., XIII, 534.
[3] Ref. Brouardel, *loc. cit.*, 534.
[4] Proceed. Phila. Co. Med. Soc., 1902, XXIII, 166.
[5] Geschichte der Pocken und der Impfung, 1901, 364.
[6] Gee, Reynolds Syst. Med., Amer. Ed., 1880, I, 124
[7] Med. Transac. Col. of Phys., London, 1768, I, 427.

Etiology. **Predisposing Causes.**—*Age* is the most important of these, varicella being preëminently a disease of infancy and especially of early childhood. It is observed in the first months very much oftener than either scarlet fever or measles (Pridham).[1] The first 3 months of life exhibit distinctly fewer cases than later, but the disease has even been described as occurring congenitally. Although the susceptibility lessens greatly after the age of 10 years yet the statements of many writers up to quite recent periods, to the effect that it is never seen in adult life, are contrary to the general experience. Thus, Wanklyn[2] found that of 200 cases of varicella 16.7 per cent. were in persons over 18 years of age. We have found that in approximately 600 cases of chickenpox admitted to the Contagious Disease Wards of the Cincinnati General Hospital during the last 10 years about 15 per cent. were in individuals over 21 years of age.

Season, climate and sex exert no predisposing influence. The individual susceptibility in childhood is very great, most of those exposed contracting it. Not infrequently, however, some children seem to be entirely immune. The comparative freedom of adults is probably in part due to the fact that most persons have already had the disease in early life. It is oftener endemic than epidemic. Thus in some years in large cities great numbers of cases are seen, in others much fewer; yet it is always present to some extent.

Exciting Cause.—This is undoubtedly a germ, the nature of which is, as yet, unknown, although various microörganisms have been reported. De Korte[3] described an ameba-like body present in the vesicles, and Greeley[4] a sporothrix which corresponds to the cytoryctes variolæ, and still other microorganisms have been reported. There is some experimental evidence that a virus occurs in the blood which can transmit the disease to animals (Rivers and Tillett).[5] Further study is needed. The method of *transmission* is not definitely known. Although the disease is in the large majority of instances acquired by direct contact or by short exposure in the vicinity of an affected child, it is not infrequently conveyed mediately through a third unaffected person or through articles of clothing and the like, and is readily carried by the air, especially early in the attack. In this way it spreads easily from one hospital ward to another. It is probable that, next to variola, chicken-pox is the one of the infectious fevers most readily transmitted mediately. Numerous inoculation experiments have been made with the serum of the vesicles, and Steiner[6] claimed to have succeeded 8 times in 10 cases, while Buchmüller[7] had negative results in 30 cases. Since efforts at protective vaccination have been made, the production of the fully developed disease in some of those thus treated is proof that it can be communicated in this way (Hotzen[8] and others). The readiness with which varicella is spread in schools shows conclusively that the *period of infectiousness* commences at the very beginning of the eruptive stage, and, there is reason to believe, even before this. It is uncertain how long it continues, or whether the virus is present in the crusts, but the duration of life is probably short. Experiments by F. H. Thomson[1] indicate that the scabbing stage does not cause transmission after the 8th day of the disease.

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[1] Brit. Med. Jour., 1913, I, 1054.

[2] Brit. Med. Jour., 1902, II, 47.

[3] Practitioner, 1905, LXXIV, 50.

[4] New York Med. Rec., 1914, LXXXVI, 204.

[5] Jour. Exper. Med., 1924, XXXIX, 777; XL, 281.

[6] Wien. med. Wochenschr., 1875, XXV, 304.

[7] Mittheil. des Verein der Aerzte in Steiermark, 1886. Ref. Swoboda, Pfaundler und Schlossmann Handb. der Kinderheilk., 1906, I, b, 724.

[8] Monatsschr. f. Kinderheilk., Orig., 1919, XV, 576.

Pathological Anatomy.—The characteristic lesions consist solely of macules and papules developing later into vesicles, which occupy only the upper layer of the epithelium and are unilocular, or often multilocular as can be demonstrated by pricking them with a needle. The areola often surrounding them is formed by distended capillaries and slight cellular infiltration. The fluid contents are of clear serum which later becomes cloudy and contains a few leucocytes. In some of the vesicles, under the influence of irritation or infection, the process extends deeper, a large exudation of leucocytes takes place, and the corium is destroyed and replaced by scar-tissue.

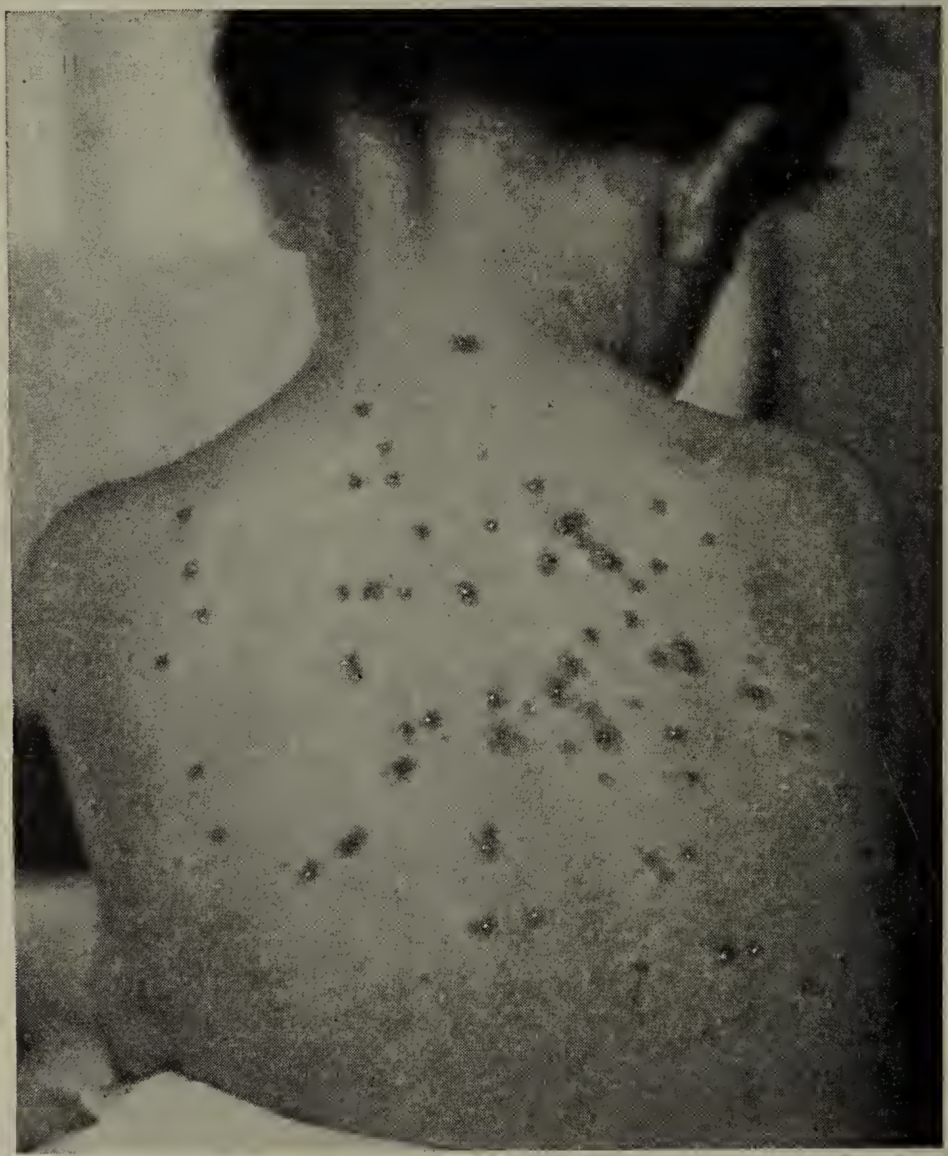

Fig. 87.—Eruption of Chicken-pox, Showing the Pearly Vesicles.
From a case in the Children's Hospital of Philadelphia.

Symptoms. Ordinary Form.—The *stage of incubation* is somewhat variable, averaging about 14 days, but being sometimes less, and extending often up to 3 weeks; in inoculation-cases from 8 to 10 days. The *stage of invasion* is usually absent, although occasionally there may be slight malaise, moderate fever, and general aching, present for 24 hours or less before the rash develops. At times a wide-spread *prodromal erythema* of a scarlatiniform or, more rarely, morbilliform type is seen a few hours before the appearance of the characteristic rash. This is generally most marked on the trunk and lower limbs. As a rule, however, the first symptom observed is the *fully developed eruption.* Tracing the history of the *individual lesion* from its onset, there is first found a red macule disappearing on pressure and of the size of a pin-head to that of a split-pea or larger. Within a very few hours a small vesicle filled with clear liquid develops upon the centre of the macule. In a much smaller number of lesions a small papule forms upon the macule, and a vesicle then very quickly develops on the apex of this. The vesicle rapidly enlarges and reaches its height within a day or less from its first appearance. In shape it is round or oval, and in size very variable, but averages that of a split-pea (Fig. 87). A narrow red areola often surrounds it.

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[1] Lancet, 1916, I, 341.

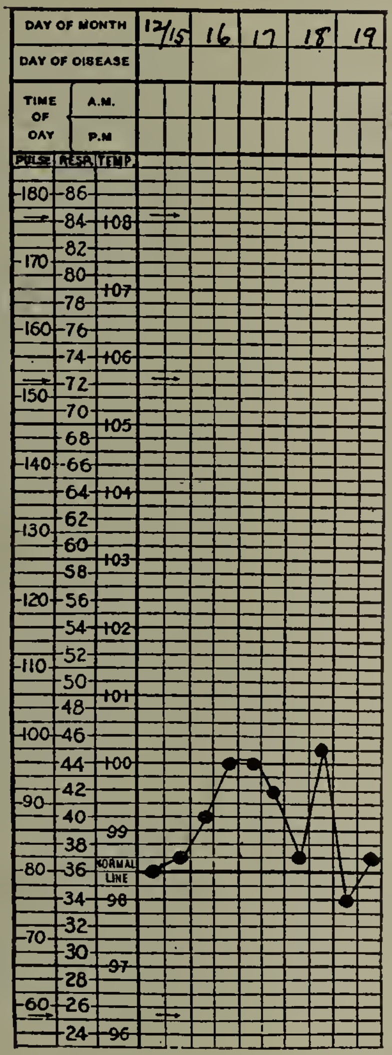

Fig. 88.

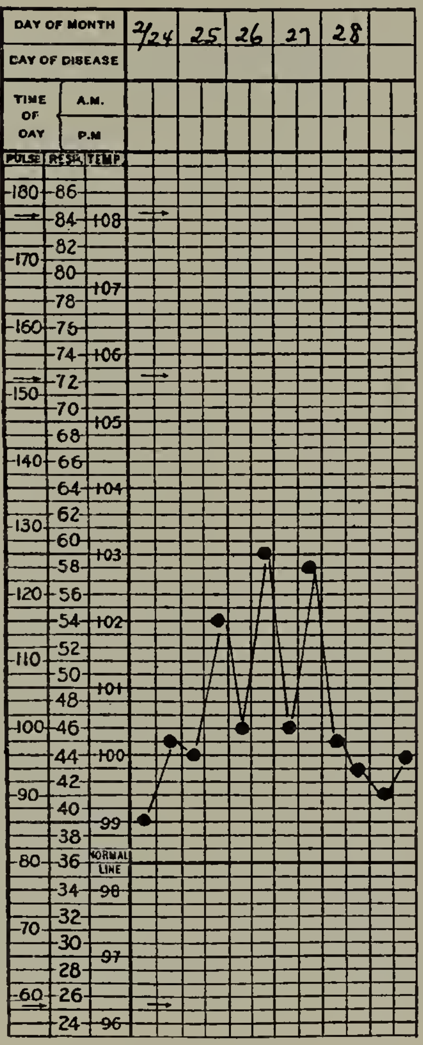

Fig. 89.

Fig. 88.—Mild Varicella.

Philip A., aged 2 years. Eruption developed on Dec. 17. Practically no other symptoms.

Fig. 89.—Moderately Severe Varicella with Unusually Well-developed Eruption.

Caroline R., aged 8 years. Feb. 25, a few vesicles on the body; Feb. 27, child dull; very numerous vesicles, including a large number on the tongue; Feb. 28, improving.

Desiccation now begins at once, the prominent rounded surface flattening a little, and the contents becoming somewhat turbid. Slight umbilication is common at this stage. After 12 to 24 hours more the areola disappears, and a yellowish crust begins to form and is complete in from 3 to 4 days. It darkens and finally falls off after a period varying from 5 or 6 days to 2 or 3 weeks leaving the skin slightly reddened for a time.

This is the course in the typical lesion. The majority of the lesions, however, do not mature. Many macules never develop further, and are no longer visible after a few hours, while a few pass into the papular stage, continue so for a day or two and then disappear. Many vesicles never grow larger than the head of an ordinary pin and dry very rapidly. On the other hand vesicles are often larger than the average, and require a longer time for full development and drying.

The rash generally makes its first appearance on the face and back, less often on the limbs. It spreads over the body from above downward, but so rapidly that the discovery of any sequence is difficult. All parts of the surface are attacked, including the palms and soles and the mucous membrane of the mouth and throat, especially the hard palate, of the nose and the genital organs, and rarely even of the larynx and trachea and the eyes. Upon mucous membranes the covering of the vesicle soon ruptures under the influence of moisture, and the lesion suggests that of aphthous stomatitis.

The number of lesions varies greatly from perhaps a dozen up to hundreds. A case of average intensity presents from 25 to 75. In mild cases there may be scarcely a dozen. They are generally most numerous on the trunk, especially the back. The forearms, hands, legs and feet are involved to a decidedly less extent. The head is generally less affected than other regions but there are nearly always some lesions on the scalp. All the vesicles which mature are generally fully crusted in from 6 to 8 days from the first appearance of the eruption.

One of the principal characteristics of the eruption is its appearance in successive crops during 3 or 4 days in the regions already involved. As a result of this fact, and of the failure of so much of the rash to mature, lesions in all stages of production from macules to crusts may be seen side by side. When a vesicle has become infected, or has been greatly irritated by the rubbing of the clothing, decided suppuration is liable to take place, and an inflamed area is produced, covered by a scab which conceals pus beneath it. The healing is thereby delayed and white scars may be left similar to those of variola.

Symptoms attending the eruption are slight or absent. Moderate fever, averaging 101° to 102°F. (38.3° to 38.9°C.) may be present during 2 or 3 days in well-developed cases, but generally falls gradually to normal in 1 or 2 days. It is higher and lasts longer if the attack is severe (Fig. 88). Often there is no fever whatever. There may be some loss of appetite and disturbance of sleep. Considerable itching of the skin is often present. The blood shows some alterations from normal, although the effect upon the polymorphonuclear and mononuclear cells respectively appears to be inconstant (Weill and Descos;[1] Nobécourt and Merklen;[2] Mensi[3]). There is certainly an absence of the decided leucocytosis seen in variola. Hoffman[4] observed a relative lymphocytosis present throughout the whole attack, and the eosinophiles moderately increased after the 1st week. Some myelocytes are present. The total number of white cells remains normal or subnormal throughout, only occasionally rising to 10,000 or 12,000 from the 8th to the 10th day. Stroh[5] and Baer[6] found the number of leucocytes usually reduced at the height of the disease.

VARIATIONS FROM THE ORDINARY COURSE.—To be mentioned here are only those variations which may be considered uncommon. Among unusual *prodromes* are vomiting, delirium, convulsions, somnolence and severe headache and backache. I have known the latter in the case of an adult to suggest variola very strongly. Blood-stained stools have also been reported.

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[1] Jour. de phys. et de path. gén., 1902, IV, 504.
[2] Jour. de phys. et de path. gén., 1901, III, 439.
[3] Gaz. degli osped. e delli clin., 1912, XXXIII, 1625.
[4] New York Med. Jour., 1923, CXVIII, 646.
[5] Zeitschr. f. Kinderheilk., Orig., 1920, XXVI, 120.
[6] Arch. f. Kinderheilk., 1921, LXIX, 198.

Distinct variations of the *cutaneous lesions* may be seen in the eruptive stage. The prodromal erythema may sometimes continue into, or first appear during, the eruptive period. A **confluent varicella** is very exceptionally observed, in which the lesions are situated on a red, swollen base common to them all, and are so closely placed that they almost touch. This is especially marked on the forehead and back, while elsewhere the discrete character of the lesions is apparent. The eyes are closed by the greatly swollen eyelids, the fever is high, the pulse rapid, and the patient clearly ill. Such cases may arouse a strong suspicion of small-pox.

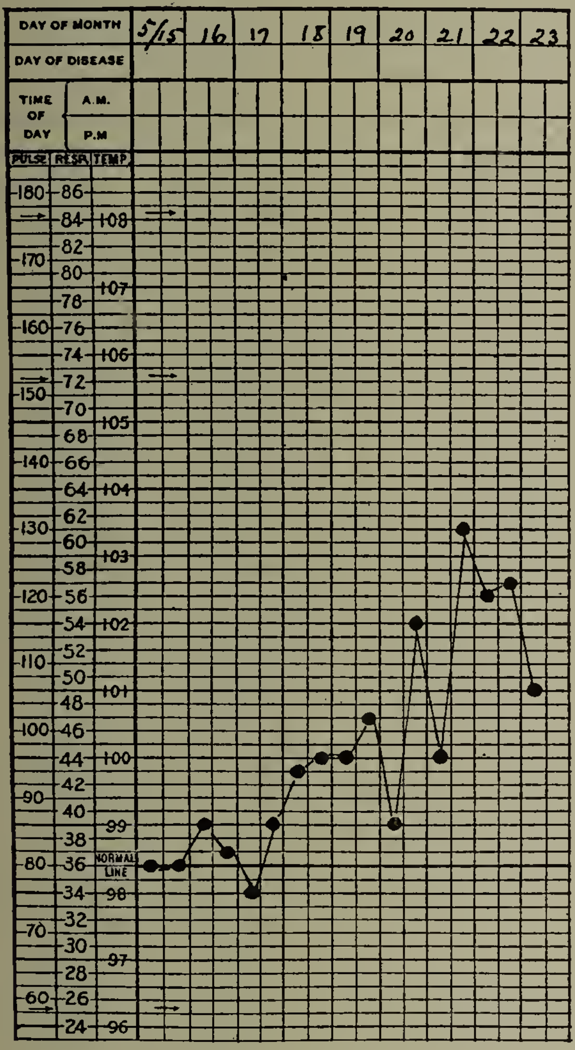

FIG. 90.

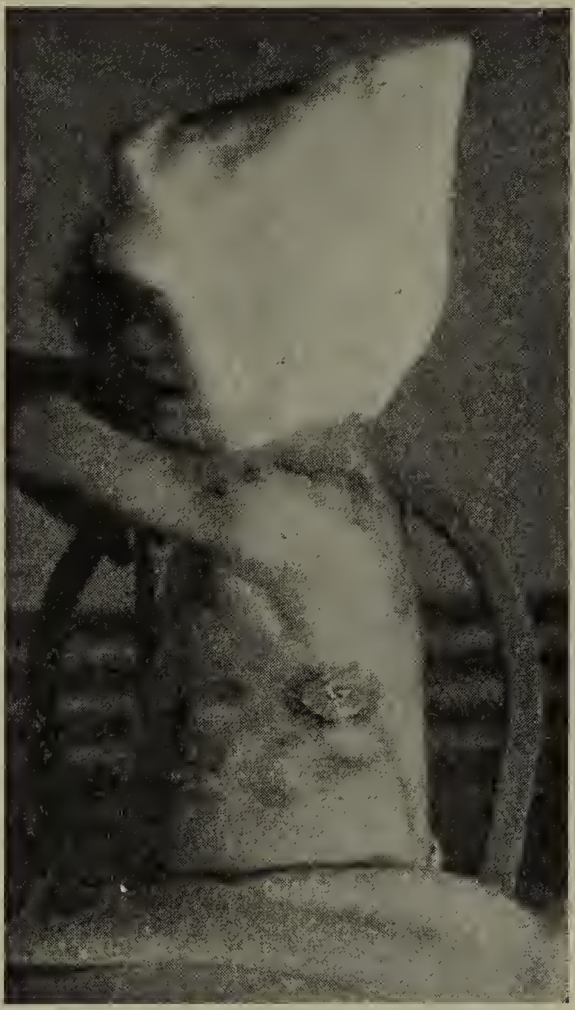

FIG. 91.

FIG. 90.—HEMORRHAGIC VARICELLA.

Elizabeth G., aged 1½ years. May 16, vesicles appearing on the back; May 19, eruption abundant and widespread; May 22, eruption very profuse and infiltrated with blood; May 23, removed from hospital on account of pertussis developing.

FIG. 91.—VARICELLA GANGRENOSA.

Occurring in an infant of 22 months, a patient in the Children's Hospital of Philadelphia. Diphtheria followed by measles and in a few days by varicella. Large vesicles, some sloughing, developed on whole body, especially the head and back. Death.

Hemorrhagic varicella (Fig. 90) is a rare form characterized by effusion of blood into the vesicles and sometimes from the gastroenteric mucous membrane as well. I have seen it but once or twice. The subject has been reviewed by Storrie[1] and others. The term **gangrenous varicella** has been applied to a complicating condition (dermatitis gangrenosa), which, although perhaps seen oftenest in connection with chicken-pox, may occur in other conditions as well. It develops only in debilitated, cachectic children. Certain of the vesicles, under the influence of pyogenic infection, enlarge and suppurate, and a gangrenous process develops in them destroying even the entire thickness of the skin. Septic symptoms and death are liable to follow. In 1 case under my[1] observation the bacillus pyocyaneus was recovered from the lesions. In another (Fig. 91) the disease occurred in a child who suffered simultaneously from rubeola, diphtheria and varicella. **Bullous varicella** is a very uncommon variety in which the eruption appears in the form of large bullæ which may reach an inch or more in diameter and which on rupture leave a painful raw area. The possibility of the eruption of varicella occasionally appearing in the form of peculiar groupings with definite localization has produced in some instances a condition which might be called the **Herpetic form of varicella.** This has excited extensive discussion, as to whether herpes zoster and varicella are in reality the same disorder, or at least closely related. Instances have been published in which herpes has developed apparently as a result of exposure to varicella, and the reverse is equally true. By many, as Comby,[2] these are considered merely coincidences. Enough observations, however, have been made to render likely that certain cases, apparently undoubted herpes zoster, are in reality varicellous in nature, and the complement-fixation tests applied by deLange[3] would seem to prove this. On the other hand, the failure of the one disease to protect against the other would indicate that, in the majority of instances, the disorders are entirely distinct. Reviews of the subject with useful bibliographies have heen published by Ker,[4] Krauss,[5] Bokay[6] and others.

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[1] Brit. Jour. Child. Dis., 1914, XI, 62.

Complications and Sequels.—These are few. One of the chief in importance, although not common, is nephritis, which develops oftenest at the end of the 1st or during the 2d week of the disease. It is of an acute parenchymatous nature, the urine containing albumin and casts. Recovery generally follows promptly, but the disease may be severe enough to be fatal, or may become chronic. A varicellous laryngitis is a severe and sometimes fatal complication rarely seen. It produces symptoms of croup and may require intubation. Multiple arthritis occurring during or at the close of the eruptive period has repeatedly been described. It may be serous or purulent in character. Anemia is a not uncommon sequel. Varicella may not infrequently be combined with other infectious diseases, such as measles, rubella, pertussis, scarlet fever, diphtheria, vaccinia, tuberculosis, erysipelas and even variola. In 234 cases of varicella Stroh[7] observed 137 instances of mixed infections. This is, of course, an unusual number.

Among other conditions reported as occasionally complicating or following varicella are pneumonia, pleurisy, retention of urine the result of swelling of the prepuce, osteomyelitis, otitis, thyroiditis, symmetrical gangrene, appendicitis, multiple sclerosis, chorea, transitory blindness, polioencephalitis, myelitis, abscesses, furunculosis, subcutaneous emphysema and adenitis. The connection of many of these with the attack of varicella seems probably entirely accidental. I have seen one case of fatal purpura hemorrhagica occur as a sequel.

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[1] Univ. Med. Mag., 1896, Aug., 837.

[2] Bull. et mém. soc. méd. d. hôp. de Paris, 1922, XLVI, 992.

[3] Berl. klin. Wochenschr., 1923, II, 879.

[4] Lancet, 1920, II, 347.

[5] New York Med. Jour., 1921, CXIV, 162

[6] Jahrb. f. Kinderheilk., 1924, CV, 8.

[7] *Loc. cit.*

Relapse and Recurrence.—**Relapse** is certainly very rare. Comby[1] observed it in 7 instance 15 days after the drying of the vesicles. Dawes[2] found 4 relapses in 30 cases of the disease; but such a frequency is undoubtedly most exceptional. A few other instances are reported in medical literature which may properly be placed in this category.

Recurrence is also extremely uncommon. One attack protects from a second in nearly every instance. In 524 cases of the disease observed by Widowitz[3] there was no instance of it.

Prognosis.—Although the disease is one of the least serious of the infectious disorders, yet fatal cases are occasionally seen. Death is usually due to complications such as nephritis, erysipelas, pneumonia and gangrenous dermatitis. Varicella of the larynx has a high mortality, as has hemorrhagic varicella. In marantic children the mere development of an ordinary varicella may not be without danger. I recall 4 such infants, sick at the same time in adjoining beds, in all of whom progress toward recovery was apparently checked by the development of chicken-pox, and a fatal issue followed. Very exceptionally in unusually severe cases death may occur during the stage of eruption; it would seem as the direct result of the toxemia of the disease.

Diagnosis.—As a rule this is easy, the eruption being so characteristic that mistakes cannot arise. It rests upon the absence or short duration of prodromal symptoms, the absence of any distinct papular stage in the great majority of the lesions, the situation of the lesions in the superficial portion of the skin, the production of a fully formed vesicle in less than a day from the first appearance of the macule, the development of the vesicles in successive crops with the resulting presence at one time and in the same locality of lesions in all stages, and the drying without, as a rule, any suppuration. Yet severe cases of varicella, especially in adults, may strongly suggest *small-pox*, and on the other hand, modified small-pox may similarly closely resemble varicella. (See Variola, p. 422.) It is to be noted that as a rule the eruption of small-pox selects especially the face and distal portion of the extremities; that of chicken-pox the trunk. To this there are exceptions. As regards *scarlet fever*, it may be difficult or impossible to determine in some cases whether one is dealing with the combination of this with varicella or with the scarlatiniform prodromal rash of the latter disease.

In rare instances the vesicular form of *impetigo contagiosa* may simulate varicella closely. The history of the attack, with the study of symptoms other than the rash, will generally eventually lead to a correct diagnosis. In other cases of varicella seen for the first time during the stage of desquamation, the resemblance to impetigo may be decided. The latter disease, however, spreads irregularly as a result of auto-inoculation by the fingers. The combination of *acne* with varicella may also obscure the diagnosis for a time. *Pemphigoid* eruptions may at first suggest the existence of varicella with unusually large vesicles.

Treatment. **Prophylaxis.**—Owing to the mildness of the disease it is seldom thought necessary to isolate patients suffering from it. Yet in the case of schools, in hospitals where children debilitated by other diseases are present, and even in private homes where there are delicate infants, isolation should be practised. This should be continued as long as crusts remain on the body; *i.e.* a period of 2 or 3 weeks, although the absolute necessity of so long an isolation is doubtful. (See p. 434.) Experience with the box-system shows that it is not as successful a preventive against the spread of the disease as it is for some other infectious disorders (Kleinschmidt).[1] Prophylactic inoculation has been attempted by Kling,[2] Rabinoff[3] and since then by many others, the children being inoculated with the serum from a fresh varicella-vesicle in the same manner as small-pox vaccination is performed. The reported results are much at variance. In some cases there is no protection whatever produced; in others fully developed varicella results. On the whole, however, the procedure appears to possess a certain protective influence, or develops the disease in an unusually mild form. It is questionable whether it can have much practical value outside of insitutions and in special cases (Knoepfelmacher).[4] The possibility of communicating other diseases than varicella by the arm-to-arm inoculation is to be born in mind. Since by this method the children really become the subjects of varicella, they are at the time a source of infection to others. The immunity acquired is an active one and probably permanent in most cases. Hess and Unger[5] believe that complete immunity results from the injection intravenously of a small quantity of serum from varicella-vesicles. Blackfan, Peterson and Conroy[6] and Weech[7] claim that temporary immunity can be given by the injection of convalescent serum within 5 or 6 days after exposure; following the method employed for measles. (See p. 400.) Schmidt,[8] however, has not been successful in preventing varicella in this way. In our[9] experience with 68 definitely exposed patients, 44 of whom were infants or children, and all of whom received prophylactic injections of convalescent serum, only 3 developed the disease.

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[1] Traité des mal. de l'enf., Grancher, etc., 2d Ed., I, 376.

[2] Albany Med. Ann., 1903, XXIV, 532.

[3] Wien. klin. Wochenschr., 1909, XXII, 1596.

Treatment of the Attack.—Usually little treatment is needed. The patient should be confined to bed and given a light diet as long as fever continues. The administration of a febrifuge is advisable. In severe cases the treatment is symptomatic. The urine should be examined frequently for albumin. Itching may be allayed by the application of a powder of camphor and zinc-oxide or of a thymolated, ichthyolated, or, in older children, a carbolated ointment; or by the employment of warm baths. Great care should be exercised to prevent scarring by irritation through scratching, and any large crusts beneath which the existence of suppuration is suspected should be removed and the lesions treated antiseptically. During convalescence the patient ought to remain in the room or in the house. Any subsequent anemia or other sequels or complications require treatment appropriate for them.

[1] Monatsschr. f. Kinderheilk, 1921, XXII, 129.
[2] Berl. klin. Wochenschr., 1915, LII, 13.
[3] Arch. of Pediat., 1915, XXXII, 651.
[4] Monatsschr. f. Kinderheilk., 1923, XXV, 367.
[5] Amer. Jour. Dis. Child., 1918, XVI, 34.
[6] Ohio State Med. Jour., 1923, XIX, 97.
[7] Jour. Amer. Med. Assoc., 1924, LXXXII, 1245.
[8] Med. Klin., 1924, XX, 642.
[9] Arch. of Pediat., 1925, XLIII, 709.

CHAPTER IX

TYPHOID FEVER

Typhoid fever, now widely disseminated and universally recognized, was first clearly distinguished from typhus fever by Gerhard,[1] using his own and Pennock's cases, only as long ago as 1837.

Etiology. **Predisposing Causes.**—Climate and season exert a certain influence, the disease being somewhat more prevalent in temperate zones and in autumn and early winter. The influence of *age* is very decided. With the exception of a very few observers, it is only within about the last sixty years that typhoid fever has been recognized by physicians as occurring in childhood, and even now perhaps most authorities claim that it is uncommon in early childhood, and very rare in the first 2 years of life.

All statistics indicate that the periods in which the greatest number of cases are seen are later childhood, youth and early adult life. There is, however, considerable variance of opinion regarding both the absolute and the relative frequency of typhoid fever before these periods. As compared with the number of cases observed later the disease is decidedly less frequent up to the age of 5 years, and especially in the first 2 years of life. That it cannot be called "rare," as is so often done, is shown by the statistics, among others, of Barthez and Sanné[2] (90 cases from 2 to 4 years), Schavoir[3] (68 under 5 years in a total of 406 at all periods of life), Montmollin[4] (15 under 2 years in 295 children under 15 years), Crespin and Saracino[5] (36 cases under 30 months old). The statistics of general hospitals are misleading, because children are usually treated at home or in special hospitals for children. Even the statistics of these special hospitals by no means represent the actual number of cases in infancy, since the majority of infants who fall ill are cared for at home. This is especially true of those in the 1st year of life. Writing in 1902 in collaboration with Dr. Maurice Ostheimer we[6] detailed 18 cases of the disease developing in the first 2½ years of life, in our own patients or, in a few instances, in those of colleagues. We collected also from medical literature, including these cases, 139 reported as occurring in the 1st year, 187 in the 2d year, and 68 in the first half of the 3d year. While undoubtedly many of these were instances of errors of diagnosis, there have certainly existed very many more in which the disease has not been recognized, or, recognized, has not been reported.

In the spring of 1906 there were in the Infants' Ward of the Children's Hospital, Philadelphia, which contained but a small number of beds, 5 cases of typhoid fever at the same time, in subjects not over 2 years of age. Writing in 1912 I[7] analyzed 75 cases occurring in the first 2½ years of life, personally attended in hospital or private practice or in the practice of colleagues in the Children's Hospital.

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[1] Amer. Jour. Med. Sci., 1837, XIX, Feb. and XX, Aug.
[2] Mal. des enf., 3d Ed., 615, III, 373.
[3] N. Y. Med. Rec., 1895, XLVIII, 803.
[4] Thèse de Neuchatel.
[5] Arch. de méd. des enf., 1919, XXII, 183.
[6] Amer. Jour. Med. Sci., 1902, CXXIV, 868.
[7] Arch. of Pediat., 1912, XXIX, 565.

Typhoid fever may even be found in the fetus and the new-born (*Fetal and Congenital Typhoid Fever*). In 1898 I[1] collected from medical literature 10 such cases which appeared to be beyond question, the germs having been recovered from the tissues or the blood. A few additional instances have since been collected (Griffith and Ostheimer;[2] Morse;[3] Vignoli[4]). Whether the fetus can suffer from the disease in utero, recover, and be born alive and well, is not yet established. The discovery of the agglutinative reaction in the blood of a healthy new-born infant, born of a typhoid mother and not yet suckled by her, would make prenatal recovery a possible thing, but would not be certain proof of this, since the agglutinating principle may have passed to the fetus from the placenta, without there having been any actual disease of the fetus present. (See p. 452.)

The youngest reported instance of typhoid fever acquired after birth is that by Gerhardt[5] in an infant of 3 weeks. I[6] have seen 1 case at 3 months.

Sex exerts practically no etiological influence, although hospital statistics generally give more male cases than female. Previous good health would appear rather to favor than to prevent infection. So also favorable hygienic conditions apart from the question of the transmitting of germs exert no protective power. Statements regarding the influence of other diseases present seem to be very contradictory. In my experience they appear not to be a factor, except that very possibly dietetic errors, producing an irritated condition of the intestinal canal, may readily predispose. Typhoid fever tends to occur in *epidemics*, which, however, are more limited in locality than in the case of many other infectious fevers. In large cities it is more or less endemic. In the year 1923 there were 946 deaths from typhoid fever in 57 cities of the United States having a population of 27,365,408. The decline in incidence is shown by the fact that in the year 1910 in these same cities, then having a population of 20,996,035, there were 4114 deaths from the disease.[7] It is particularly where the malady occurs in several members of one family that children are liable to be attacked. The *individual susceptibility* seems less than that in many other infectious diseases. Whether or not the lesser frequency in infancy is due to a lesser susceptibility or to an absence of equal exposure is uncertain, but the latter seems more probable. The fact that the milk consumed is so often subjected to a high temperature and the germs consequently killed would readily account for this.

Exciting Cause.—The exciting cause of typhoid fever is the *bacillus typhosus* of Eberth, an actively motile organism of the colon-bacillus group found widely spread throughout the tissues of the body and in the blood, bile, sputum, stools and urine.

Transmission.—It is certain that in the vast majority of cases the transmission of the germs is by the feces or the urine. The feces, however, do not appear to be a favorable substance for the actual growth of the bacilli, and those found there have persisted in ulcers or in a chronically inflamed appendix, or have entered with the bile, the gall-bladder being a region where the bacilli may persist for long periods. Whether the expired air conveys the germs to any noteworthy extent is doubtful. Water used for drinking and other purposes is the most frequent carrier, and numerous large epidemics have been traced to this source, while milk contaminated by germ-containing water, or by the infected hands of milkers, is a common carrier in early life unless it had been boiled before it was ingested. Kober[1] analyzed 195 epidemics of typhoid fever and found that the disease was transmitted in probably 148 in this way. The bacilli may also be transported from typhoid stools by flies (Vaughan)[2] and possibly also from the same source by the wind (Pfuhl).[3]

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[1] Phila. Med. Jour., 1898, II, Oct. 15.
[2] *Loc. cit.*
[3] Med. News, 1903, LXXXIII, 193.
[4] Rev. franc. de gynéc. et d'obstet., 1922, XVII, 646.
[5] Handb. der Kinderkr., II, 373.
[6] Phila. Med. Jour., 1898, II, Oct. 15.
[7] Jour. Amer. Med. Assoc., 1924, LXXXII, 389.

In the ordinary sense of the term, however, typhoid fever is but little contagious, and the isolation of patients is not essential. Although hospital infection has repeatedly occurred and the disease has spread in families from the sick to the well, this has not been through mere proximity, but through lack of sufficient care in disinfection. Unquestionably the soiled hands of a nurse, or garments or other articles soiled by feces and urine, can transmit the germs to the food or other objects which enter the mouths of children. The acquiring of the disease in this way occurs probably most frequently in early life. Whether typhoid fever may be contracted through germs transmitted in the milk from a mother suffering with the disease has been deemed uncertain, although the passage of the agglutinating principle is well recognized. (See pp. 442; 452.) Heiman,[4] however, reports a carefully studied instance of transmission by breast-milk, cultures of the milk revealing the typhoid bacilli.

However transmitted the germs are probably always absorbed through the digestive tract and rapidly pass into the blood. An exception is seen in the case of fetal typhoid fever, where the transmission is by way of the placental blood. The *period of greatest infectiousness* appears to be during the 2d and 3d week of the disease, or later until healing of the intestinal ulcers has taken place, but Conradi[5] from a study of 600 cases of all ages found the bacilli in the feces even during the stage of incubation, and believed that the infection is often transmitted during this period. The *tenacity of life* of the germ both within and without the body is often very great. It may survive under favorable circumstances for months, or even for years, and it is not destroyed by ordinary cold or by drying, but is killed by a temperature of 60°C. (140°F.), by disinfectants and by sunlight. In a case reported by Soper[6] it seemed probable that the germ had continued present in the intestines of an individual during 5 years, and cases of the persistence for much longer periods are on record. (Dean,[7] 29 years; Bolduan and Noble,[8] 46 years; Champtaloup,[9] 37 years; and others.) Such long persistence is, however, very unusual. The urine, too, is capable of containing the bacilli over long periods, and Krause[10] found this the locality in 59 per cent. of the carriers studied.

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[1] Amer. Jour. Med. Sci., 1901, May.
[2] Amer. Jour. Med. Sci., 1899, CXVIII, 10.
[3] Zeitschr. f. Hyg., 1893, XIV, 1.
[4] Jour. Amer. Med. Assoc., 1919, LXXIII, 913.
[5] Deutsch. med. Wochenschr., 1907, XXXIII, 1684.
[6] Jour. Amer. Med. Assoc., 1907, XLVIII, 2019.
[7] Brit. Med. Jour., 1908, I, 562.
[8] Jour. Amer. Med. Assoc., 1912, LVIII, 7.
[9] Brit. Med. Jour., 1919, II, 338.
[10] Beiträge z. Klin. d. Infectionskr. u. z. Immunitätsforsch., 1917, V, 75; 81.

Pathological Anatomy.—The lesions so characteristic of the disease in fatal cases in adults are notably altered in early life. In fetal typhoid fever there is an entire absence of intestinal ulceration, perhaps the natural result of the placental mode of entrance of the germs. In the majority of congenital cases, too, intestinal lesions have not been reported, and this is occasionally true of older subjects. After these periods the lesions vary somewhat with the age of the patients. In the first 2 years of life the process is more hyperplastic than destructive, and the solitary and agminated glands are swollen, projecting, and of a pinkish color. Slight and generally superficial ulceration may be present, and in some cases there is no intestinal involvement whatever. The swelling of the mesenteric glands is usually very pronounced and the spleen is always acutely enlarged and soft; and these two conditions are suggestive; but there is nothing in the post-mortem findings in infancy which is positively diagnostic of typhoid fever.

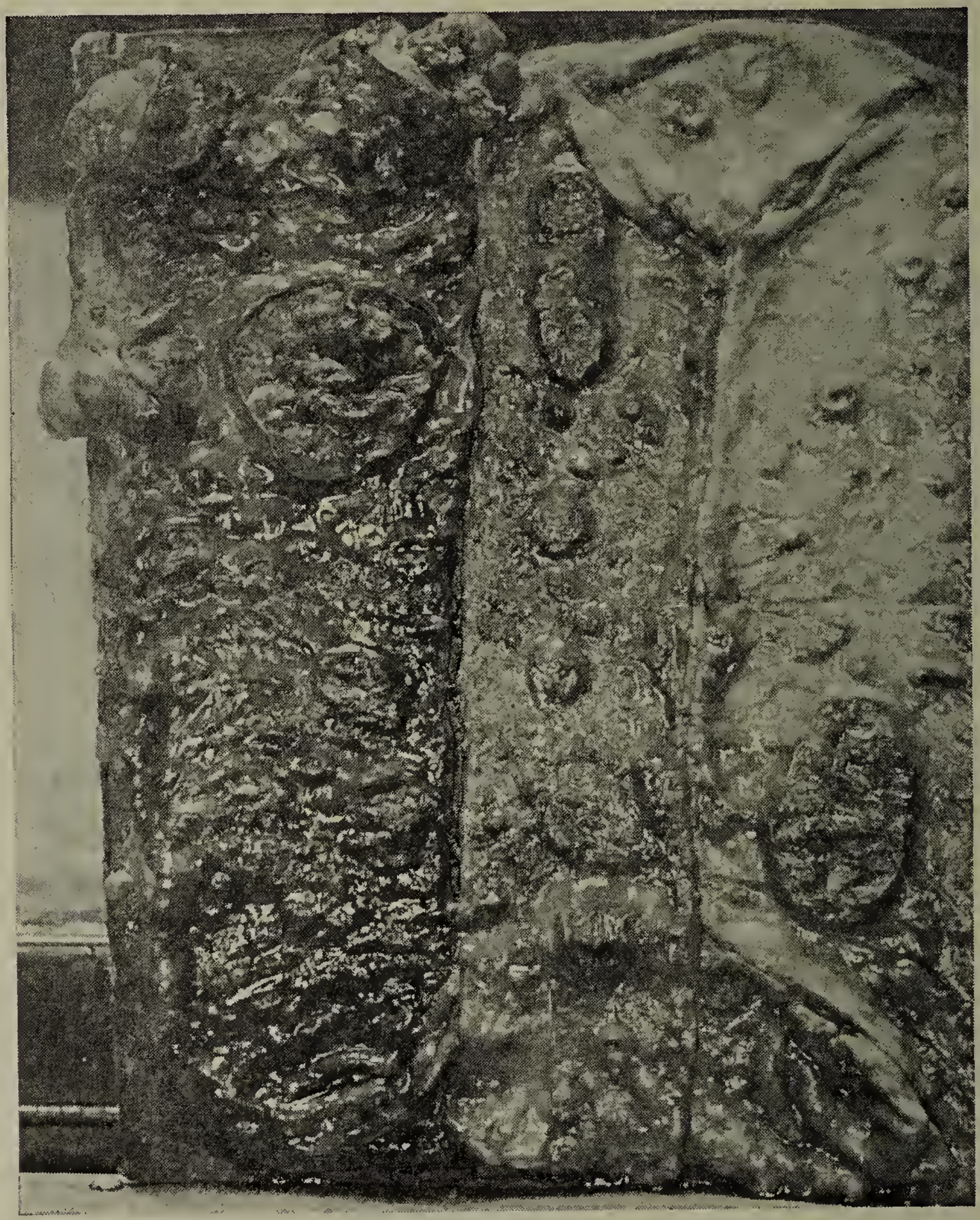

Fig. 92.—Ulceration in Typhoid Fever.

Child of 6 years. Autopsy showed an unusual degree of intestinal lesions for this time of life.

In early childhood, between the ages of 2 and 6 years, the lesions are very similar to those just described, except that the intestinal ulceration is somewhat more marked. It is still, however, generally superficial and the process is predominatingly hyperplastic. There are, however, many exceptions to this rule, and very extensive ulceration may occur even at 6 years (Fig. 92).

In later childhood intestinal ulceration becomes much more frequent and decided, although in most cases less marked than in adult life. It is only as the age of puberty is approached that the lesions are practically the same as in the adult.

Typhoid bacilli are found in the various secretions and excretions, in different organs, the rose spots and the blood. They may be discovered in the stools often before the serum-reaction can be obtained. The tissue-degenerations occurring in adult cases are absent or less marked in early life.

Symptoms.—The period of *incubation* is variable and difficult of precise determination. It may in general be placed at from 1 to 2 weeks. Languor, loss of appetite, and allied symptoms characterize it in the majority of cases. The actual beginning of *invasion* is marked by the development of fever, which continues in average cases about a week before the second or *eruptive stage* is ushered in by the appearance of the roseola.

The symptoms of the attack vary according as the disease occurs in the new-born, in infancy after this period, in early childhood (2 to 6 years), or in later childhood. These divisions are to be viewed merely as an artificial classification, since a great many exceptions exist, and subjects in one age-class may exhibit the symptoms of those pertaining to another. The peculiarities of the average case as observed in the latter part of early childhood and the first portion of later childhood may be reviewed as those characteristic of the general type for early life.

Ordinary Course.—The principal symptoms distinguishing the disease in early life taken as a whole are: (1) The indefinite and uncharacteristic onset; (2) the shorter duration and greater mildness of the attack; (3) the disposition for nervous symptoms to over-balance intestinal ones. This does not mean at all that the nervous symptoms are actually more marked than in adult life.

In many cases the **onset** is very abrupt, particularly in young children, the disease becoming thoroughly developed in the course of a few days. Vomiting may be one of the first symptoms in such cases. The disease is occasionally ushered in by convulsions, or may begin violently with the manifestations of meningitis so well-marked that the true nature of the malady is discovered only later. In the majority of cases, however, the onset is peculiarly insidious and even long-continued, with symptoms so little pronounced that the roseola and enlarged spleen may, perhaps, be found upon the first examinatiod. The child had been walking about with some degree of malaise, loss of appetite, slight headache and thirst, and with fever which had possibly not been recognized by the relatives. It is then difficult to determine just when the attack commenced. In other cases the initial stage is much shorter, the fever lasting only 2 or 3 days before the typhoid roseola appears.

When the disease has reached the second, or **eruptive stage** there is generally an absence of the severe nervous manifestations which constitute the typhoid state as seen in adult life; apathy and slight nocturnal delirium being the symptoms oftenest seen. The whole course of the attack is decidedly curtailed, especially the **stage of decline.** An average duration of the attack may be placed at from 14 to 20 days.

To this general description there are, of course, very numerous exceptions, and cases may vary in duration from that of the abortive type to that greatly prolonged; and in severity from the mildest to the most pronounced.

The symptoms can best be studied individually. Of the *gastro-intestinal* symptoms, vomiting is a frequent initial manifestation, and even later is more common than in adults, and sometimes almost or quite uncontrollable. I have seen this the direct cause of death. Sore throat is an occasional initial symptom and the tonsils may be red and swollen. The appetite is diminished and thirst is present. The tongue is generally coated, and often exhibits the red triangle at the tip and the red edges frequently described as characteristic of the disease. Dryness develops, as a rule, only in the severer cases in older children.

The condition of the bowels is variable. Constipation and diarrhea are about equally frequent, the former perhaps preponderating. Yet this depends upon the epidemic, and there have been years in which nearly all the cases under my care had diarrhea, although generally not severe. The stools may show the typical pea-soup appearance. Involuntary evacuation occurs only exceptionally and in the severest cases.

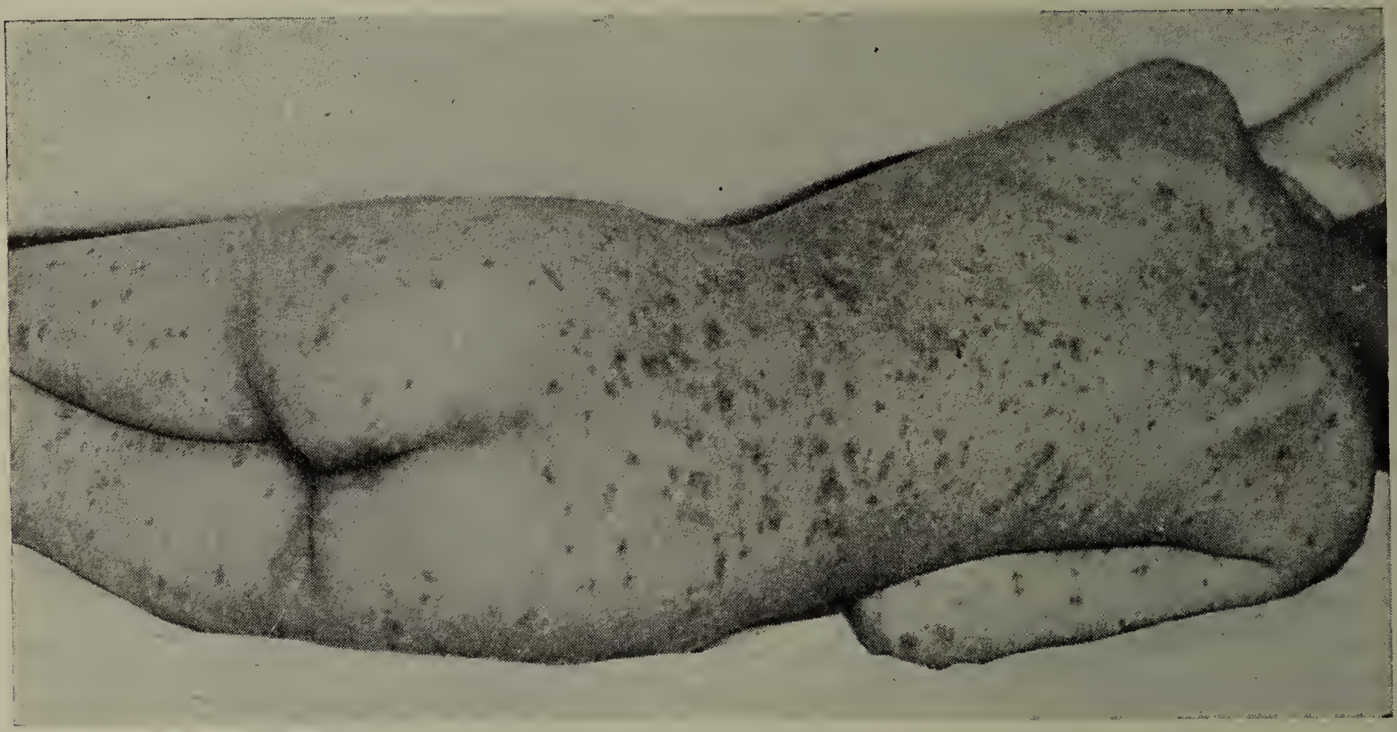

Fig. 93.—Typhoid Fever with an Unusual Degree of Development of the Eruption.

Minnie S., admitted to the Children's Hospital of Philadelphia Feb. 22, aged 5 years. Illness began 5 days before. Spots on abdomen observed the day before admission. The day after admission the spots were very numerous, and were seen also on the face. Spleen palpable. Recovery.

The abdomen is generally only moderately distended and may be slightly tender in the right iliac fossa, but great distention and decided tenderness are much less often seen than in adult life.

Enlargement of the spleen is always present, although not always demonstrable. It can be detected in probably 80 to 90 per cent. of all cases in children. Russow[1] observed it in 85 per cent. of 1034 cases in children. It is discoverable at about the same time as the eruption. As long as it persists the typhoid process cannot be considered completed. Lymphatic glandular enlargement is not infrequent but is generally slight. The typhoid *roseola* is sometimes stated to be less often present or less well developed in children. In my own experience this is not the case, and in 671 cases reported by Morse[2] the eruption was observed in 60 per cent. Henoch[3] found it absent only 19 times in 381 cases. In average cases it is discoverable about the end of the 1st week. As in the adult, the spots appear in successive crops, each lasting about 3 days. Exceptionally the eruption is well-marked over the whole body, involving even the face, and sometimes suggesting closely the early appearance of the rash of measles (Fig. 93). The spots continue to develop as long as the infection is active in the system. Sudamina are common, particularly in older children. A branny desquamation during convalescence has been described as especially liable to occur in children. I have noted it frequently

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[1] Pädiatrija, 1911, No. 3. Ref. Monatsschr. f. Kinderheilk., Ref., 1912, XI, 224.
[2] Bost. Med. and Surg. Jour., 1896, CXXXIV, 205.
[3] Kinderkrankheiten, 1895, 773.

The *temperature* often runs a very irregular course. When typical it resembles that of adult cases except for the shorter duration and the frequent absence of the terminal remittent character (Fig. 94). In the initial stage it shows an evening rise and morning fall, with the gradual step-like ascent lasting until about the end of the 1st week. In very many cases, however, this stage is shortened greatly, and the fever rapidly rises to its maximum without the step-like character. In the second stage, that of the "acme," "fastigium," or "eruptive period," the temperature remains continuously high, from 103° to over 104°F., (39.4° to 40°C.) with but little variation between morning and evening elevations. The high temperature, however, is generally much better tolerated than in adult life. In one instance a girl 10 years showed a temperature of 107°F. (41.7°C.) without the slightest sign of discomfort or of nervous symptoms. The temperature of the third stage, or stage of

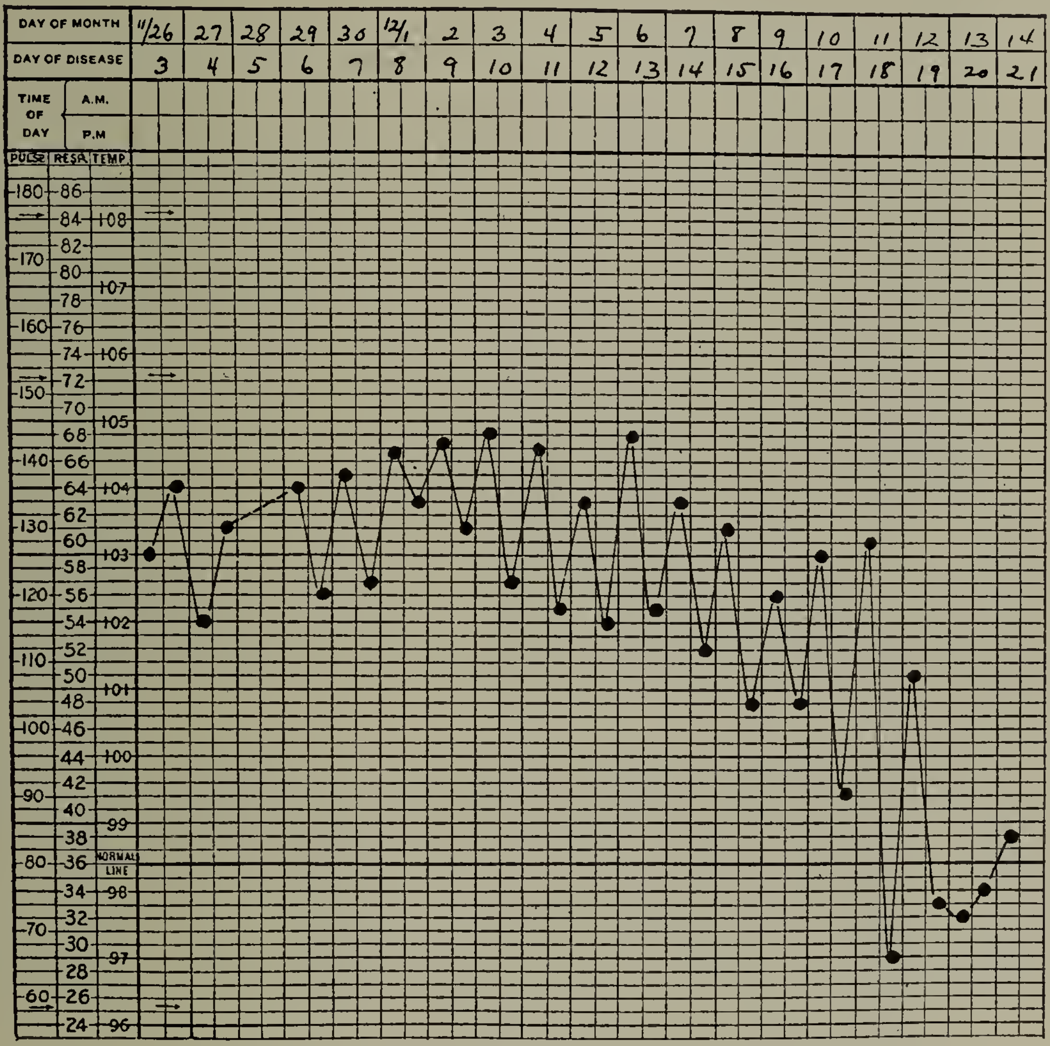

FIG. 94.—TYPHOID FEVER, AVERAGE CASE.

Evelyn P., aged 11 years. Nov. 26, chilly 2 days ago, been feeling tired, slight sore throat; Nov. 28, spleen enlarged to palpation, appetite poor; Nov. 30, general condition excellent, slight diarrhea, leucocytes 10,400; Dec. 2, rose spots found, Widal reaction negative; Dec. 6, mind entirely clear, slight diarrhea, Widal negative; Dec. 9, been no nervous symptoms except moderate apathy; Dec. 15, convalescent, appetite still poor.

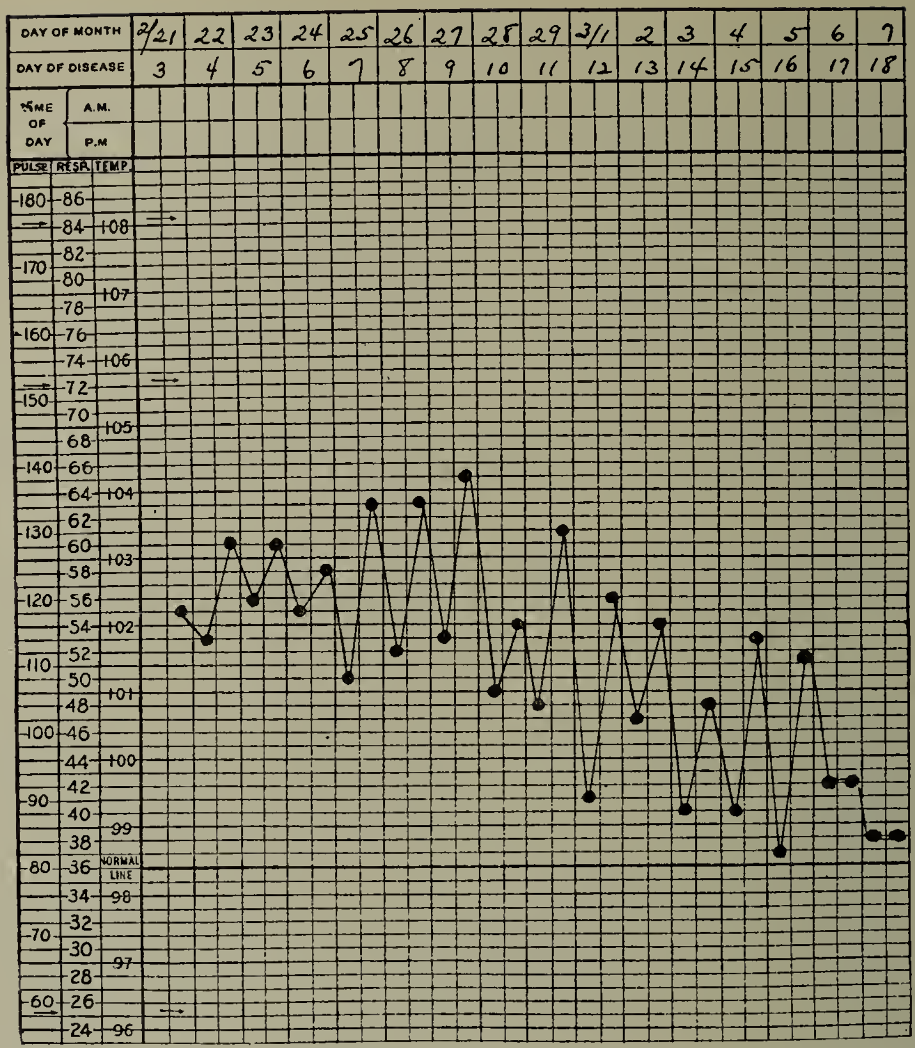

FIG. 95.—TYPHOID FEVER, ORDINARY FORM FOR CHILDREN.

Theresa F., aged 7 years. Came home from school with headache on Feb. 19; first seen Feb. 21; had not been in bed. During attack exhibited loss of appetite, enlarged spleen, very little apathy, Widal reaction. Was bright all the time. A few spots first found during a relapse.

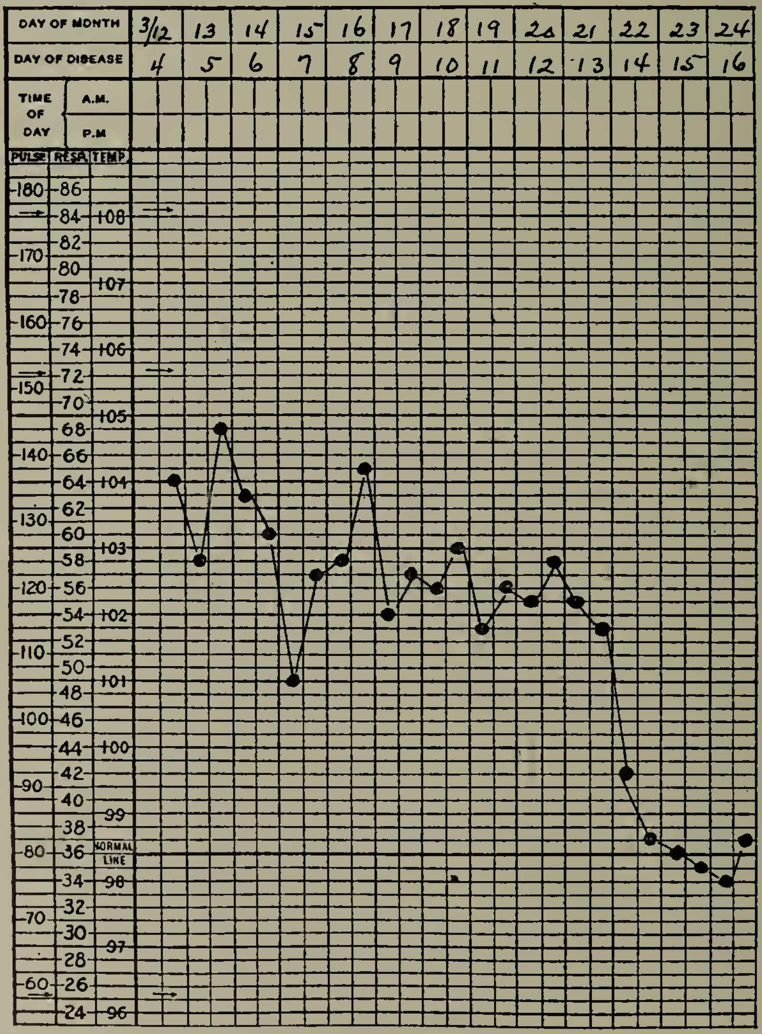

FIG. 96.—TYPHOID FEVER WITH CRITICAL FALL.

George C., aged 7½ years. Sudden onset with convulsion. During attack exhibited roseola, splenic enlargement, moderate diarrhea, good general condition. Temperature fell critically on the 14th day of the attack.

decline, is of much shorter duration than in adults (Fig. 95). According to Morse's[1] statistics the prolonged remittent form is absent in about ½ of the cases. This absence doubtless depends on the lesser degree of intestinal involvement. The final fall is commonly much more rapid than in adults, and is often almost critical (Fig. 96).

Although the average duration of the fever is from 2 to 3 weeks, yet numerous exceptions are observed. Sometimes it lasts little over a week, while in other cases it continues high with little change for 3 or 4 weeks or more. Before complete defervescence takes place there is shown a tendency for the temperature to become elevated from insignificant or undiscoverable causes (Fig. 97). This may in some cases result in an unusual prolongation of an irregular pyrexia. In other cases this is probably the result of an unusual persistence of the infectious process. In children apparently otherwise convalescent I have seen fever continue for 6 or 7 weeks (Fig. 98) and in one instance for over 12 weeks. How irregular and deceptive the course of the temperature may be at times is illustrated in the accompanying chart (Fig. 99) of a case in which only the blood-culture made the suspected diagnosis finally certain. The chart does not represent the temperature-record at the same time daily

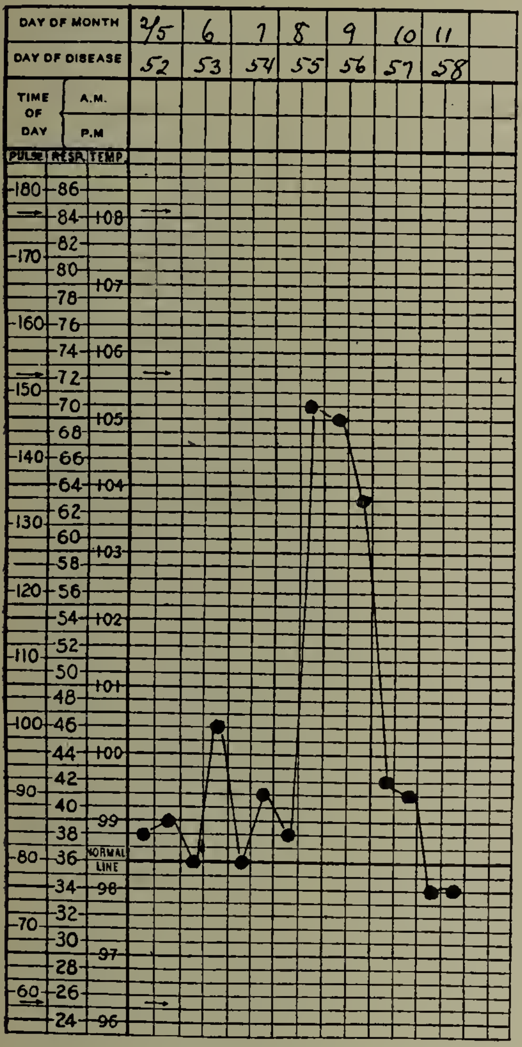

FIG. 97.

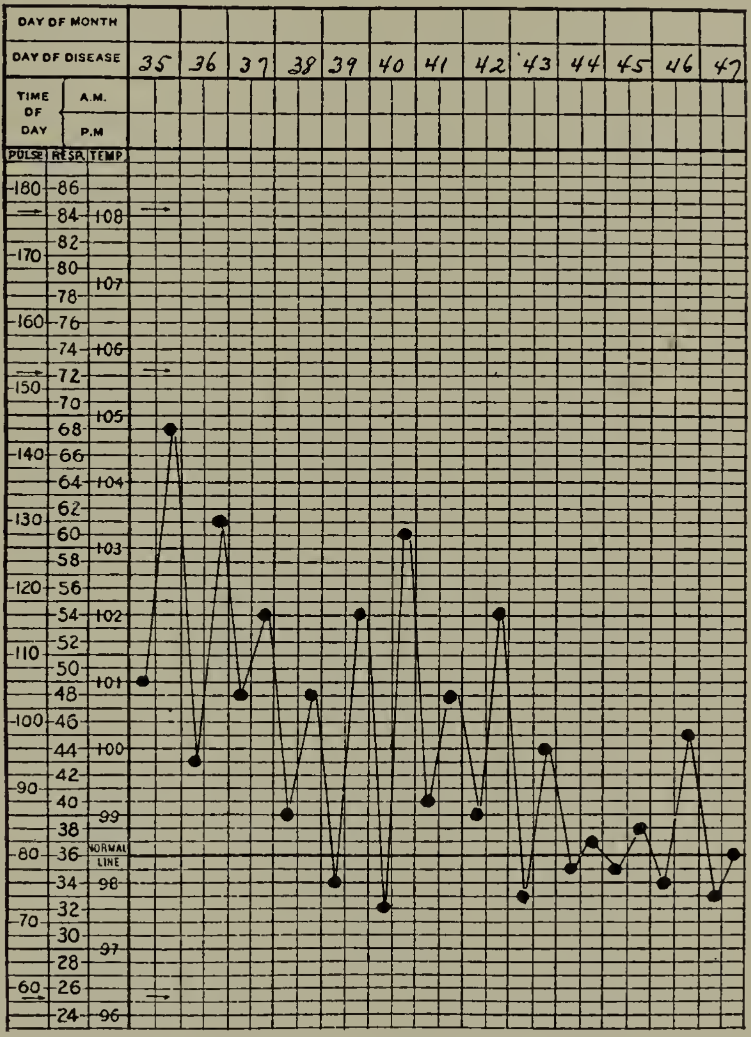

FIG. 98.

FIG. 97.—RECRUDESCENCE IN TYPHOID FEVER.

Chas. L., aged 5 years. Recovering from relapse.

FIG. 98.—TYPHOID FEVER, PROTRACTED CASE, NO COMPLICATIONS.

Rose B., aged 8 years. Disease ran ordinary course, rather mild, splenic enlargement and rose spots. Then developed irregular fever without other symptoms or discoverable cause. This lasted until the 46th day.

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[1] Bost. Med. and Surg. Jour., 1896, CXXXIV, 205.

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but rather the maximum and minimum for each day, there having been an entire absence of regularity in the hours of these occurrences.

The chief characteristic of the *blood* in typhoid fever is the absence of leucocytosis. The normal number of leucocytes belonging to the different ages of children (p. 59) must be taken into consideration in this connection, as also the very great ease with which many secondary conditions may produce leucocytosis in children with typhoid fever. An increase in the number of leucocytes is consequently not positive evidence that typhoid fever is absent, nor is a low leucocyte-count, which is diagnostically much more valuable, proof that no inflammatory complication exists. We have seen a Type II pneumococcus-pneumonia followed by an empyema complicate typhoid fever without rise in the total white count. Allowing for the influence of age, the differential count gives results identical with those seen in adults; there being a decrease in the number of neutrophiles and an increase of the mononuclear cells, especially the lymphocytes. The eosinophiles are diminished. The percentage of hemoglobin and of red cells is decidedly reduced as the disease advances. The typhoid bacilli may often be found in the blood before the Widal reaction can be obtained. After convalescence from severe attacks the hemoglobin especially shows a percentage-reduction. The

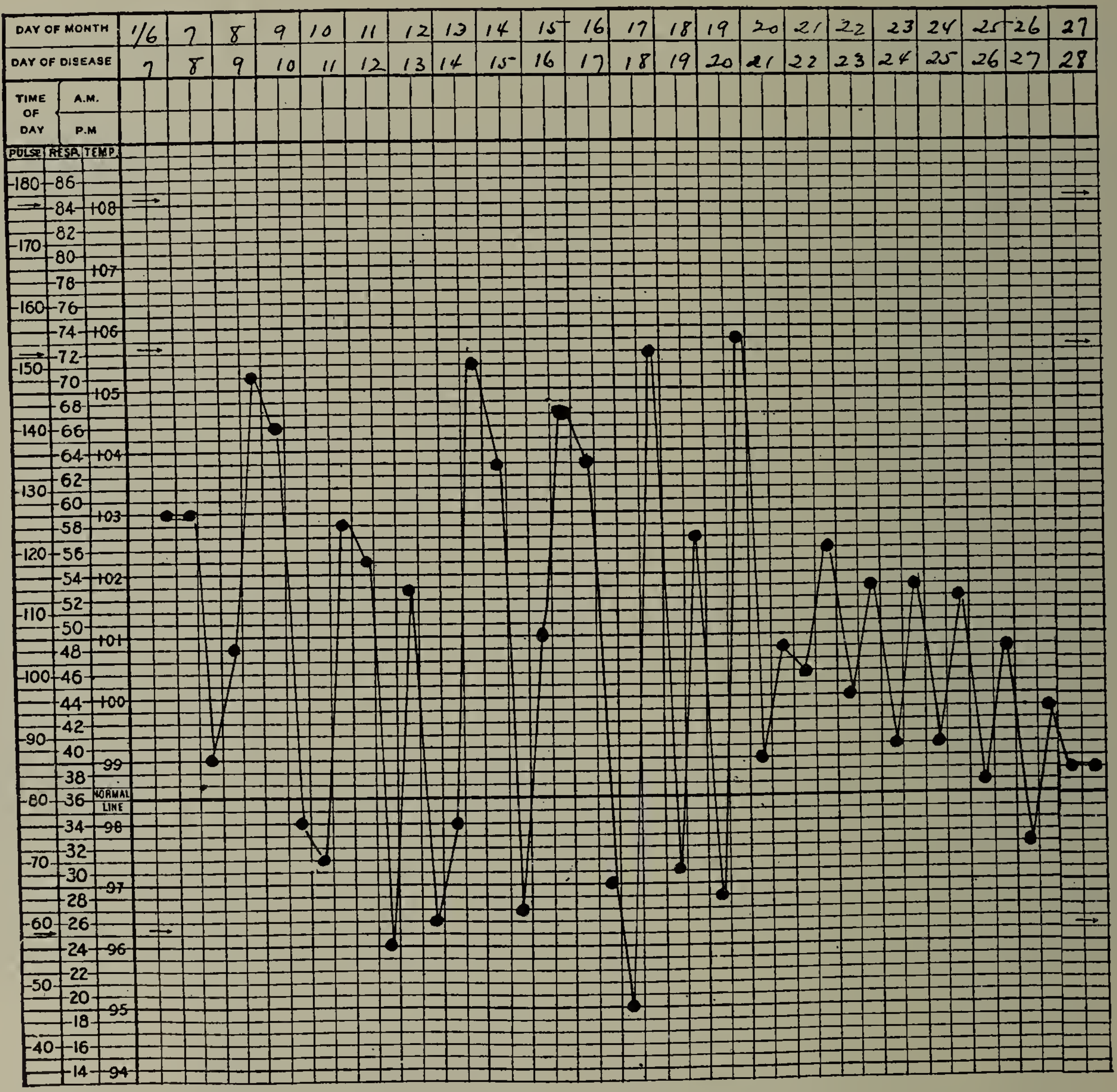

FIG. 99.—TYPHOID FEVER, UNUSUALLY IRREGULAR TEMPERATURES.

Christopher S., aged 8 years. Patient in the Children's Hospital, Phila. Jan. 7, intense headache, leucocytes at first 14,200, tache, rigidity of neck, Kernig's sign, ankle-clonus, profuse sweating and chills, suspected of being malaria, cerebral abscess, meningitis, or sepsis, lumbar puncture negative, no plasmodia; Jan. 14, blood-culture positive for typhoid bacilli, rose spots, positive agglutinative reaction, leucocytes now 9800.

blood-serum added to a fresh culture of typhoid bacilli causes a cessation of the movements and a clumping of the germs ("serum," "agglutinative," or "Widal" reaction). It occurs in about 95 per cent. of cases. It was present in 94 per cent. of 66 of my cases in the first 2½ years of life. It is usually not obtainable before the 5th or 6th day of the attack, or sometimes not until this is well advanced or the patient even convalescing. It may even be absent during the entire course of the primary attack and develop during a relapse. It may continue for months after the disease is over.

The *pulse*, as in adults, is not infrequently slower than the elevation of temperature would call for. The decided tendency to dicrotism, common in adults, is observed generally only in older children and then not very often. Not infrequently it is quite irregular and peculiarly slow during convalescence. In bad cases it may be irregular or unusually rapid during the course of the disease. The *arterial tension* is low. The cardiosphygmographic studies made by Schlieps[1] on 100 children with typhoid fever showed that the commonly observed arrhythmias are dependent upon sinus-irregularities and do not affect the prognosis. In severe attacks a dusky-red flushing of the cheeks sometimes indicates the vasomotor disturbance present.

Of symptoms connected with *respiration* cough is common yet seldom troublesome, and coarse râles are often heard in the chest. Epistaxis is probably not as often an initial symptom as in adults. Its occurrence in 40.9 per cent. of 550 cases as reported by Adams[2] would appear to be unusual. Except in the mild cases *emaciation* is commonly very decided and is sometimes extreme. *Nervous symptoms*, although more marked than intestinal, are, as stated, generally less severe than in adult life. Headache is quite common, especially at the outset, but is not often intense or persistent. In severe cases there may be much pain in the limbs, or the back or joints. I have known stiffness of the muscles of the neck to occur, without the mental symptoms which would suggest meningitis. The abdominal reflex is diminished. Many children remain in the best of spirits throughout the attack and do not feel or appear particularly ill; while others are usually irritable, especially if disturbed. Oftenest, however, the principal and the most characteristic nervous symptom is a decided apathy, with a tendency to be quiet and to sleep, which is increased when the temperature rises. Prostration is seldom as great as in adult cases, except after unusually severe attacks or in patients in whom some debilitating cause, such as severe diarrhea, has been present. Delirium is generally absent or slight, occurring only at night, but in severe cases may become very pronounced. Convulsions sometimes usher in the attack (Fig. 96) even in cases which are not later severe. Coma, stupor, coma vigil and subsultus are rarely seen except in older children. A condition strongly suggesting meningitis is sometimes observed during much of the attack. This is not frequent, although probably oftener seen than in adults (p. 457 and Fig. 106).

The *urine* exhibits a decided diazo-reaction. Febrile albuminuria may be present if the temperature is high, but evidences of nephritis are not as common as in adults. Acetone is occasionally found.

VARIATIONS IN TYPE.—Different types of typhoid fever seen in early life may be described. The classification may be made either (*A*) according to the age of the patient, or (*B*) according to the characteristics of the attack in general.

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[1] Jahrb. f. Kinderheilk., 1911, LXXIV, 386.

[2] Amer. Jour. Med. Sci., 1910, CXXXIX, 638.

(*A*) Based upon *age* the following types may be mentioned:

1. **Fetal and Congenital Typhoid Fever.**—The only distinction between these two varieties is that in the latter the child is born alive and consequently exhibits symptoms.

The majority of pregnant women suffering from typhoid fever abort. The collected statistics of Etienne[1] show this occurrence in 70 per cent. of pregnant women with the disease. Nothing in the superficial appearance of the body of the infected fetus indicates the nature of the disorder, but the germs may be recovered from the blood and the organs. In the congenital cases the infant is born alive, sometimes prematurely and sometimes at term. Life may continue only a few minutes, the cause of death not being apparent. In others the disease may last several days, or even 2 weeks or longer. The symptoms are usually entirely uncharacteristic. Fever is generally present; and convulsions, jaundice, diarrhea, constipation, tympanites, roseola, enlarged spleen, cough, vomiting, intestinal hemorrhage and purpura have been reported. Death almost always occurs. As already pointed out the discovery of the Widal reaction in these cases cannot be looked upon as diagnostic, since it may be found in apparently healthy children born of typhoid mothers, the agglutinating principle having entered either by way of the placental circulation or through the milk. (See p. 443.)

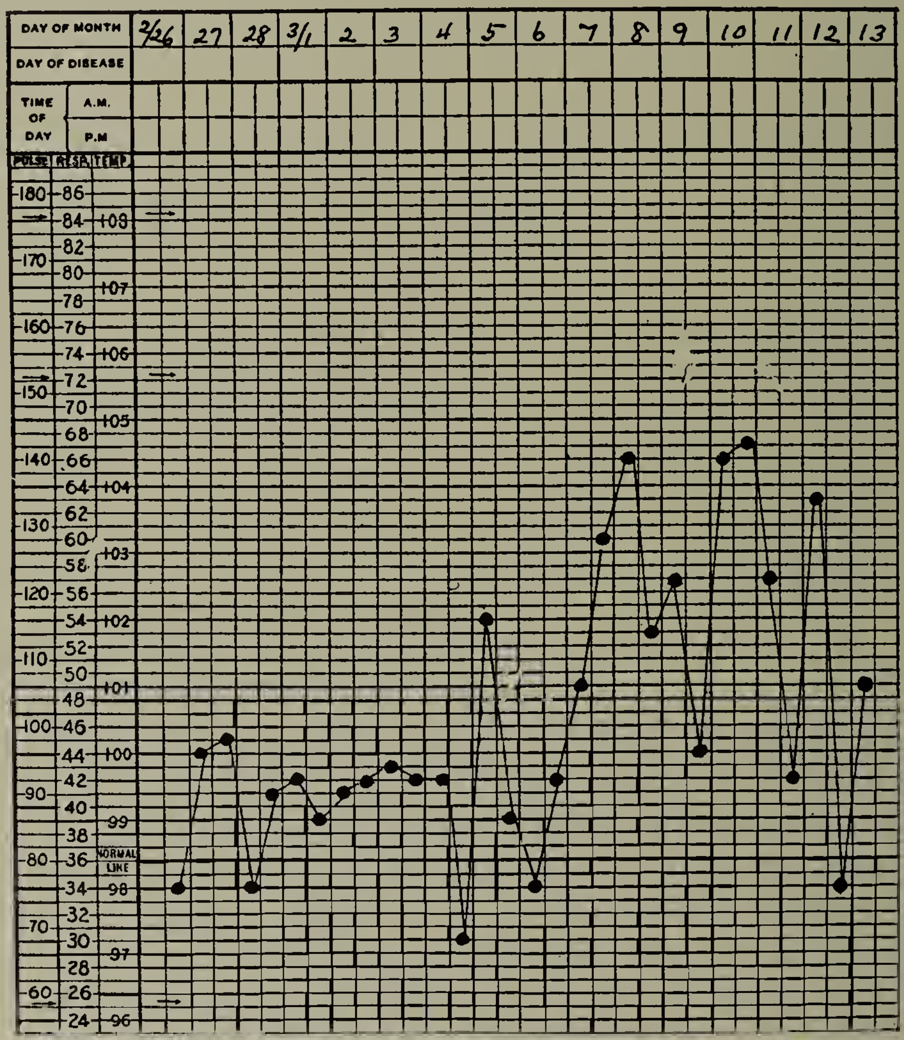

FIG. 100.—INFANTILE TYPHOID FEVER.

William H., aged 3 months. Child restless, with slight cough, abdominal distention, moderate diarrhea with greenish stools, increasing weakness, stupor and loss of appetite. Death on Feb. 13. Diagnosis supposed to be ileocolitis. Autopsy showed spleen soft and much enlarged, Peyer's patches and mesenteric glands hyperplastic, Widal reaction in heart's blood. Case illustrates severe course with uncharacteristic symptoms.

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[1] Gaz. hebdom., 1896, XLIII, 184.

2. Infantile Typhoid Fever.—Under this heading may be included cases in which the affection was acquired after birth and during the first 2 years of life. The disease in this period, and especially in the 1st year, is often marked by the absence of most of the characteristic symptoms, the diagnosis being impossible, except by blood-culture, until the typhoid eruption or the agglutinative reaction is discovered. The temperature is generally high and very irregular in type, and is usually attributed to some of the many other causes of fever in early life. Very often the disease may be supposed to be an ileocolitis of moderate severity, since diarrhea is a frequent symptom common to both (Figs. 100, 107). Diarrhea, tympanites and vomiting are rather more frequent at this time of life than later. Bronchitis is of common occurrence; epistaxis uncommon. The patient is often prostrated and may seem more ill than the symptoms rationally explain. The pulse is rapid and nervous symptoms may be decided, fretfulness and restlessness being more frequently seen than apathy. As in fetal typhoid the disease often takes the form of a blood-infection with local symptoms little marked (Fig. 107).

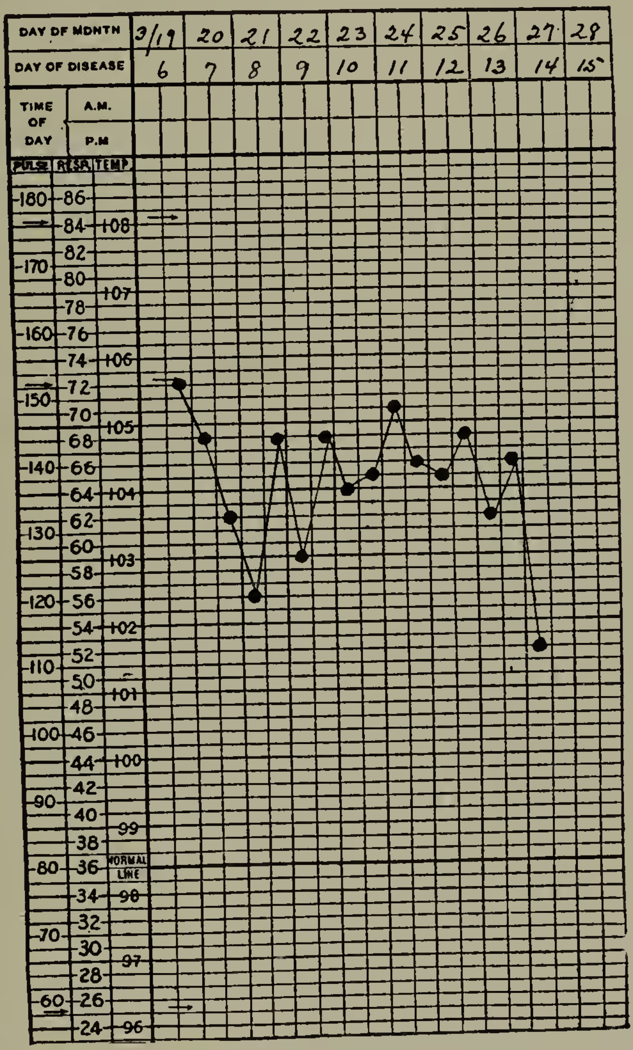

FIG. 101.—TYPHOID FEVER IN EARLY CHILDHOOD, SEVERE CASE.

Sadie T., aged 3 years. Rapid onset with fever and prostration and loss of appetite. During attack suffered from slight diarrhea, numerous râles in chest, occasionally dyspnea, apathy, sopor, sometimes great irritability, occasional vomiting, severe abdominal distention, cyanosis. Death on the 14th day of the disease.

3. Typhoid Fever in Early Childhood.—At this period, from the age of 2 up to that of 6 years, the attack is usually of a more benign type than at any other time of life. It is now that the characteristics already described as those of the disease in early life are most prone to show themselves. Diarrhea is less common than either in infancy or in later childhood and is seldom troublesome. The temperature is more suggestive of typhoid fever than in infancy, but that of the third stage is generally much abbreviated and not of a remittent type. The course is short. Nervous symptoms are nearly always mild except in the cases with a meningitic onset. Complications are not as frequent as later. To this description there are of course numerous exceptions, and the attack may be very severe (Fig. 101).

4. Typhoid Fever in Later Childhood.—This type is distinctly more like that seen in adults, and the nearer the child is to puberty, the closer is the resemblance liable to be. This is especially true after the age of 10 years (Fig. 102). Diarrhea may now be troublesome, and hemorrhage and perforation are more liable to occur than earlier, all this depending upon the greater likelihood of intestinal ulceration at this time of life. The symptoms of the typhoid state are more prone to develop, but only in severe cases to any great degree. The course is apt to be longer than in early childhood, and the fever of the third stage is oftener of the remittent type.

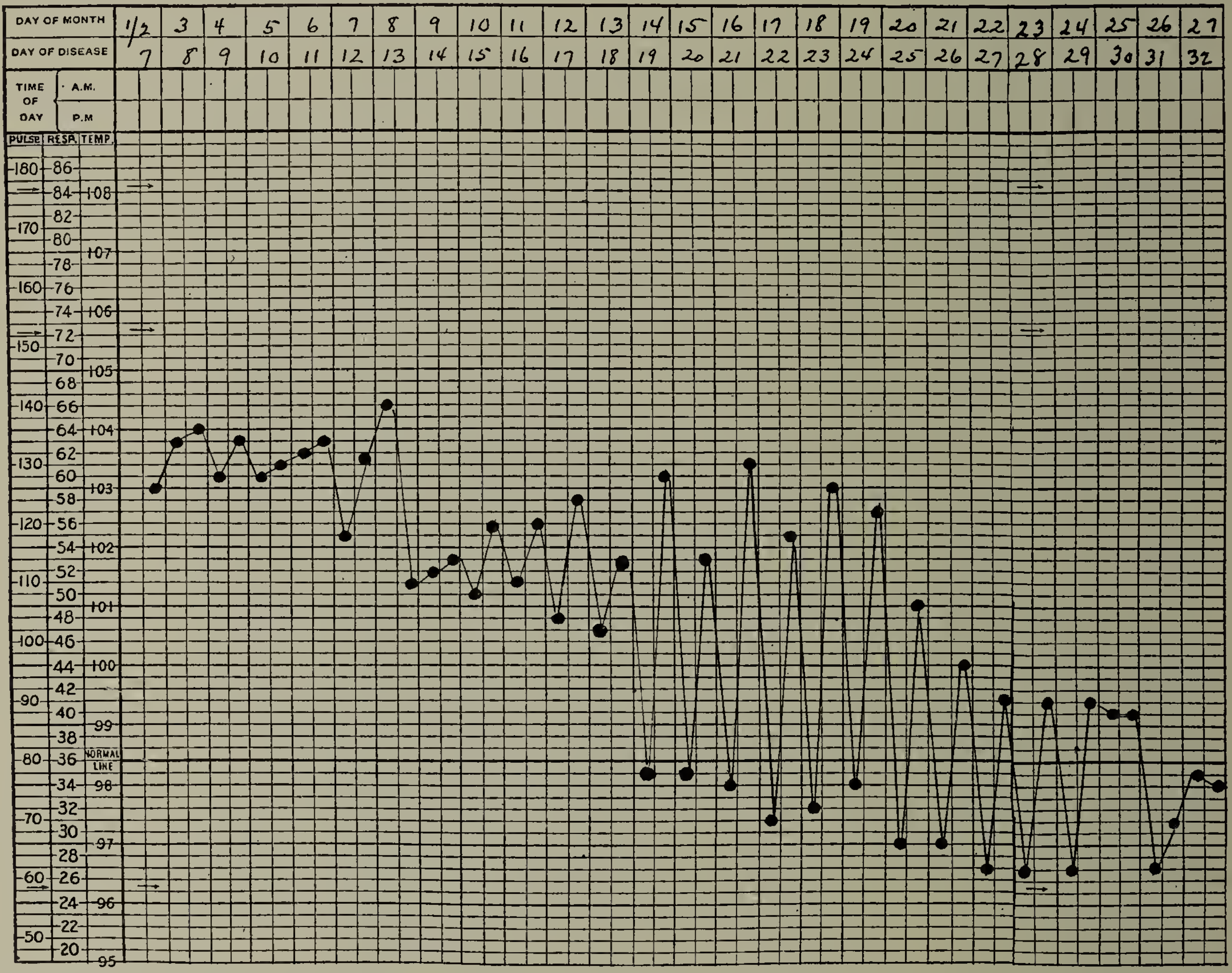

FIG. 102.—TYPHOID FEVER, LATER CHILDHOOD. SEVERE CASE OF ADULT TYPE OF DISEASE.

Thelma M., aged 11 years. Suffered from headache, stuporous most of time, muttering delirium, carphologia, occasionally actively delirious, coated tongue, dry lips, troublesome abdominal distention.

(*B*) Based also upon the *symptoms* as a whole, regardless of age, the following varieties may be mentioned:

1. Abortive Form.—In this variety all the characteristic early symptoms may be mild (Fig. 103) or be present in the ordinary or a severe form (Fig. 104), but the course is greatly abbreviated, lasting only 8 to 10 days. These cases are not infrequent, being much more common than in adults.

2. Mild Form.—This variety may be abortive also, as regards its duration, but the term is much better applied to those cases in which all the symptoms are peculiarly mild although the attack is not unusually curtailed. It is the form very commonly seen in early childhood. There is no diarrhea, loss of appetite, or prostration; and, in fact, practically no subjective symptoms except possibly slight headache and a trifling degree of apathy. Only the continued fever of moderate degree, the Widal reaction, and possibly the discovery of rose spots and enlargement of the spleen or of bacilli in the blood indicate the presence of the disease. (Fig. 105.) Even the temperature may in occasional cases scarcely exceed 100°F. (37.8°C.) (*Afebrile form*). Sometimes only the occurrence of the case in a family outbreak makes the diagnosis clear. Interesting cases of this afebrile typhoid fever are reported by Meyer,[1] confirming the observation of former investigators to the effect that the disease may run an entirely afebrile course.

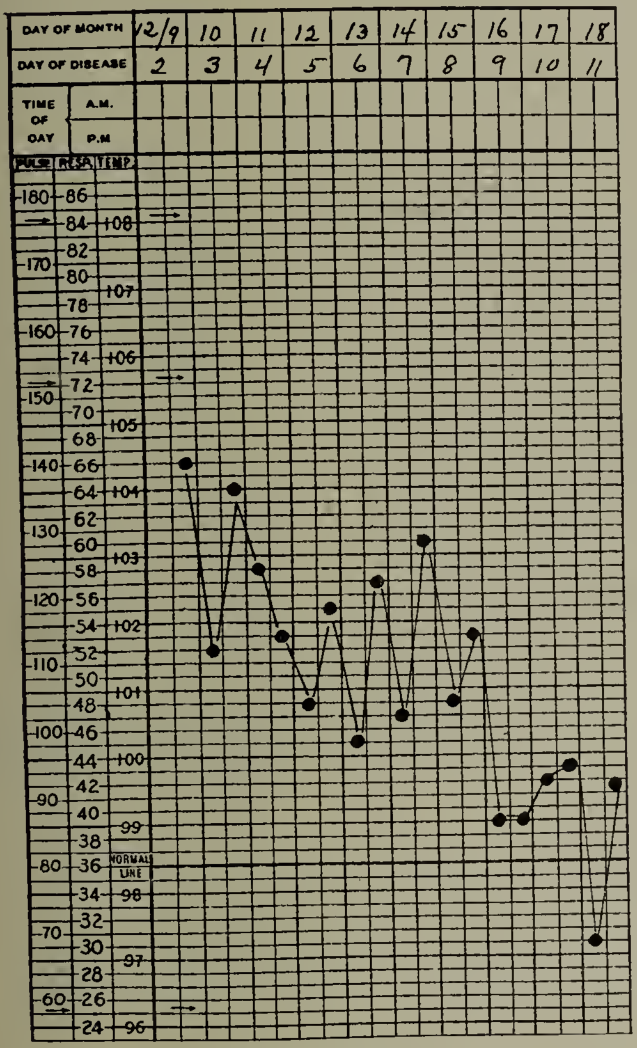

FIG. 103.

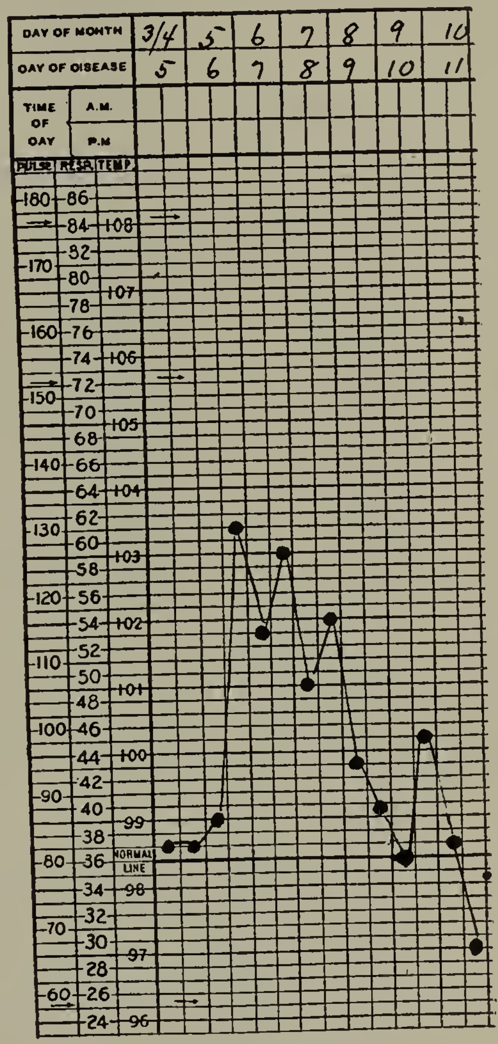

FIG. 104.

FIG. 103.—ABORTIVE TYPHOID FEVER, COURSE SHORT AND SYMPTOMS MILD FROM BEGINNING.

Thelma M., aged 11 years. Onset sudden. Symptoms consisted solely of slight weakness and diffused aching, fever, splenic enlargement. Fever ceased on 9th day and child out of bed on 11th day of disease. Case supposed to be influenza until a positive Widal reaction and later a very severe relapse (Fig. 102) showed the nature of the original attack.

FIG. 104.—ABORTIVE TYPHOID FEVER, SEVERE INITIAL SYMPTOMS.

Annie A., aged 7½ years. Symptoms of invasion consisted of fever, very frequent vomiting, diarrhea. Then improved, felt perfectly well and was bright, no apathy, vomiting and diarrhea ceased. Enlargement of spleen and roseola (?) present. Widal reaction positive. No fever after 10th day of disease.

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[1] Münch. med. Wochenschr., 1915, LXII, 1367.

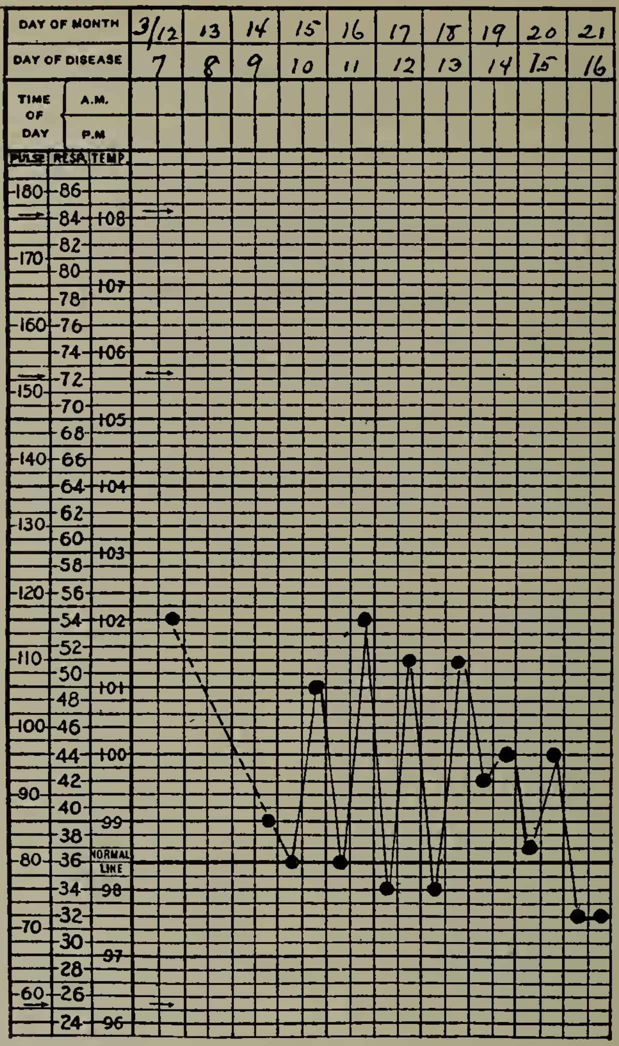

FIG. 105.—MILD TYPHOID FEVER.

Daniel B., aged 12 years. At no time any suggestive symptoms whatever complained of, no apathy. Spots found Mar. 12, assumed to be the 7th day of the disease. Positive serum-reaction.

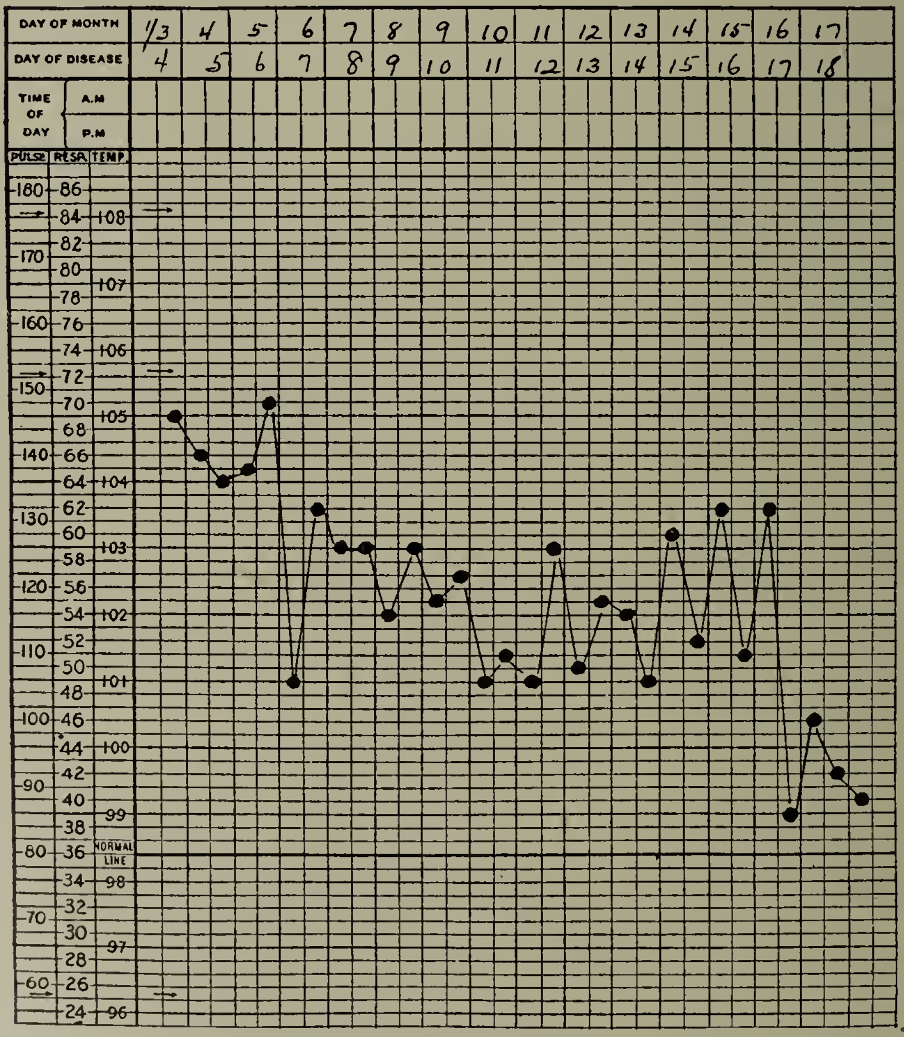

FIG. 106.—NERVOUS FORM OF TYPHOID FEVER. (MENINGITIC TYPHOID.)

John S., aged 10 years. Sudden onset with nose-bleed and severe headache. Wildly delirious by next day, with headache and vomiting. Condition continued and on 6th day of disease was unconscious, delirious, lying on side in gun-hammer position, well-marked abdominal tache. By 8th day of disease all nervous symptoms had greatly improved and the ordinary appearance of typhoid fever was present.

3. Nervous Form.—This occurs not infrequently, and is often the cause of great difficulty in diagnosis. The disease may begin abruptly as a pseudomeningitis, with repeated convulsions continuing several days; or there may be unconsciousness, grinding of the teeth, intense restlessness, rigidity of the neck, and possibly an unusually active delirium. (*Meningitic typhoid*) (Fig. 106). These early symptoms may soon give place to the more usual ones of the disease. Sometimes, however, particularly in older children, severe nervous symptoms may not develop until later. In these cases the spinal fluid is usually normal except sometimes for increase in amount.

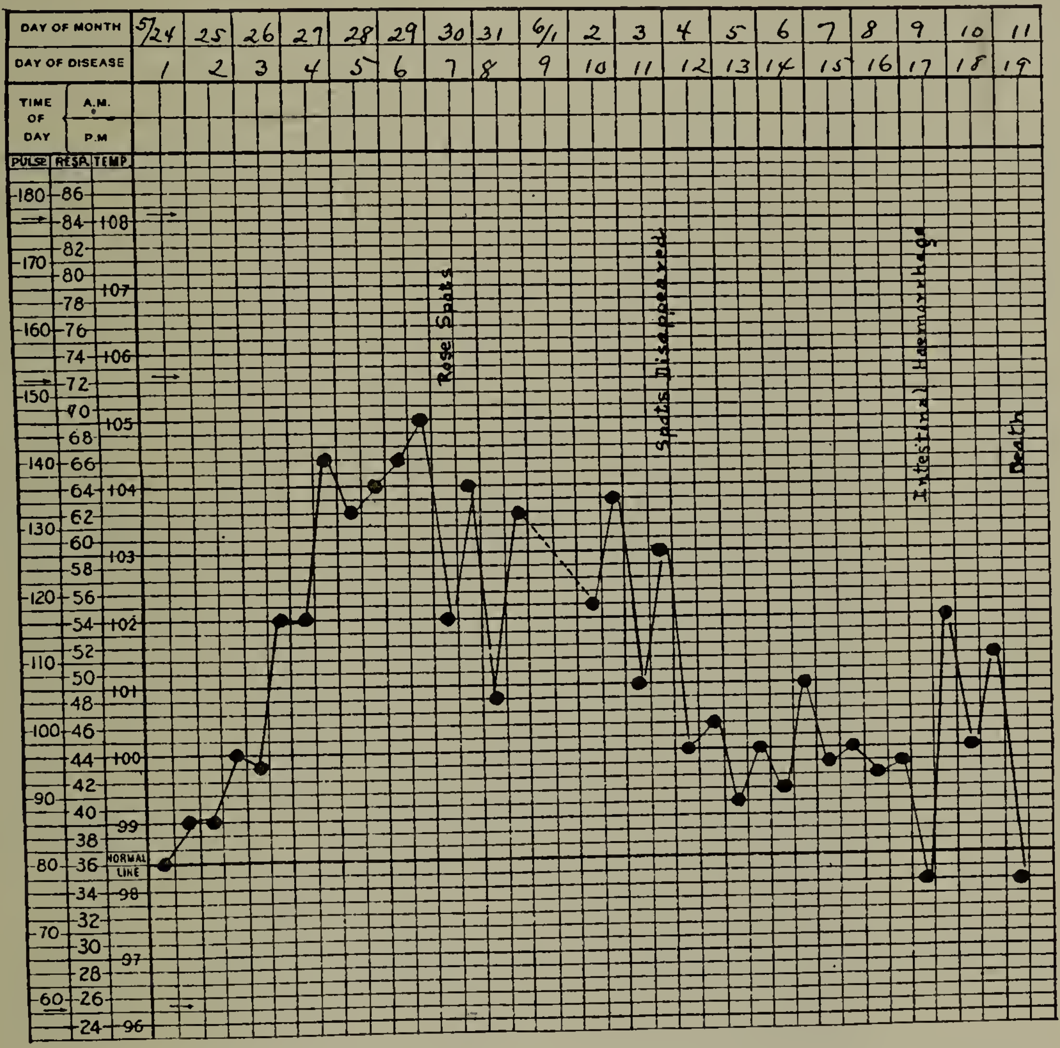

FIG. 107.—TYPHOID FEVER WITH FATAL INTESTINAL HEMORRHAGE.

Martin D., aged 5 months. Case shows short duration of 1st and 3d stages as well as of the attack as a whole. Case supposed at first probably to be pneumonia, although there were no positive symptoms of any sort. Chart shows temperature from the day of onset. Child suffered from restlessness, crying, loss of appetite, diarrhea, vomiting, later rose spots and enlarged spleen. Improved rapidly then developed intestinal hemorrhage with return of vomiting, abdominal pain and tenderness, feeble circulation. Death Autopsy. Later found that the mother also was in the Philadelphia Hospital with typhoid fever.

Complications and Sequels.—Complications and sequels are not common in average cases in children. Those of the *respiratory tract* are perhaps most frequent. Bronchitis may be present, especially in the severer cases of the disease. Pneumonia occurs, although perhaps less often than in adults. Severe laryngitis is an occasional complication. I have known a child to be aphonic for 5 or 6 weeks. In rare instances ulceration with stenosis may occur and intubation or tracheotomy be required. In 1 instance a tracheotomy tube was still being worn after 3 years. Some of the cases of laryngeal involvement depend upon a laryngeal perichondritis. I have observed this in a few instances. Pleurisy is unusual. Abscess and gangrene of the lung have both been reported, and hypostatic congestion may occur in severe cases. Disorders of the *circulatory apparatus* are for the most part infrequent. Pericarditis and endocarditis are rare, venous or arterial thrombosis very exceptional. More or less anemia is a natural sequel in severe cases. A hemorrhagic tendency with the production of purpuric eruptions, bleeding of the gums and other evidences of the hemorrhagic diathesis, is uncommon at any time of life (*Hemorrhagic typhoid*). Epistaxis may be so severe that it may be ranked as a complication. I have seen it the direct cause of death. Disturbances of the *digestive system* are important. Parotitis is only occasionally seen, but generally proceeds to suppuration (Vol. II, p. 24, Fig. 242). Severe aphthous stomatitis is sometimes observed, or ulcerative stomatitis going on to necrosis of a small portion of the bone with loss of teeth. Pseudo-membranous tonsillitis occasionally occurs. Noma, although uncommon, is more liable to develop after typhoid fever than after any other disease except measles. Severe diarrhea is not infrequent in later childhood. Fecal impaction is an unusual complication, of which I have seen but 1 instance. Intestinal hemorrhage is rare as compared with adult life, and is met with almost only in children of 10 years or older. Only 9 cases; *i.e.*, 1.6 per cent. of 553 cases of typhoid fever in chil-

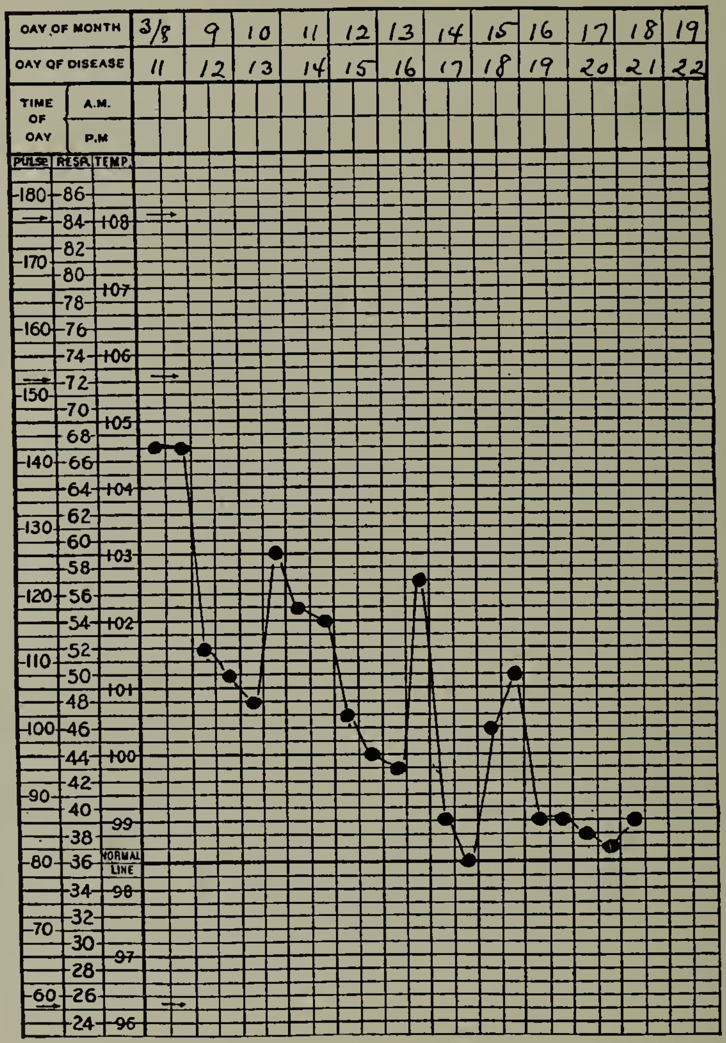

FIG. 108.—TYPHOID FEVER WITH INTESTINAL PERFORATION, ILLUSTRATING ABSENCE OF CHARACTERISTIC SYMPTOMS OF PERFORATION AS OFTEN SEEN.

Mary B., aged 4 years. Admitted to the Children's Hospital Mar. 8. Said to have had slight symptoms of typhoid fever for 10 days. Attack ran a mild course, although with considerable abdominal distention. Vomited twice in the morning of the 18th and twice in the afternoon. Had occasional slight abdominal pain at first, no distention or tenderness at any time. Sat up in bed through the morning and did not look ill. In the afternoon looked more ill and temperature had risen to 105.6° F. by 6 P. M. Strength now failed rapidly, but entirely without abdominal symptoms. Died 6 A. M. on the 19th. Autopsy showed perforation.

dren collected by Morse,[1] suffered from this symptom. I have, however, observed it in many more cases than this, in 1 instance in an infant of 5 months (Fig. 107). Costinesco[2] reported it 22 times in 762 cases of typhoid fever in children. It has been witnessed in some of the congenital cases, but is then rather a manifestation of a general hemorrhagic state dependent upon the septic condition of the blood. Intestinal perforation is another unusual complication in early life. Henoch[3] saw it but once in 381 cases, Morse[4] not at all in 284 cases, Reunert[5] 4 times in 471 cases, and Schultz[6] but 8 times in children. It is, however, not so rare as often supposed. Montmollin[7] reported it 7 times in 90 cases in children, Adams[8] in 11 of 337 cases and Setbon[9] 26 times in 1506 collected cases. Out of 289 cases of operation for typhoidal perforation collected by Elsberg[10] 25 occurred in children less than 15 years of age, and Jopson and Gittings[11] add 45 reported cases to this list. I[12] have personally observed 9 instances, 6 of these cases previously reported, 1 being a girl of 4 years and another of 6 years. Generally, however, it occurs only in patients near the end of later childhood. It is to be noted that the symptoms of perforation in early life are not infrequently much less marked than in adults and very misleading, and that the diagnosis is often extremely difficult. The usual fall of temperature with symptoms of collapse and severe abdominal pain may entirely fail to develop. This failure occurs more frequently than is the case in adults (Fig. 108). Greenwald and Eliasberg[13] record a case of peritonitis in an infant of 16 months, which developed without intestinal perforation. They could find only 1 other such occurrence reported in childhood. Typhoidal cholecystitis is probably of much more common occurrence than formerly supposed. Reid and Montgomery[14] collected 18 cases in children. We have observed it only once. It may rarely exhibit itself in an acute form with perforation and secondary peritonitis, as in cases reported by Bittner.[15] Slight pain and tenderness in the region of the gall-bladder, disappearing spontaneously in a short time, is of frequent occurrence.

Among *nervous complications* and sequels a temporary aphasia is more liable to occur in early life than later. Henoch[16] observed this in 20 of his 381 cases. I have seen it complete for several weeks. The children sometimes appear to be suffering not so much from inability to express their thoughts in words, as from a dullness of mind which has removed the desire. Post-typhoidal insanity is a sequel decidedly rare in children. Adams,[17] among others, reported 4 cases, and I have observed it a number of times. Chorea is a not infrequent sequel.

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[1] Bost. Med. and Surg. Jour., 1896, CXXXIV, 205.
[2] Thèse de Paris, 1897, 35.
[3] Kinderkrankheiten, 1895, 770.
[4] Bost. Med. and Surg. Jour., 1896, CXXXIV, 205.
[5] Deutsch. med. Wochenschr., 1889, XV, 1063.
[6] Jahrb. d. Hamburgischen Staatskrankenanstalten, 1889, I, 7.
[7] Thèse Neuchatel, 1885.
[8] Arch. of Pediat., 1904, XXI, 81.
[9] Thèse de Paris, 1902.
[10] Ann. of Surg., 1903, XXXVIII, 71.
[11] Amer. Jour. Med. Sci., 1909, CXXXVIII, 625.
[12] Amer. Jour. Med. Sci., 1905, CXXX, 581.
[13] Amer. Jour. Dis. Child., 1925, XXIX, 365.
[14] Bull. Johns Hopk. Hosp., 1920, XXXI, 7.
[15] Prag. med. Wochenschr., 1914, XXXIII, 279.
[16] Kinderkrankheiten, 1895, 771.
[17] Trans. Amer. Ped. Soc., 1896, VIII, 177.

Purulent meningitis, which may be due to the typhoid or other germ, is a very unusual complication. Cole[1] collected reports of 14 cases including his own, and Baumgartner and Olsen[2] tabulated 13 others since recorded. The literature is reviewed by Weston and Radbill[3] who report 2 cases due to the typhoid organism, one of which appeared to be primary in the meninges. The great majority of the cases showing meningeal symptoms are suffering from meningismus or a serous meningitis. Paralysis resulting from neuritis has been reported, and rarely a hemiplegia of cerebral origin. *Otitis* is a complication occurring much more frequently than in adults. It is often non-purulent, producing decided but temporary deafness; often purulent with consecutive perforation. Probably oftener the deafness appears to depend upon an inflammation of the auditory nerve or of the labyrinth. Purulent otitis was reported in 2.7 per cent. of Adams[4] cases.

Affections of the *genito-urinary apparatus* may occur. Nephritis is an occasional complication much less often seen than in adults. It generally recovers as convalescence from the typhoid fever proceeds. Cystitis and pyelitis are not common sequels in early life. *Suppurative processes* in various regions may take place. One of the most troublesome forms is furunculosis, which may be very extensive and severe. Subcutaneous abscesses are often observed. Bed-sores are very much less frequent than in adults and are observed only in neglected cases in older children. Suppuration of the mesenteric glands may occur, and cases have been reported in which the symptoms strongly suggested intestinal perforation (Rowland).[5] Suppuration of the joints or bones and necrosis of the bones are occasional sequels. The typhoid spine may sometimes be observed in early life, though less often than in adults.

Cutaneous eruptions sometimes occur as complications. A rubeoloid and, less often, a scarlatiniform rash are occasionally seen, and sometimes urticaria. These may be present either early or later in the attack. Sudamina are very frequent. Herpes is rare, but probably more common than in adult life. Other *acute infectious diseases* may complicate typhoid fever or immediately precede or follow it. This is true, for instance, of scarlet fever, measles, rubella, varicella, pertussis, diphtheria, cerebrospinal fever, malaria, and erysipelas. The occurrence of a secondary diphtheria seems to be especially frequent.

Relapse.—Relapse certainly is not less, and perhaps more, frequent than in adult life. Koplik and Heiman[6] in an analysis of 160 cases of typhoid fever in children reported relapse in 15 per cent.; and Adams,[7] in 550 cases, 8.7 per cent. with relapse. In my series of 75 cases in the first 2½ years of life there were 3 undoubted instances of relapse. It develops even as early as the 3d or 4th, but oftenest from the 14th to the 17th, day after the temperature of the first attack has reached normal. Relapse must, of course, be sharply distinguished from the *recrudescence* of fever which lasts but a day or two and is brought about by many slight causes (Fig. 97). It is characterized by a return of the usual symptoms, including the reappearance of the roseola and of the enlargement of the spleen. Not uncommonly some degree of splenic enlargement persists during the afebrile interval. The relapse may equal, exceed, or fall below the first attack in duration and severity. It is generally of shorter duration. The age of the child seems to exert no decided influence on the tendency to its development. In 1 instance it occurred in a female infant of 9 months (Fig. 109). Even more than one relapse may be observed. In 1 case under my observation 3 relapses were witnessed; and David[1] reported 5 relapses in a boy of 11 years.

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[1] Johns Hopk. Hosp. Rep., 1904, XII, 299.
[2] Arch. Int. Med., 1920, XXV, 537.
[3] Arch. of Pediat., 1926, XLIII, 160.
[4] *Loc. cit.*
[5] Jour. Amer. Med. Assoc., 1906, XLVI, 507.
[6] Arch. of Pediat., 1907, XXIV, 1.
[7] *Loc. cit.*

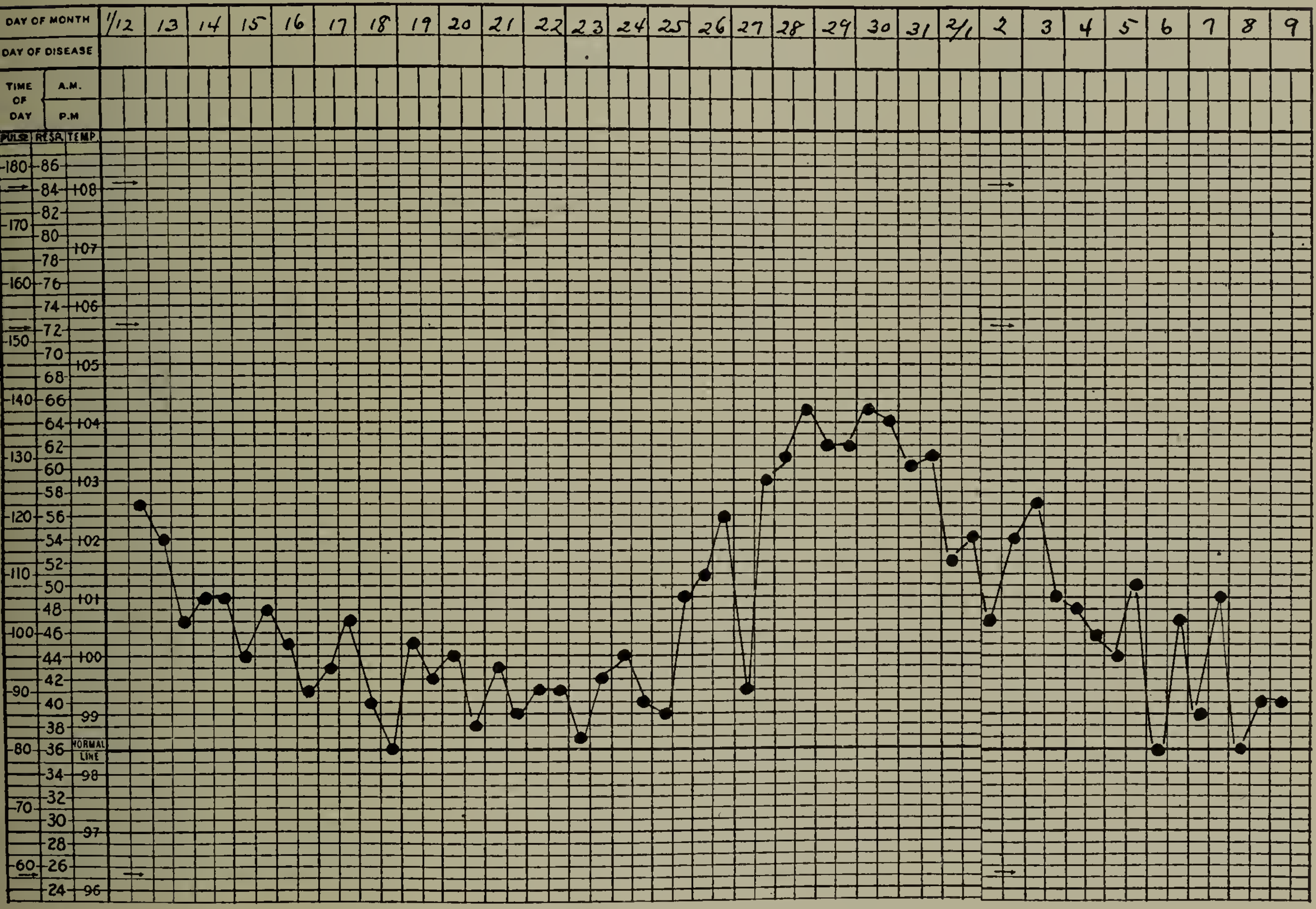

FIG. 109.—RELAPSE IN TYPHOID FEVER.

Irene T., aged 9 months. Showing relapse with return of fever, roseola, and splenic enlargement.

Recurrence.—One attack of typhoid fever usually confers lasting immunity. This is, however, by no means so generally true as in the case of scarlet fever and measles. The immunity may be only a temporary one. Even 3 or 4 attacks of typhoid fever may rarely occur in the same person.

Prognosis.—The mortality of typhoid fever in early life is, on the whole, less than later. In the 192 cases in children reported by Schavoir[2] only 2 died, a mortality of 1 per cent. This is exceptionally low. In Morse's[3] 284 cases the mortality was 6 per cent. against that of 13.5 per cent. in 3396 adult cases. Of 432 cases occurring in the Children's Hospital of Philadelphia during 14 years 23; *i.e.* 5.32 per cent., died. In general the mortality may be placed at from 4 to 5 per cent., yet this varies very much with the *age*. In infancy the number of fatal cases is large. Of 278 collected cases[4] not over 2½ years of age 57 per cent. died. Although this figure is exceptionally high, due probably to the fact that very many of the milder cases were not considered worthy of being reported, and is not to be considered representative, it serves to show that the disease at this period is much more fatal than later. In the later reported[1] 75 cases under 2½ years the mortality was 12 per cent. In early childhood the mortality is at its lowest and probably does not exceed from 2 to 4 per cent. It increases steadily as the age of puberty is approached. The lesser mortality of early life depends in part upon the lesser severity of the disease, and in part upon the lesser frequency of dangerous complications and sequels.

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[1] Zentralbl. f. inn. Med., 1912, XXXIII, 1071.

[2] Med. Rec., 1895, XLVIII, 803.

[3] Bost. Med. and Surg. Jour., 1896, CXXXIV, 205.

[4] Griffith and Ostheimer, Amer. Jour. Med. Sci., 1902, CXXIV, 868.

Among *unfavorable prognostic symptoms* are very severe diarrhea; obstinate vomiting; persistent dryness of the tongue; a marked degree of tympany; a weak, rapid pulse; and the development of unusual degrees of stuporous mental states or other nervous phenomena. The disappearance of the diazo-reaction is claimed to be a favorable symptom, indicating that a fall of temperature will occur in a few days (Rolleston).[2] The slow, irregular pulse of convalescence is not an unfavorable symptom.

Diagnosis.—This offers many difficulties in early life, especially in infancy. In typical cases the continued fever with no other discoverable cause, the enlargement of the spleen, the rose spots, the agglutinative reaction, the absence of leucocytosis, the discovery of typhoid bacilli in the feces, urine ane blood, and the occurrence sometimes in family epidemics serve to render the diagnosis easy, although it may be days before any conclusion can be reached. Most important is the agglutinative reaction, and it is upon this that the diagnosis must rest in many obscure cases. Yet in many instances in which other symptoms are positive, the agglutinative reaction can at no time be obtained. This does not militate against the correctness of the diagnosis.

The absence of leucocytosis is often a valuable diagnostic sign, but the presence of this condition does not exclude typhoid fever. The diazo-reaction is only a corroborative indication since it may occur in some other infectious fevers. Continued temperature without evident sufficient cause is always suspicious, but as this may develop also in acute miliary tuberculosis, grippe, ileocolitis, low-grade bronchopneumonia and other conditions, it cannot be regarded as a safe diagnostic symptom.

Typhoid fever is liable to be confounded in early life with a number of other affections. First among these, especially in infancy, is *ileocolitis*. Diarrhea is quite common in typhoid fever at this period of life and tympanites may occur in either disease. The intestinal condition in ileocolitis is, however, generally much more severe and out of proportion to the degree of fever and a leucocytosis is usually present. Later the discovery of rose spots with decided enlargement of the spleen and a positive agglutinative reaction may make the diagnosis of typhoid fever clear.

Malaria of a continued febrile type may simulate typhoid fever very closely. Leucocytosis is absent in each, and enlargement of the spleen present. The discovery of the plasmodium and the absence of the Widal and diazo-reactions serve to distinguish malaria. The course of the temperature, with regular intermissions or remissions, is likewise characteristic.

Grippe is, at times, especially in the early stage, readily supposed to be typhoid fever. Later the short course of the attack and the development of characteristic symptoms serve to distinguish it. There are not infrequently instances, however, in which the fever of grippe is unusually prolonged, the prostration decided, and the symptoms little characteristic, and these cases may cause much perplexity. Leucocytosis may be absent in grippe and enlargement of the spleen present in both diseases. The discovery of the Widal reaction and of rose spots will settle the diagnosis.

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[1] Griffith, Arch. of Pediat., 1912, XXIX, 565.

[2] Lancet, 1905, I, 290.

Acute miliary tuberculosis in many instances resembles typhoid fever closely. It exhibits a fever of the continued type, the absence of leucocytosis, and the presence of splenic enlargement without any discoverable cause or any localization of the tuberculous process. The subcutaneous tuberculin-reaction cannot be sought for on account of the continued presence of fever, and the cutaneous reaction is not to be depended upon in this condition. The persistent failure of the Widal reaction and of rose spots to appear, the continuance of the pyrexia beyond the duration of that of typhoid fever, and the possible development later of localizing symptoms in the brain or the lungs may finally serve to distinguish tuberculosis. Sometimes a decided dyspnea without discoverable pulmonary lesions to account for it is present in miliary tuberculosis. Occasionally tubercles may be discovered in the choroid. Often however, the diagnosis cannot be made during life.

A *continued fever oftenest of intestinal origin,* dependent probably on a mild toxemia the result of chronic digestive disturbances, often occasions difficulty in diagnosis, especially in the first 2 years of life. Fever of this nature is liable to be of very irregular type, and moderate diarrhea is often, although not always, present. Only careful watching of the course of the case and the continued absence of the rose spots, enlarged spleen and Widal reaction serve to exclude the presence of typhoid fever.

Meningitis may at first resemble typhoid fever. The vomiting and apathy often seen in the latter may suggest tuberculous meningitis, while the cases of typhoid fever of the meningitic type, beginning with convulsions and with severe cerebral symptoms, may point to cerebrospinal fever. In tuberculous meningitis, however, there is a tendency to leucocytosis, and in cerebrospinal fever the leucocytes are very much increased in number. The diazo-reaction is present in both forms of meningitis as well as in typhoid. The meningitic symptoms of typhoid fever, however, generally occur early and disappear soon. They consist usually, too, of symptoms of excitement, and rarely in children assume the form of paralysis and coma. Kernig's sign is of little value as a differential symptom. In doubtful cases the continued absence of the Widal reaction and the results of lumbar puncture serve to distinguish meningitis.

Treatment. Prophylaxis.—The prevention of the direct spread of the disease from the patient is to be sought by careful disinfection of the urine and feces and of all the bed and body linen; the linen by submerging in a 5 per cent. carbolic acid solution and afterward by boiling; the excretions by mingling them with this solution or with equal parts of chloride of lime and water. The hands of the attendants should be washed and disinfected after handling the patient. The convalescent patient cannot be considered safe so far as the transmission of the disease to others is concerned until 2 or 3 bacteriological examinations of the urine and feces show that bacilli are no longer to be found. Hexamethylenamin or hexylresorcinol may often be given with advantage to free the urine from germs in patients where they tend to persist. It is interesting to note that extirpation of the gall-bladder in the case of chronic carriers does not ensure cure of the condition. In our case of typhoidal cholecystitis previously mentioned, with recurring fever, local symptoms and persistence of typhoid-bacilli in material obtained by the duodenal tube, an intravenous injection of mercurochrome was followed by disappearance of the symptoms and of the organisms.

The prevention of extension in general is, of course, to be accomplished chiefly by the employment of water which is entirely above suspicion. When this cannot be obtained all water to be used for drinking and bathing, and for the washing of vegetables, fruits, eating utensils, nursing bottles and nipples, and the like, should be boiled. The systematic heating of milk to a temperature of 60°C. (140°F.) for 5 minutes will destroy any typhoid germs present.

Immunizing Treatment.—The work which was originally done in this line by Wright,[1] Pfeiffer and Kolle,[2] and others, employing injections of cultures in which the germs had been killed by heat, has long since passed the experimental stage. The results reported in the United States Army by Russell,[3] Lyster[4] and others have been most convincing. In the American Expeditionary Forces during the late World War, where typhoid prevention was universally employed, the incidence of the typhoid group of diseases was less than 0.1 per cent. as compared with 20 per cent. for the Spanish-American War in which such preventive inoculation was not used (Vaughan).[5] Even if a vaccinated individual becomes infected with bacillus typhosus the course of the disease is likely to be mild, unless 8 months have elapsed since vaccination.

Treatment of the Attack.—This is purely expectant and symptomatic; the matter of the greatest importance being that of avoiding an excess of treatment. No specific treatment has yet been proven certainly effective, although the results obtained encourage further trial. Wright[6] employed inoculations with dead bacteria, Chantemesse[7] and Josias[8] injections of an immunizing serum, and Jež,[9] a liquid prepared from the organs of immunized animals and given by the mouth. Good results are reported by all. Favorable results with the serum in modifying the course of the attack in the case of children have later been recorded by Josias;[10] and with the vaccine by Ortiz, Acuña and Belloc,[11] Kharina-Marinucci,[12] Spolverini[13] and others. Bond and Barrier[14] claim good results with mercurochrome given intravenously. Treatment directed to the disinfection of the intestinal tract has not proven to be of any special value. This at least has been the experience of many observers as well as my own.

No matter how well the child feels, confinement to bed is imperative. This need not be continued as long after defervescence as is commonly required with adults. The diet should be easily digestible but abundant, one in which milk forms a prominent part being the best in most cases. Acidophilus milk may be used in place of sweet milk with good results

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[1] Lancet, 1901, II, 1107.
[2] Zeitschr. f. Hyg., 1896, XXI, 203.
[3] Jour. Amer. Med. Assoc., 1914, LXII, 1371.
[4] Jour. Amer. Med. Assoc., 1915, LXV, 510.
[5] Jour. Amer. Med. Assoc., 1920, LXXIV, 1074; 1145.
[6] Lancet, 1901, I, 399.
[7] Annals de l'instit. Pasteur, 1892, VI, 755.
[8] Ann. de méd. et de chir. inf., 1903, VII, 438.
[9] Wien med. Wochenschr., 1899, XLIX, 346.
[10] Acad. de méd., 1906, March 6. Ref., Arch. f. Kinderheilk., 1908, XLVII, 454.
[11] Arch. de méd. des enf., 1915, XVIII, 575.
[12] La Pediat., 1920, XXVIII, 641,
[13] La Pediat., 1921, XXIX, 289.
[14] Arkansas Med. Soc. Jour., 1924, XXI, 71.

(see p. 155). There is no necessity, however, of making milk the only food, and a regimen may well be used in which there is a high carbohydrate percentage in the form of gruels and the like. The fancies of the patient are to be humored as far as possible, since, although over-feeding is to be avoided, it is certain also that many patients are under-fed. Drinking water should be offered freely and often. As convalescence begins the appetite returns, and the diet should be decidedly increased in quantity and variety. This may usually be done earlier than has been the custom in adult cases, owing to the lesser degree of intestinal involvement present. The increase must, however, be made cautiously, since disturbance of digestion appears undoubtedly to favor the development of relapse, and certainly can cause a recrudescence of fever.

The surface of the body should be kept clean by sponging with water or alcohol and water, and the mouth should be washed with a solution of boric acid several times a day. The need of the patient for undisturbed sleep must be emphasized. It is well at night to lengthen decidedly the intervals between the administerings of medicines or other treatment, and to forsake regularity, utilizing the times when the patient wakes. Of course in very severe cases this suggestion is not applicable.

Treatment of special symptoms may next be considered. Of the *temperature* it is well to remember that children both attain decided elevation oftener and endure it better than do adults. Consequently mere elevation, if not prolonged and if unattended by unfavorable symptoms, need not cause alarm and does not require treatment. In fact the chief object of hydrotherapy is not so much the reduction of temperature as the stimulating effect upon the circulation and the controlling of nervous manifestations. Should the treatment seem indicated, either sponging or tubbing may be employed. Where there is active objection on the part of the child to the use of water it will be found that tubbing is not only more effective than sponging but much less troublesome both to the nurse and to the patient, since it requires a shorter time and causes no more, or less, opposition. Sponging, to have any antipyretic value, must be kept up for from 10 to 20 minutes and repeated frequently. Often the cold or warm pack is very efficient and disturbs the child but little. Cold tub-baths are, however, almost never required and children bear them badly. A graduated bath of 95°F. (35°C.) reduced by the addition of cold water to 85°F. (29.4°C.) is that most often serviceable. It may be given every 3 hours when the temperature exceeds 103°F. (39.4°C.), and continued from 5 to 10 minutes according to the effect desired and the tolerance shown. The child while in the bath should be rubbed vigorously and briskly as a stimulant to the circulation. The head should, meanwhile, be kept cool by the repeated application of cloths dipped in cold water.

Very often even the graduated bath causes a greater degree of cyanosis and of weakness of the pulse than can be considered safe. In such cases the warm tub-bath of 95° to 100°F. (35° to 37.8°C.) is frequently very efficacious. Yet not uncommonly even this is followed by imperfect reaction and should be abandoned. In other cases the intense excitement and opposition shown when a bath is given produces injurious fatigue. In fine, hydrotherapy, while a most valuable agent in typhoid fever, must never be employed in early life as a routine measure without a close study of its effects upon the individual case; otherwise more harm than good may result. If there is much shivering and blueness after the bath an alcoholic stimulant should be administered. Frequently it is advisable to precede it by this also. The temperature of the patient may be taken half an hour after the bath is over, to determine what degree of lowering has taken place.

The tub-bath is contraindicated in cases of intestinal hemorrhage and in subjects extremely prostrated, where moving would be too exhausting. In some cases a satisfactory reduction of temperature may be obtained by the nearly constant application of an ice-bag to the abdomen, with one or more layers of towel intervening. In infants ice must always be employed guardedly, as it is not always well tolerated. It is only exceptionally, where hydrotherapy cannot be employed and where it is desired to reduce unusually high temperature, that the cautious administration of coal-tar antipyretic drugs may be required. In the cases where the terminal stage of typhoid fever is unusually prolonged, or where an irregular temperature persists from causes entirely undiscoverable, the continued employment of fairly large doses of quinine is occasionally very serviceable. Sometimes these patients are finally most benefited by getting them out of bed into a chair. I have often seen apyrexia promptly follow this procedure.

Prostration demands stimulating treatment, especially by alcohol. While the use of alcohol, as of stimulants of any sort, is probably not a necessity in the majority of cases of typhoid fever, yet in small amounts it is often, I think, an excellent conserver of energy. The condition of the heart-strength is the chief guide. Any degree of prostration, weakness of pulse, nervous exhaustion, or impairment of the first sound of the heart necessitates stimulation by this or by other drugs. It is easier to maintain a fair condition of the pulse by moderate stimulation than it is to revive it if it has commenced to fail decidedly. The dosage of brandy or whiskey in typhoid fever at the age of 2 years may vary from ½ to 1 fl. dram (2 to 4) every 3 to 4 hours, depending upon the urgency. Cardiac weakness may call also for digitalis, camphor, caffeine or similar drugs. This is true, however, only of the bad cases.

Vomiting, if severe, requires that the milk be alkalinized, diluted, skimmed, peptonized, or entirely replaced for a time by other food, such as albumen-water, cereals, or broths free from fat. Frequently it renders it advisable to give drugs hypodermically as far as possible. *Diarrhea* need not be interfered with if there are only 4 to 5 moderate-sized stools daily. If more numerous, or if large and watery, the condition can generally be controlled by small doses of silver, bismuth, salol, some of the tannic acid derivatives, or opium. The last-mentioned drug must be given cautiously if there is any tendency to coma or to decided tympany. *Constipation*, although often requiring treatment, is generally not troublesome. No purgatives should be administered except early in the attack. Small glycerine or larger soap-and-water enemata may be employed. Glycerine suppositories are often useful. It need scarcely be said that any injections must be given gently without undue pressure.

Tympanites is occasionally sufficiently annoying to demand relief. The local application of turpentine stupes will generally relieve it satisfactorily. In other cases the careful insertion of a rectal tube is of benefit. Sometimes injections of soap-and-water or of milk of asafetida are useful. Asafetida by the mouth is frequently serviceable in this condition.

Nervous symptoms at times require treatment. As already stated, hydrotherapy is to be employed not so much for the mere reduction of temperature as for the alleviation of the attendant nervous phenomena. Delirium, sopor, great restlessness, headache, and the like, if associated with high temperature, are often benefited by hydrotherapy. In other cases we may use small repeated doses of bromides or of antipyrine or phenacetin. Sleeplessness may well be combated by bromides or veronal. A warm bath in the evening may suffice to relieve it.

Complications and sequels need treatment appropriate for them. In intestinal hemorrhage the foot of the bed should be elevated, an ice-bag applied to the abdomen, and morphine given hypodermically in sufficient dose to quiet intestinal peristalsis. Epinephrin in doses of 5 to 10 minims (0.31 to 0.62) of the 1:1000 solution, given subcutaneously, calcium chloride or lactate (5 to 10 grains (0.324 to 0.648) 4 times daily) or gelatin (10 per cent. solution) may be tried. Intestinal perforation demands operative interference at the earliest possible moment. The debility and anemia which often persist after severe cases of typhoid fever require medication with iron, strychnine and other tonics, and often a sojourn at the seashore or in the mountains.

PARATYPHOID FEVER

This is a disease which has come into considerable prominence in recent years. It is produced by the action of the paratyphoid bacillus, either of the varieties "A" and "B" being the agent. The latter is that very much most frequently found. Symptomatically the disorder generally resembles typhoid fever almost exactly, the chief distinction being that the agglutinative reaction with the typhoid bacillus is absent, while it is obtained with the variety of the paratyphoid bacillus which is the causative factor in the case. Sometimes, however, instead of the ordinary typhoidal symptoms, there are those of a dysenteric, choleriform, respiratory, or septic nature; the last especially in very young infants. The affection may occur isolated or in small epidemics, and may affect any age. It appears to be uncommon in the 1st year of life, but an instance of the A type occurring in an infant of 8 months is reported by Eckert[1] and a number of instances by others of infants with Type B. Congenital infection by the B bacillus was observed by Nauwerck and Flinzer,[2] Stolkind and Lorey[3] and others. The lesions appear to be very similar to those of typhoid fever. Decided ulceration of Peyer's patches is uncommon, but this is equally true of typhoid fever in early life. Moreover hemorrhage and perforation have been reported. The diagnosis can be made only by cultural studies of the germs and by the agglutinative reaction. In 117 children with digestive disturbances, not suggesting typhoid fever, Breuning[4] found 11 cases; *i.e.*, 9.5 per cent. with paratyphoid bacilli in the stools. As far as experience has yet extended the mortality appears to be decidedly less than that of typhoid fever. The disease as it occurs in infancy and childhood has been exhaustively reviewed by Cannata[5] and later by Marschhausen[6] and Stolkind and Lorey,[7] the last with long bibliography.

[1] Berl. klin. Wochenschr., 1910, XLVII, 1102.
[2] Münch. med. Wochenschr., 1908, LV, 1217.
[3] Brit. Jour. Child. Dis., 1918, XV, 161.
[4] Münch. med. Wochenschr., 1914, LXI, 1050.
[5] Annali di clinica medica, 1911, II, 285.
[6] Monatsschr. f. Kinderheilk, Orig., 1919, XV, 615.
[7] *Loc. cit.*

CHAPTER X

CEREBROSPINAL FEVER

(Epidemic Cerebrospinal Meningitis)

Although localizing itself largely upon the cerebrospinal meninges, and belonging with other forms of meningitis, the disease is so manifestly infectious and often epidemic, with a complex of symptoms so peculiarly its own, that it seems properly included with others in the category of Infectious Diseases. It probably existed at a much earlier period, but it was first clearly described by Vieusseux[1] in 1805 in Geneva, and shortly afterward in the United States (Danielson and Mann;[2] Strong[3]); and since then has appeared in different countries with varying frequency.

Etiology. Predisposing Causes.—Climate and season exert a decided influence, the disease being confined to temperate climates and the majority of epidemics beginning in cold weather. Defective sanitation in general is likewise important, and outbreaks are consequently peculiarly liable to occur among soldiers in camps. Trauma of the head, exposure to heat, and mental and physical over-exertion certainly predispose. The previous health and the existence of other diseases are without direct influence.

Age is a powerful etiological factor, children and adolescents being especially susceptible, and infants in the 1st year being in no way exempt. Of 2916 cases in the Silesian epidemic in 1905 reported by Flatten[4] 8 per cent. occurred in the 1st year, 47 per cent. from birth to 5 years, and 29 per cent. from 5 to 10 years. Of 2179 cases in the epidemic in New York City in 1904 and 1905 (Billings)[5] 15 per cent. were under 1 year and 67 per cent. under 10 years of age. It has been observed even in the new-born (Commandeur and Nordman;[6] Koplik;[7] Miller;[8] Barron;[9] Root[10]). Of the *individual susceptibility* little can be said with certainty. It is undoubtedly slight, since comparatively so few of those exposed are attacked. As a rule but a single case occurs in a family, although 2 or 3 or even more cases in a house are sometimes seen. *Epidemic influence* is very marked. Years may pass with but few cases in a locality, and then an outbreak may occur. These epidemics are generally limited in extent, perhaps to one city, while at other times a considerable part of a country, or even several countries, may be involved. The outbreak may continue for months or years and then cease entirely, or only sporadic cases develop. The severity of epidemics varies greatly in different localities and on different occasions.

[1] Hufeland's Jour. d. pract. Arzneykunde, 1805, XIV, 3 St., 181.
[2] Med. & Agricult. Register, Bost., 1806. Ref., Osler, Pract. of Med., 1903, 101.
[3] Dissertation on the Disease Termed Spotted Fever, 1810.
[4] Klin. Jahrb., 1905–6, XV, 211.
[5] Jour. Amer. Med. Assoc., 1906, XLVI, 1670.
[6] Lyon méd., 1907, CVIII, 1081.
[7] Arch. of Pediat., 1916, XXXIII, 481.
[8] Arch. of Pediat., 1917, XXXIV, 824.
[9] Amer. Jour. Med. Sci., 1918, CLVI, 356.
[10] Amer. Jour. Dis. Child., 1921, XXI, 500.

Exciting Cause.—The disease is clearly an infectious one, now believed to be due to the *diplococcus intracellularis meningitidis,* or meningococcus, described by Weichselbaum[1] in 1887; for although a symptom-complex resembling that of cerebrospinal fever may undoubtedly be produced by other germs, the disease occurring as a primary affection is generally dependent upon the meningococcus (Councilman),[2] and cases with other bacteriological relationships are better classified under Meningitis Simplex. (See Vol. II, p. 608.)

The germ was first obtained by lumbar puncture from patients during life by Heubner[3] who succeeded also in producing the disease in animals by inoculation. It is a diplococcus, different in form from the pneumococcus, and having many resemblances to the gonococcus, being, as this is, Gram-negative, although not so invariably. It is found in large or small numbers in the inflammatory exudate, chiefly within the cells; frequently on the nasal and pharyngeal mucous membrane, but only, as a rule, comparatively early in the attack; sometimes in the blood (Haden;[4] Bloedorn[5]), and also in the purpuric eruption which may accompany the disease (Babes;[6] Netter, Salamier and Blancher;[7] Sharpe;[8] Brown[9]). Goodwin and v. Sholly[10] observed it in the nose in the first 2 weeks in 50 per cent. of the patients. It has been discovered also on the conjunctiva. Of very important bearing upon treatment is the fact as pointed out by Gordon[11] and others that there are different strains of the meningococcus, as shown by their immunological reactions. At least two types prevail, one responsible for 75 to 80 per cent. of the cases (Flexner).[12]

Vitality of the Germ.—Nothing is certainly determined regarding the *life-history* of the germ outside the body. Its vitality appears to be slight. It grows badly on most culture media, and it is readily killed by low or high temperature, or by drying. Even from the spinal fluid as obtained by puncture the germ disappears soon; often long before the patient has recovered. Occasionally, however, it persists for months. In chronic cases of the intermittent form the germ may sometimes be found only during the exacerbations. Sometimes it is not discovered at all until the disease is well advanced.

Mode of Transmission.—The method of transmission of the disease is not definitely known. Soil and water appear not to be factors. Whether it is carried by the air to any extent is doubtful. Spread of the infection by domestic animals is possible. Only in rare instances has the conveyance by clothing been proven. Direct transmission from the sick to the well is the exception. I have never seen a case develop in a hospital-ward in which other cases of the disease were under treatment, and this has been a frequent experience of others also. Spreading by the schools does not seem to occur.

[1] Fortsch. d. med., 1887, V, 573.
[2] Jour. Amer. Med. Assoc., 1905, XLIV, 997.
[3] Jahrb. f. Kinderheilk., 1896, XLIII, 1.
[4] Arch. Int. Med., 1919, XXIV, 514.
[5] Amer. Jour. Med. Sci., 1921, CLXII, 881.
[6] Bull. Sect. Scient. Acad. Roum., 1915–16, No. 10, 368. Ref., Jahrb. f. Kinderheilk., 1917, LXXXV, 151.
[7] Brit. Jour. Child. Dis., 1917, XIV, 264.
[8] Arch. of Pediat., 1916, XXXIII, 641.
[9] Amer. Jour. Dis. Child., 1924, XXVII, 598.
[10] Research Lab. Dept. of Health, New York City, 1905, I, 177.
[11] Kennedy and Worster-Drought, Brit. Med. Jour., 1917, I, 261.
[12] Jour. Amer. Med. Assoc., 1918, LXXI, 638.

It is a noteworthy fact, however, that the microörganisms have repeatedly been found on the respiratory mucous membrane of healthy individuals during the existence of epidemics, and it is very possible that dissemination occurs in this way. Goodwin and v. Sholly[1] found them in the nose in 10 per cent. of those in contact with patients, and Kutscher[2] in the nasal and pharyngeal mucus in 75 per cent. In spite of this prevalence of carriers, the acquiring of the disease through them is uncommon. Other investigators think that infection by the food is probable. The *mode of entrance* of the germ into the body is also unknown. The fact that it is often found on the nasal mucous membrane of patients has led to the belief that it reaches the meninges directly, by penetrating the cribiform plate of the ethmoid bone; others believe its entrance is by the way of the lymph-vessels from the nasopharynx; and still others, that the portal of entrance is the intestine.

Pathological Anatomy.—The characteristic lesion is an acute fibrino-purulent inflammation of the pia-arachnoid of the brain and spinal cord. In malignant cases, fatal within a few hours, the dura and pia appear merely intensely congested, swollen, and possibly cloudy. Cellular infiltration may perhaps be discoverable only with the microscope. In cases lasting 2 to 3 days only a small amount of purulent exudate is found. In the severe cases which have continued a longer time subarachnoid exudate is evident; at first simply cloudy, but later usually distinctly fibrino-purulent. Over the cortex it occurs oftenest in streaks following the fissures and vessels; or it may be in the form of scattered yellowish or greenish-yellow placques, sometimes covering the greater part of the convexity. It is generally most abundant at the base, forming a uniform yellowish layer with much thickening of the meninges. The choroid plexus is involved, and the ventricles are dilated by cloudy or distinctly purulent fluid. On the cord it is situated chiefly over the posterior portion and especially in the regions below the cervical. The spinal nerve-roots and the sheath of the cranial nerves may be surrounded by the exudate.

The exudate consists chiefly of polymorphonuclear leucocytes in a fluid, which, although more or less fibrinous, is never so to the extent seen in pneumococcic meningitis. In the acute cases large cells are also discovered, probably derived from the connective tissue or the lining of the lymph-spaces. Meningococci, mostly within the cells, are present in the exudate as well as in the edematous meningeal tissue which is found between the areas of distinct cellular infiltration. In chronic cases, running a course of a month or more, the exudate has disappeared to a large extent, and its purulent character given place to a condition of a more mucous appearance. The meninges are left edematous and thickened and the ventricles may be greatly dilated.

The brain-tissue itself is affected to some extent, being congested and softer than normal, and exhibiting cellular infiltration together with meningococci in the superficial layers and especially along the vessels. In chronic cases cocci are scarce and found only with difficulty. The cranial nerves are infiltrated. The spinal cord shows similar changes but with fewer cocci. The nerve-roots and nerve-ganglia also exhibit evidences of inflammation.

The bones of the skull and the dura mater of the brain and cord are intensely congested. The spleen may be enlarged but is less often so than in most other acute infectious diseases. The lungs may exhibit bronchitis, hypostatic congestion, or the lesions of a complicating pneumonia, the consolidation being in the form of quite small foci consisting of purulent infiltration sometimes distinctly hemorrhagic (Councilman, Mallory and Wright)[1] and not developing in connection with the bronchi. These foci may be scattered, or massed in the form of croupous pneumonia.

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[1] Jour. Infect. Dis., 1906, Suppl. Vol., 21.
[2] Med. Klin., 1907, III, 314.

The lesions of endocarditis or pericarditis may occasionally be found. The heart, liver and kidneys exhibit degenerative changes, and sometimes the lesions of nephritis are present. Ecchymoses in the skin, punctate hemorrhages in the endocardium, abscesses in various parts of the body including the joints, suppuration in the internal ear, and inflammation of the eyeball are sometimes met with.

Symptoms.—ORDINARY FORM.—The clinical picture of the disease varies greatly in different subjects, but certain clearly defined forms are recognizable. A general description of the ordinary type follows:—The duration of *incubation* is not definitely known. In a few cases Netter[2] reported it as between 3 and 11 days, and Flatten,[3] in his studies on the Silesian epidemic, from 3 to 4 days or less. Bolduan[4] placed it at from 1 to 4 days. It seems impossible to reach a positive conclusion. The attack may be ushered in by *prodromes* lasting 1 or 2 days, and consisting of malaise, headache, vertigo, chilliness, pain in the back, and loss of appetite. As a rule, however, the *onset* is sudden, with fever, severe headache, prostration, vomiting, severe pain in the neck, back and limbs, and sometimes convulsions (Fig. 110). Very rapidly a peculiar degree of stiffness of the neck develops, and in well-marked cases decided retraction of the head as well. The slightest forcible moving of the head causes a cry of pain. Delirium, which is sometimes violent, great restlessness, and irritability are common. Sensitiveness to light and noise, and cutaneous hyperesthesia are marked. As the disease advances vomiting, pain, and irregular fever continue, and more or less opisthotonos develops. There is often rigidity of the extremities. The face is congested, strabismus or alteration of the pupils appears, sleep is disturbed, and grinding of the teeth or general convulsions may occur repeatedly. There is frequent crying out with pain. Herpes or other eruptions of the skin may appear, if they have not done so at the beginning, prominent among them being those of a purpuric nature. As intracranial pressure increases the pulse becomes slow and often irregular, the respiration irregular, stupor replaces delirium, and complete coma follows. In infants with open fontanelle the increase in intracranial pressure is readily shown by the tension and bulging in this region.

The *duration* of the disease, even in what may be called average cases, is extremely variable. Roughly speaking it may be placed at from 2 to 4 weeks, not including convalescence, but may be much shorter or longer than this. Improvement is characterized by the diminution of the pain and rigidity, permanent lessening of fever, cessation of vomiting and bettering of the mental state.

Some of the symptoms must be considered more in detail.

Convulsive movements are frequent in infancy and early childhood; less so in older persons. They may be general and usher in the attack, and then cease; or recur at intervals later, being then either general

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[1] Epidem. Cerebro-Spin. Meningitis, 1898.
[2] 20th Cent. Pract. of Med., XVI.
[3] *Loc. cit.*
[4] Research Lab., Dept. of Health, N. Y. City, 1905, I, 140.

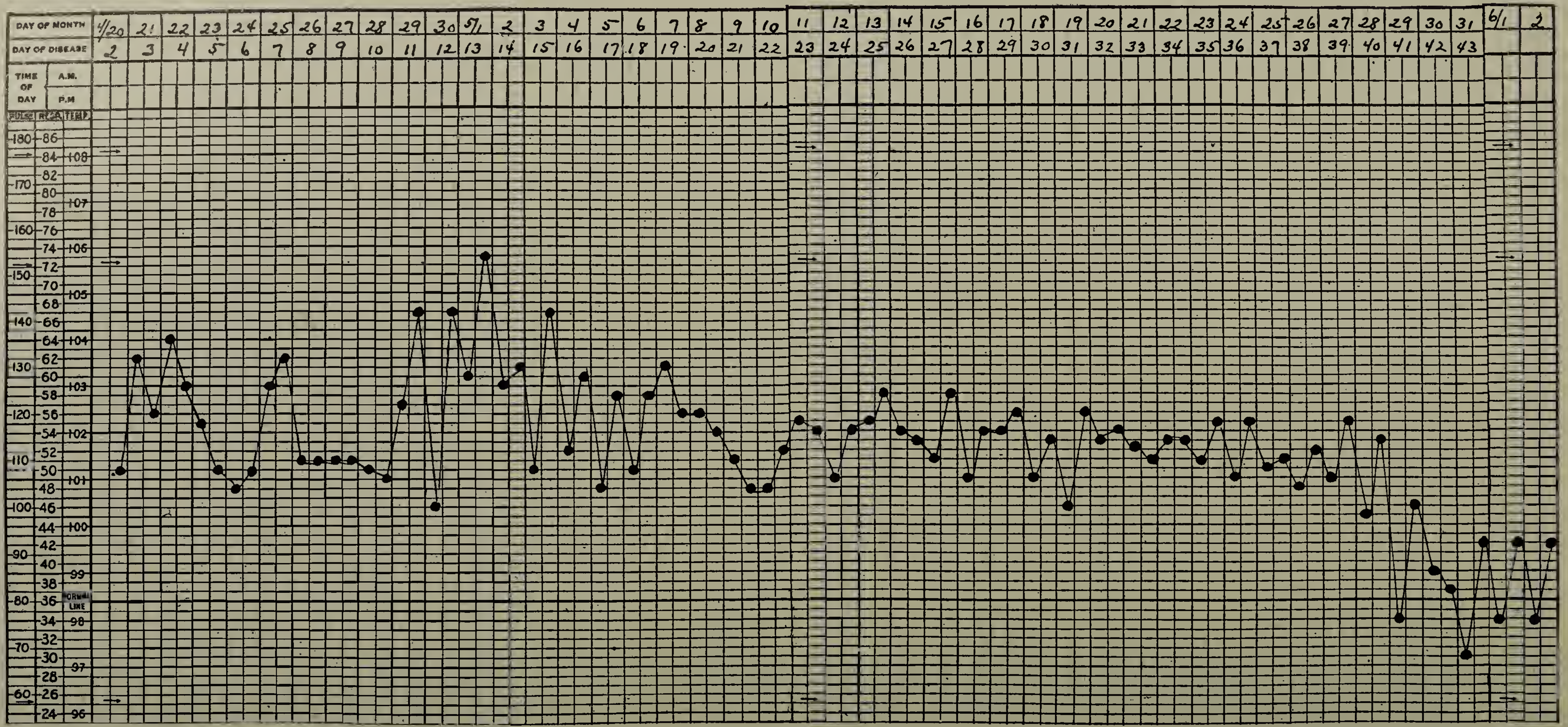

Fig. 110.—Cerebrospinal Fever, Ordinary Form.

Louis T. Taken ill suddenly, Apr. 19, with fever, vomiting, pain in head, and stupor. Two other children ill with the disease. Course of the attack characterized by stiffness of neck, irritability, stuporous condition, pain on moving, herpes, Kernig's sign and leucocytosis of 37,800. Very slow improvement began on Apr. 28 in spite of rise of temperature. Decided improvement after May 29. No serum-treatment.

or local. Sometimes convulsions occur from time to time in the chronic cases after most other symptoms have disappeared. Grinding of the teeth is common and tremor may be observed.

Pain is a very common symptom, beginning early and continuing throughout the acute portion of the attack. It is situated chiefly in the head, but may involve also the back, abdomen and the limbs, especially the lower. It is subject to sudden exacerbations, particularly at night, and is often so distressing that it occasions loud outcries (the "hydrencephalic cry"). It is especially marked on any forcible movement of the body. *General hyperesthesia* is very constant, the patient being greatly disturbed and often crying out on hearing a loud noise, being exposed to bright light, or on the mere touching of the skin.

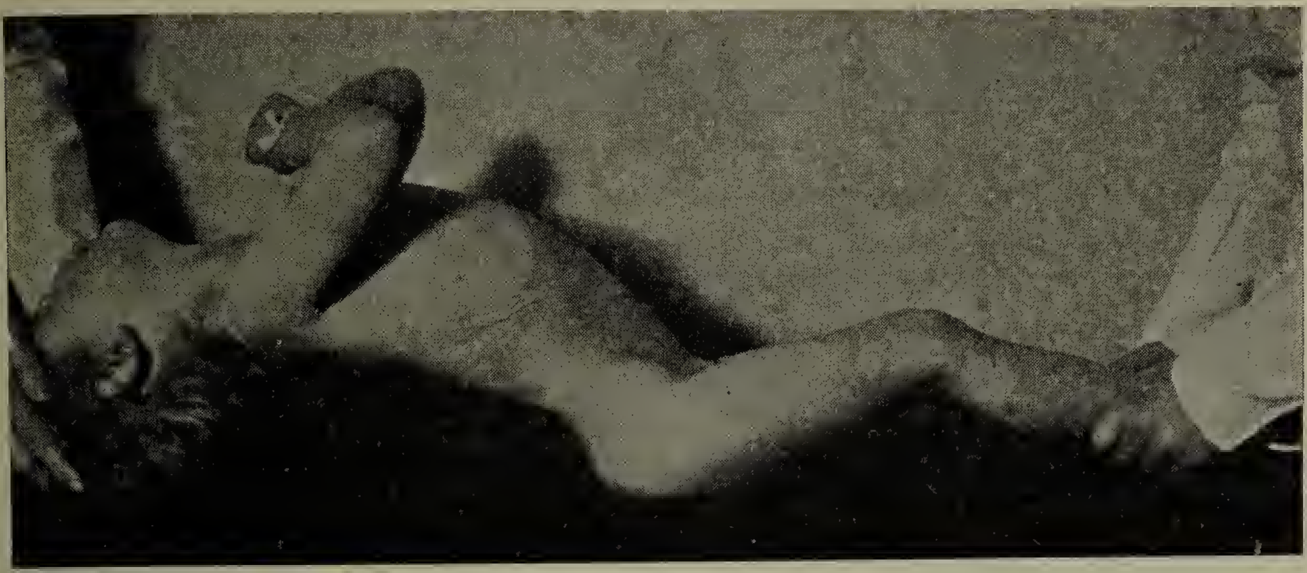

Fig. 111.—Opisthotonus in Cerebrospinal Fever.

Boy of 3 years in the Children's Hospital of Philadelphia. See history with Fig. 117.

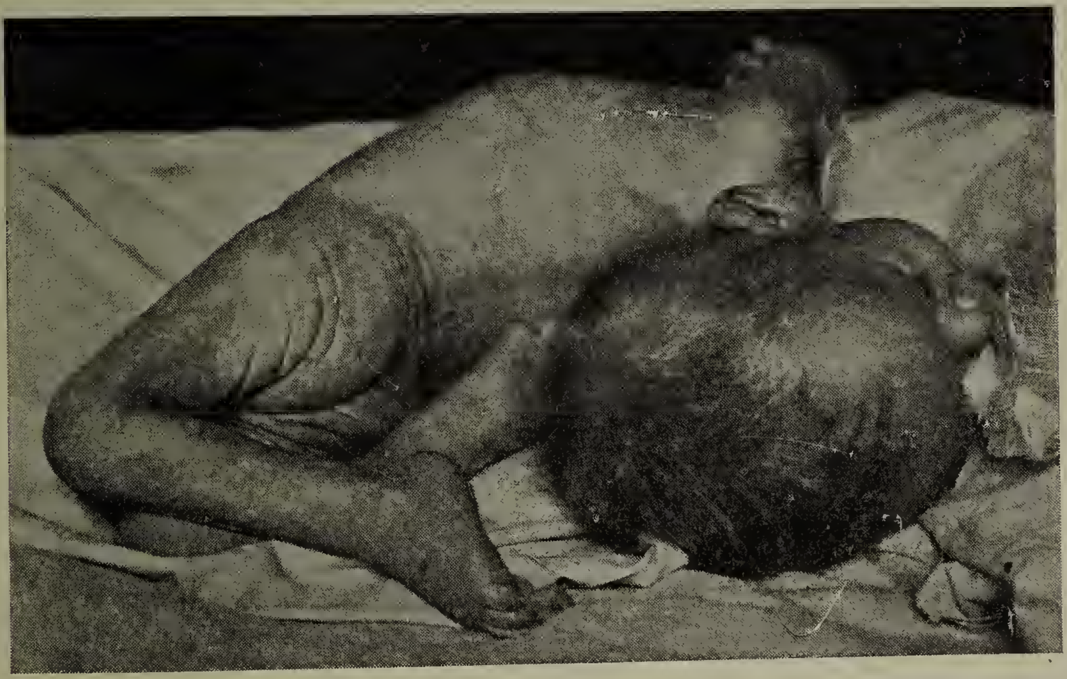

Fig. 112.—Opisthotonus in the Subacute Stage of Cerebrospinal Fever.

Boy of 1 year, ill for 4 or 5 weeks with irregular temperature, emaciation, leucocytes 33,500, spinal fluid under great pressure and almost clear, 300 cells to the c.mm., 80 per cent. polymorphonuclears, meningococci, death.

Muscular rigidity is almost always seen, chiefly in the form of stiffness of the neck and some degree of retraction of the head (Fig. 111). If the head is lifted forcibly from the pillow the trunk follows it without any bending of the neck taking place. It can, however, be turned from side to side without difficulty. In severe, long-continued cases the occiput may even press against the back beneath the scapulæ (Fig. 112). It not infrequently happens, however, that the stiffness of the neck is intermittent, not being discovered at one examination although found at another. One of the earliest signs present, even when rigidity is slight, may be brought out by flexing the head forward toward the chest; whereupon there will occur involuntary flexion of the legs on the thighs and of the thighs on the trunk (Brudzinski's sign). Rigidity with anterior curving of the spine is common, and the children often lie on the side with the arms flexed stiffly and drawn over the chest, the legs flexed, and the thighs drawn to the abdomen;—the so-called "gun-hammer" position. The muscles of the face may be tense and the *risus sardonicus* present (Fig. 113). Trismus may occur, the abdomen is often scaphoid, and Kernig's sign—viz., the inability to extend the leg by passive movement when the thigh is at right angles with the trunk—is generally observed, as in all forms of meningitis. The *tendon-reflexes* are uncharacteristic, being either normal, increased or absent. Other physical findings indicative of cerebrospinal irritation are described elsewhere in (Vol. II, p. 610).

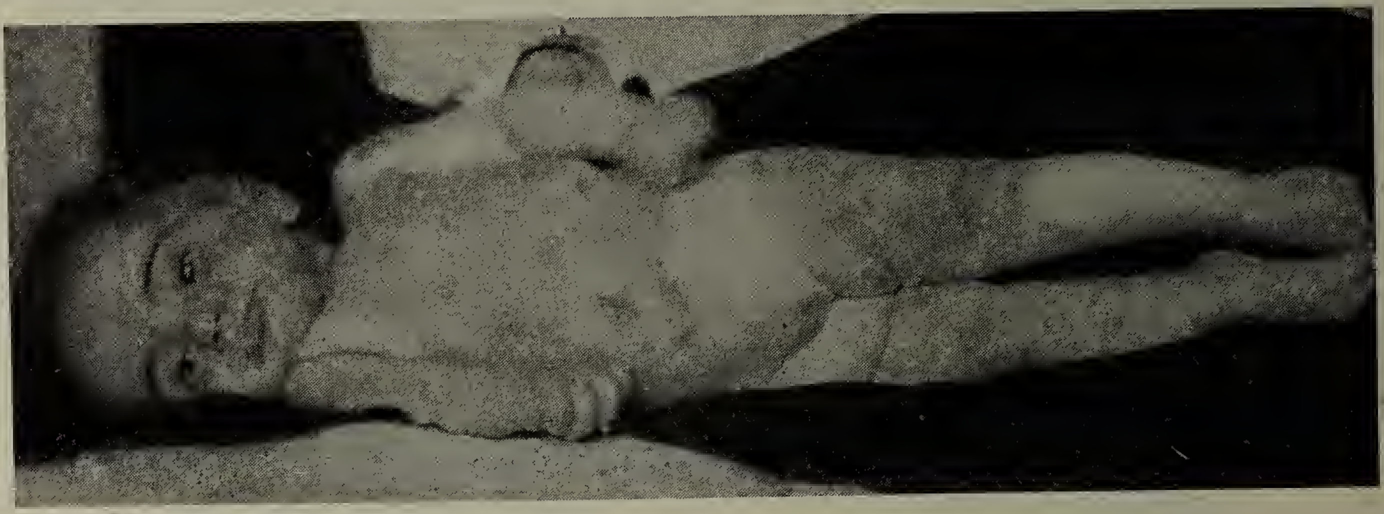

Fig. 113.—Risus Sardonicus in Cerebrospinal Fever.

Infant of 7 months in the Children's Hospital of Philadelphia. Died after 45 days of illness. Photograph taken on 21st day. Shows the facies as well as the spastic condition of the extremities.

The *mental symptoms* are variable. Great restlessness is common early in the attack and may persist. The mind may be clear much of the time, but delirium is frequent and may be intense or even maniacal, the child tossing wildly about the bed, or even jumping out of it. In other cases it is merely of the wandering type, and either constant or intermittent; or it may be followed or replaced even at the onset by a more or less apathetic or even stuporous condition. The degree of delirium does not appear always to bear any definite relationship to the other symptoms or to the gravity of the attack in general. In severe cases coma is liable finally to supervene, or it can even be one of the earliest symptoms. As in the case of delirium, it may vary greatly from day to day, sometimes rapidly disappearing or reappearing; or coma and delirium may alternate. The expression of the face in the acute condition is that of excitement and irritability, except in the mild cases.

Among *digestive disturbances* vomiting is an early symptom, present in the majority of cases. It may be frequent enough to debilitate the patient greatly. Generally it subsides as the disease advances, but to this there are many exceptions. It is cerebral in origin, and may or may not be attended by coating of the tongue. Appetite is diminished. Constipation is generally observed.

The *temperature* is irregular and entirely uncharacteristic. It generally rises rapidly, and it then remains high or diminishes; but sudden remissions or intermissions as well as sudden rises to 105°F. (40.6°C.) or over are liable to occur. A temperature of 101 to 103°F. (38.3 to 39.4°C.) is an average one. Some patients never exhibit much fever. In fatal cases unusual hyperpyrexia is sometimes seen (Fig. 117). As a rule, however, there is little connection in acute attacks between the height of the fever and the severity of the disease. Irregularity is especially marked as convalescence goes on, or as the disease passes into a chronic state.

The *pulse* bears little relationship to the temperature. It is usually more rapid than normal, especially if there is great general debility. It is subject to sudden changes in rate, and may be slow or irregular if intracranial pressure is increasing. The arterial tension is low.

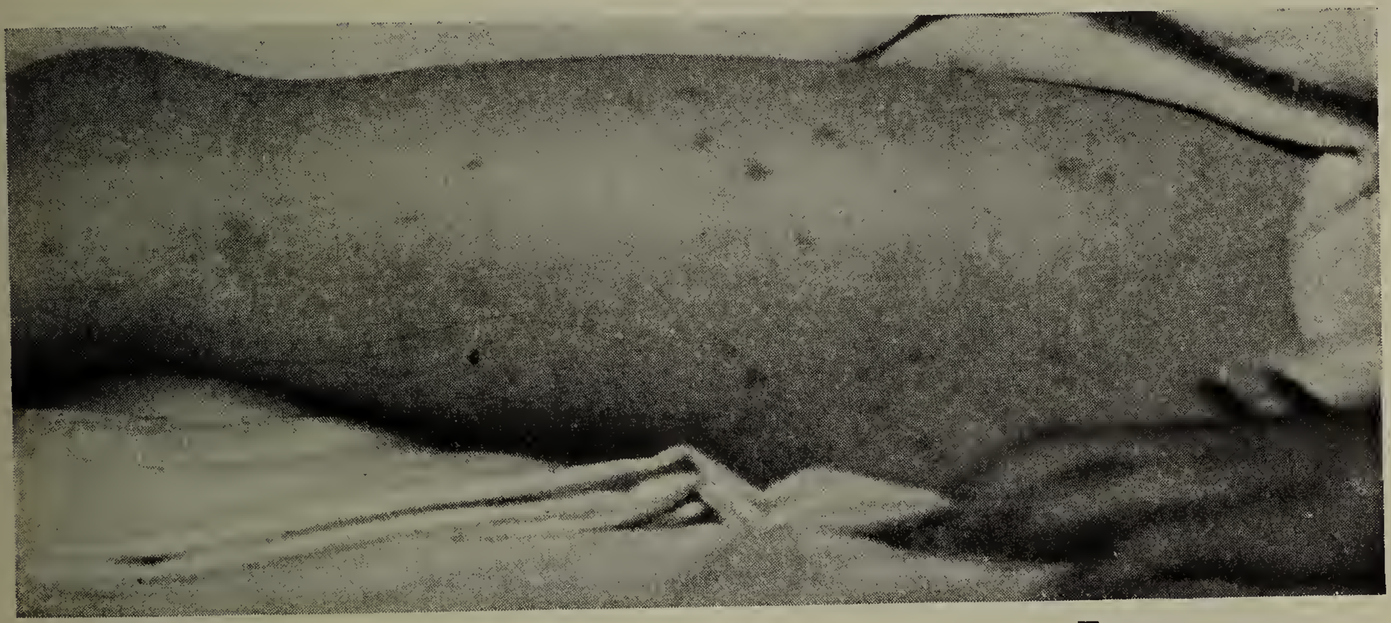

Fig. 114.—Purpuric Eruption in Cerebrospinal Fever.

Boy of 6½ years, a patient in the Children's Ward of the University Hospital, Philadelphia. Rash appeared on the 4th day of the disease, abundant on all extremities. Case a severe one; improved temporarily, but terminated fatally.

The *respiration* is not characteristically affected. Its rate may be decidedly increased by the presence of pain. In advancing cases it may become sighing, irregular, or even approaching the Cheyne-Stokes type.

The *cutaneous symptoms* are interesting; the most frequent generally being herpes, which is present in a large proportion of cases. It is usually situated on the face, but sometimes elsewhere. As a rule an early symptom, it may not develop until later, or it may come out in crops. A petechial or larger purpuric eruption is common, its frequency varying greatly with the epidemic (Fig. 114). In the New York outbreak of 1905 it was recorded in 19 per cent. of the cases; while in the earlier epidemics in the United States it was so common that it gave rise to the title of "Spotted Fever." In the epidemic in Philadelphia in 1917 and 1918 I observed it in comparatively few cases and since then have not seen it frequently. It may appear early or later, and seems to bear little relation to the severity of the attack. In some cases larger cutaneous hemorrhages occur. A well-marked *tache cérébrale* is a common symptom, as in all forms of meningitis (see Vol. II, p. 610, Fig. 354), and often there may be noted an irregular flushing of the trunk when exposed, or of the face, the evidence of the vasomotor disturbance present. In any case at all long-continued *emaciation* is very decided and in chronic cases is liable to become extreme.

The *blood* always presents an early and very marked leucocytosis, especially of the polymorphonuclear cells, equalling sometimes as much as 40,000 or more to the c.mm. The eosinophiles disappear. The *urine* is normal or exhibits a febrile albuminuria. Small amounts of sugar are occasionally observed.

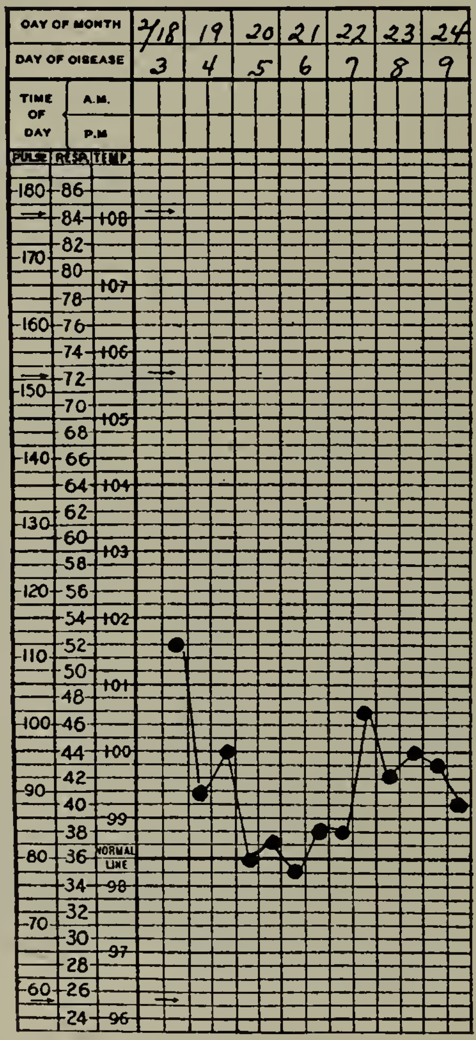

FIG. 115.

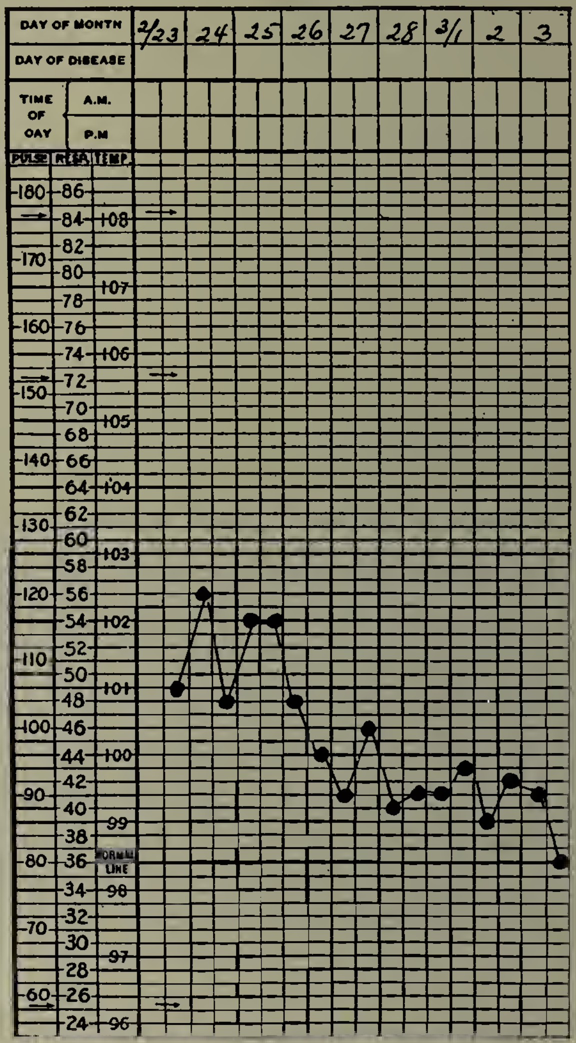

FIG. 116.

FIG. 115.—CEREBROSPINAL FEVER, ABORTIVE FORM.

Milton L., 2 years old. Onset with repeated convulsions, prolonged unconsciousness, delirium, fever. 2d day showed stuporous condition and fever, the child being apparently very ill. Rapid improvement followed, the mind becoming quite clear by the 4th day, and convalescence being entirely established by the 8th or 9th day. Meningococci found in the spinal fluid.

FIG. 116.—CEREBROSPINAL FEVER, MILD FORM.

Mary K., aged 5 years. One of three children of the family ill with the disease. Exact date of onset uncertain, but had been slightly ill for not over a week. Feb. 23, sleeps much of time, mind seems entirely clear, expression placid, apparently no pain, no irritability, no hyperesthesia, abdominal tache marked, neck slightly stiff, head slightly retracted but only if the child lies on her side, no other rigidity, no herpes or petechiæ Feb. 26, improving, much brighter; Feb. 28, greatly better, stiffness of neck gone; Mar. 10, out of bed, entirely well. No serum used. Children's Hospital of Philadelphia.

The *eyes* may show injection or decided inflammation of the conjunctiva. The pupils are often variable at first, and dilated later or react slowly. Alternate dilatation and contraction of the pupils regardless of light or accomodation (hippus) may be present. Strabismus is common. Gingold[1] describes a form of this which is seen in basilar meningitis. It develops only when the head is flexed on the chest, and disappears when the head is relaxed. It may be accompanied by a retraction of the upper eyelids. Nystagmus also may occur. Sometimes the movements of the eyeball are slower than in the ordinary nystagmus and only one eye may be affected or, if both, the movements may not be associated (Pieraccini).[1]

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[1] Arch. of Pediat., 1920, XXXVII, 19.

The *cerebrospinal fluid* as obtained by lumbar puncture is increased in amount; turbid in acute cases and often quite purulent; and exhibits an increase of globulin. It contains numerous polymorphonuclear leucocytes together with meningococci in varying numbers, free, or principally within the cells. The germs frequently disappear early in the attack, and in more chronic cases the fluid may be almost or quite clear, and without any preponderance of polymorphonuclear cells, and meningococci can usually no longer be discovered. Not infrequently a fluid, purulent at first, rapidly becomes nearly or quite clear, to exhibit a return of the purulent condition later, if recovery is not prompt. In some cases the fluid may be clear at first and become purulent later. Sometimes, too, no germs may be found early, but develop subsequently.

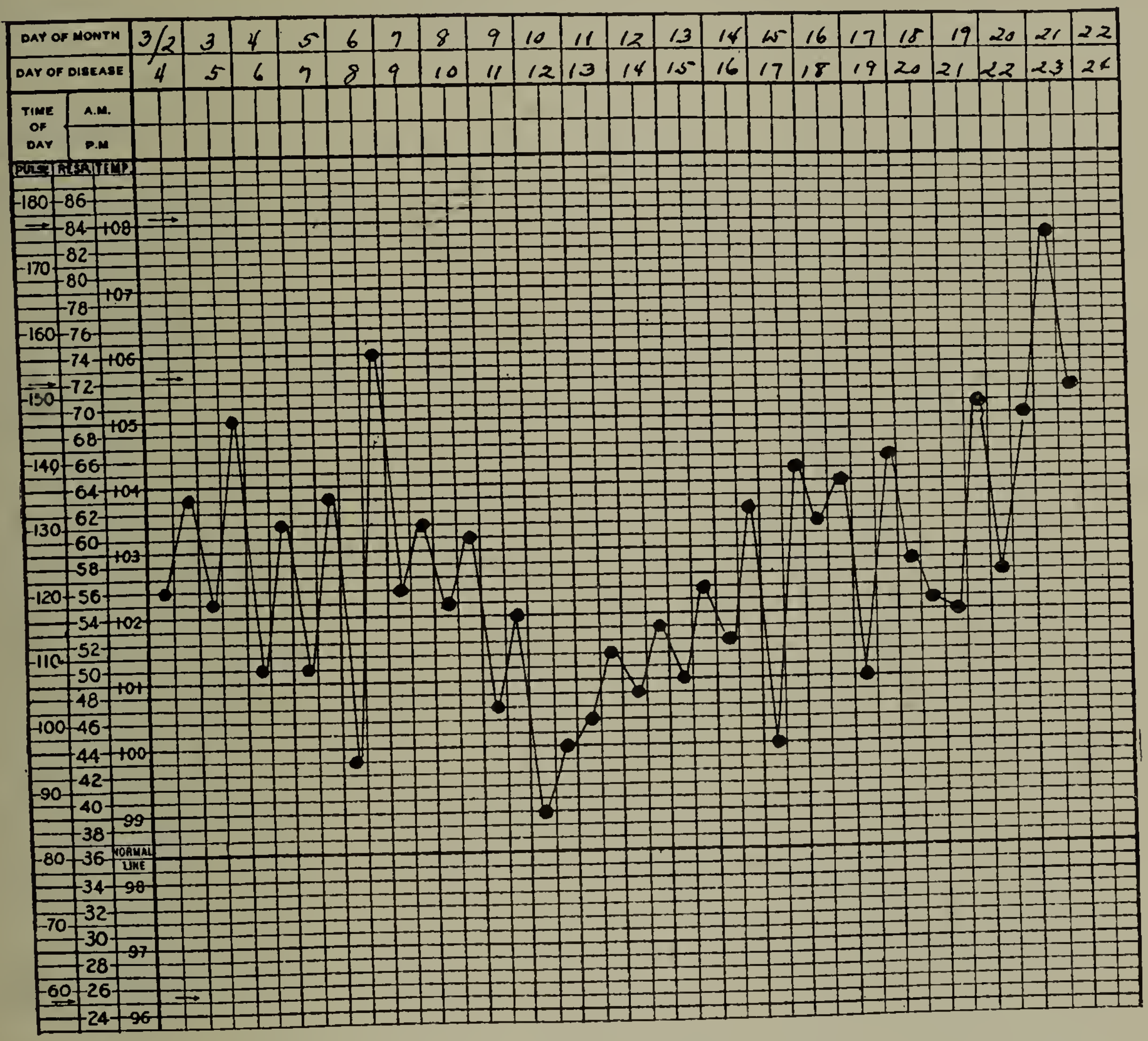

FIG. 117.—CEREBROSPINAL FEVER, SEVERE FORM, LONG COURSE.

Howard S., 3 years old. Admitted to the Children's Hospital Mar. 2, 1917. Onset with fever, vomiting and headache on Feb. 27. On admission rigidity of neck, irritable when disturbed, increased knee-jerks, Kernig's sign, hyperesthesia, cloudy fluid containing meningococci. While in hospital had continued retraction of head, widespread petechiæ, stupor, hyperesthesia, sometimes very restless. Lumbar puncture and injection of serum done repeatedly, but seemed to produce severe reaction, with prostration. Restlessness and irritability increased after serum injected. Examinations of blood at different times showed leucocytes varied from 12,000 to 23,000; Mar. 11, appeared to be improving, but relapsed. Washing out of spinal canal with salt solution tried on the last few days of life, but child grew worse. Death on Mar. 21.

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[1] Rivista critica di clin. med., 1922, XXIII, 181. Ref. Jour. Amer. Med. Assoc., 1922, LXXIX, 1277.

Variations from the Ordinary Form.—Even among cases of the ordinary type there is the greatest variation in the symptoms. Some begin with very severe manifestations which soon ameliorate. The patients, however, may fail to convalesce at once, but pass perhaps through weeks of illness of much diminished severity. Other cases begin mildly but soon grow more severe. Others reach a fatal ending through some one of the numerous complications which are prone to develop. At times the infection, beginning as a meningococcus-septicemia, may remain as such and only after a considerable time localize in the meninges (Bloedorn).[1] The variations are often so decided that a number of special types are described. Among these may be mentioned (1) the Abortive form; (2) the Mild form; (3) the Severe form; (4) the Malignant form; (5) the Chronic form; and (6) the Intermittent form.

1. **Abortive Form.**—In this variety the disease begins abruptly and severely, but in 2 or 3 days the threatening symptoms disappear and the patient rapidly recovers (Fig. 115).

2. **Mild Form.**—In cases of this sort the symptoms are mild from the outset, or soon become so after a severe onset (Fig. 116). There may be only slight headache and nausea, occasional vomiting, slight stiffness and pain in the neck, and little fever. The mind is clear, or nearly so. The patient may not even be confined to bed. Sometimes the symptoms are so trivial and uncharacteristic throughout that diagnosis would be impossible if the case were an isolated one. In 1 case of 3 occurring simultaneously in a family under my care the only symptoms present were slight fever and cerebral tache and a slight rigidity of the neck; all of which would have passed unnoticed had the case occurred alone. In another instance, also 1 of a family group of 3, the patient was suffering from a mild attack of pneumonia; only the tache and the very moderate rigidity of the neck indicating that this disease was a complication of a very mild cerebrospinal fever.

3. **Severe Form** (Fig. 117).—In this all or many of the symptoms are intensified. The type does not differ materially from that described as the ordinary variety, except for a greater severity of the manifestations. The course may be short or prolonged.

4. **Malignant or Fulminating Form** (Figs. 118 and 119).—This variety is characterized by the extremely sudden onset, the intensity of the symptoms, the tendency to severe collapse, and the shortness of the course. The child may be stricken suddenly while at play. It may suffer from repeated convulsions and die in less than 24 hours, the diagnosis being impossible unless the case be one of a family group or a lumbar puncture be made. Sometimes coma and collapse are the earliest or the only symptoms, or there may be most violent repeated vomiting or intense headache. Wide-spread cutaneous hemorrhages may develop and hemorrhage from the mucous membranes take place.

5. **Chronic Form.**—This might be called one of the terminations of the disease. The symptoms at first do not differ from those of the ordinary type. Instead, however, of disappearing gradually, they continue in a modified form. Fever may be absent for a considerable time and all the symptoms may ameliorate. Then with the recurrence of elevated temperature the stiffness of the neck increases, vomiting returns, delirium or stupor reappears, and convulsions may occur. This condition may last a variable time, to be followed apparently by the beginning of certain convalescence, when a second recurrence takes

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[1] *Loc. cit.*

place. In this way the disease may be protracted for months. Not all the symptoms mentioned need be present. In fact, the symptomatology is likely to be very variable. In some cases the remissions in temperature are unattended by improvement in other symptoms. Emaciation is liable to become extreme. I have the record of 1 case, finally fatal, lasting 251 days. During much of this period the child seemed at intervals almost entirely well. The sections of the case-history appended (Figs. 120 to 124) give a synopsis of the symptoms as occurring at different times in the course of the disease. There is no disorder more discouraging to the family and the physician than this chronic form of cerebrospinal fever. (See Sequels, below.) There is also a condition observed in infancy known as chronic basilar meningitis which is probably a variety of chronic cerebrospinal fever. (See also Chronic Meningitis, Vol. II, p. 625.)

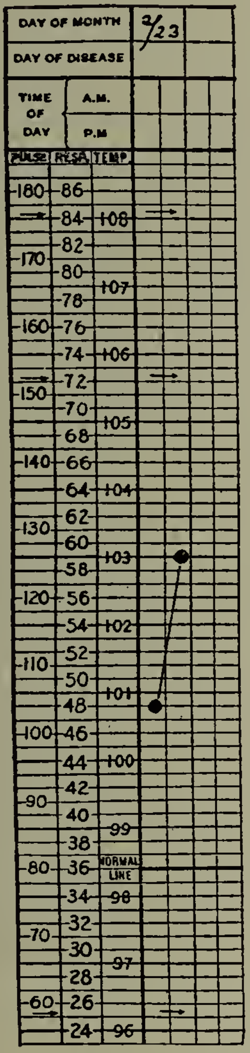

Fig. 118.

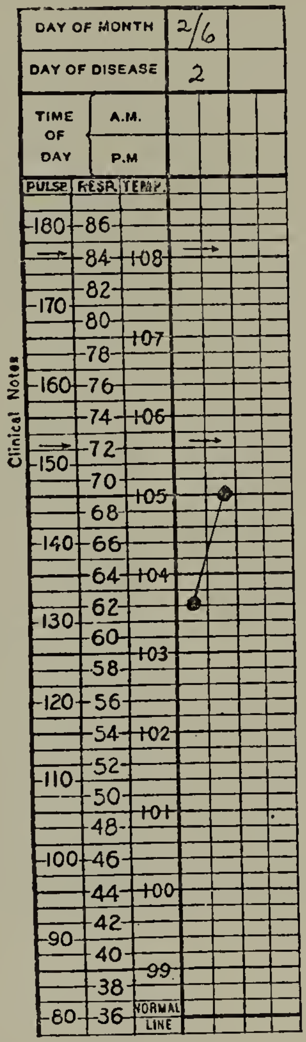

Fig. 119.

Fig. 118.—Cerebrospinal Fever, Malignant Form.

Charles K., 2 years old. One of three in family with the disease. Taken ill suddenly on Feb. 22 with convulsions, and these continued without interruption. Death on the afternoon of Feb. 23 without having regained consciousness. Children's Hospital, Philadelphia.

Fig. 119.—Cerebrospinal Fever, Malignant Form.

Ethyl C., 9 months old. Feb. 5, taken ill suddenly in the afternoon with vomiting lasting an hour. Then became drowsy, weak, and with rapid respiration; Feb. 6, stuporous, rolling of eyes, rigidity of arms and legs, cyanosis, leucocytosis 15,200. Meningococci in the spinal fluid. Death in the afternoon.

6. Intermittent Form (Fig. 125).—This is in reality one of the varieties of the chronic type, characterized by a temperature-curve which strongly suggests malarial fever. There are not the longer and irregular periods of freedom from fever characteristic of the variety just described. Improvement in symptoms may or may not attend the drops in temperature.

Complications and Sequels.—These are numerous and often of a very serious character, being even the direct cause of death or of permanent disability. The most important are those affecting the nervous system and the special senses. The *eyes* may exhibit neuritis of the optic nerve, due to involvement by the exudate at the base of the brain; or the purulent process may extend along the pia-arachnoid of the nerve and produce a purulent choroido-iritis. In other cases a neuritis of the 5th nerve is followed by purulent conjunctivitis or keratitis. These lesions may develop early or later in the attack. Complete or partial blindness may result. The *ears* are very often involved. Westenhöffer[1] believes that otitis media is present in all cases in children. If purulent it may be the cause of loss of hearing. Absolute deafness from inflammation of the labyrinth, resulting from extension of the process along

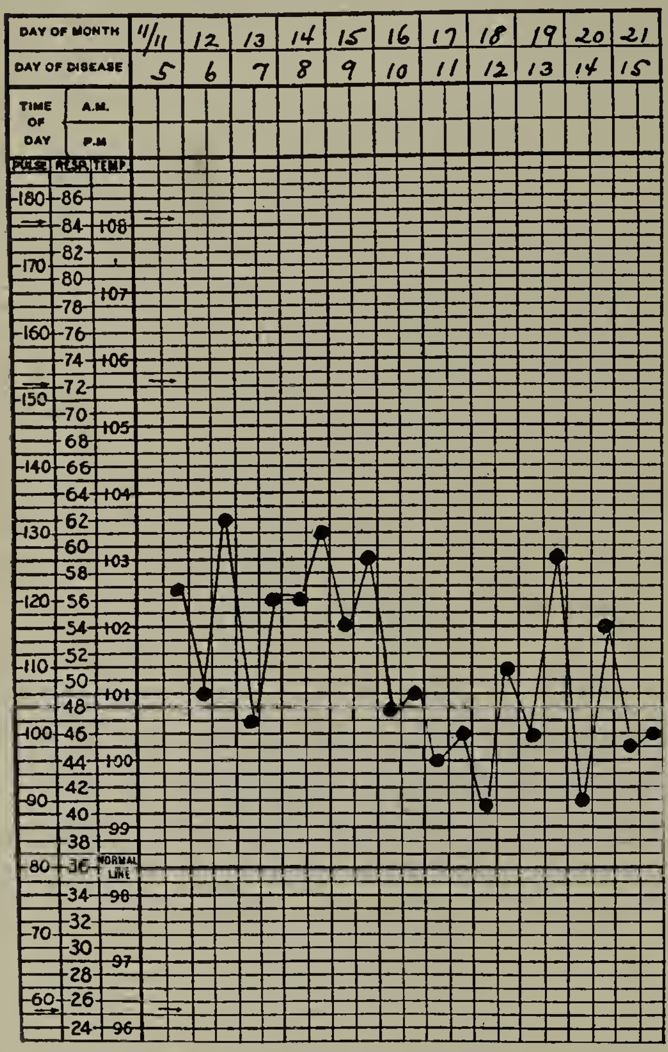

Fig. 120.

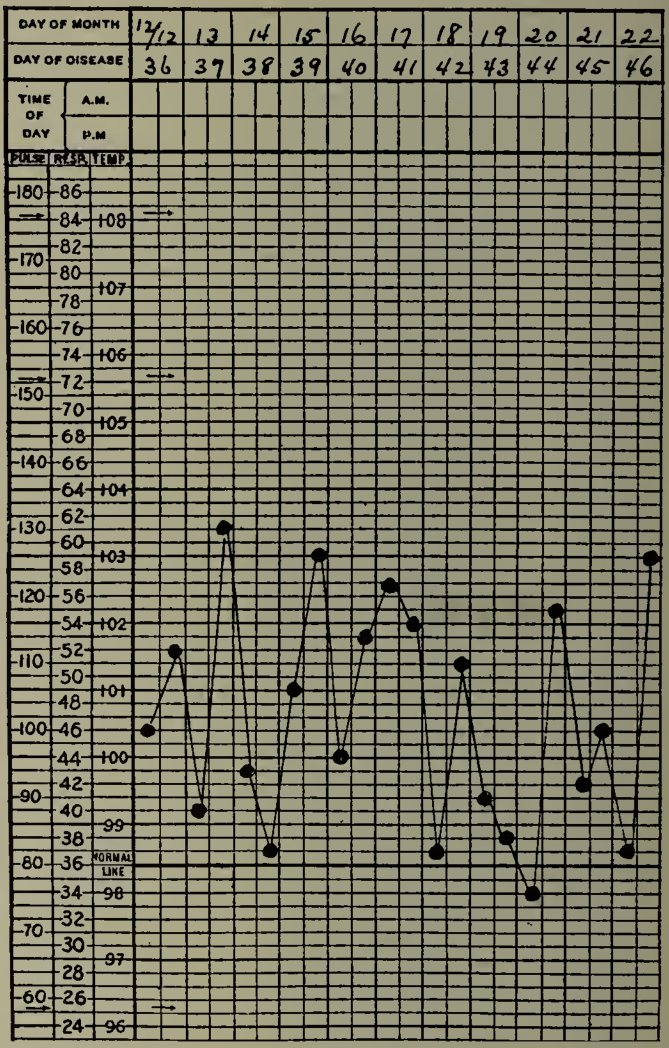

Fig. 121.

Fig. 120.—Cerebrospinal Fever, Chronic Form.

Willie R., 6½ years old. Children's Hospital. Chart of 5th to 15th day inclusive.

I. *Acute Stage.*—Nov. 11, 1904. Irritability, delirium, pain in neck, back and limbs, strabismus, rigidity, retraction of head, hyperesthesia, leucocytosis 19,520. Lumbar puncture showed thick pus with meningococci; Nov. 16, gradual improvement; Nov. 18, rational to a considerable degree; Nov. 21, petechiæ; Nov. 26, improving decidedly, slight hyperesthesia and some rigidity and pain remaining.

Fig. 121.—Cerebrospinal Fever, Chronic Form.

Willie R. (*Continued*). Chart of 36th to 46th day inclusive.

II. *Partially Intermittent Temperature.*—General condition in December much improved. Still strabismus, slight stiffness of neck, and frequent pain in the limbs and back. Began to walk by the end of December.

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[1] Klin. Jahrb., 1905–6, XV, 657.

the auditory nerve, is a not infrequent sequel. Moos[1] found 38 deaf-mutes in 64 recovered cases of cerebrospinal fever. Probably the majority of cases of deaf-mutism in institutions owe their origin to this disease.

Disordered mental states may occur as sequels, among them being aphasia and mental impairment. Among 539 cases of mental defect in Norway, reported by Looft[2] 3.7 per cent. resulted from cerebrospinal fever. *Headache* is sometimes very persistent. *Hydrocephalus* is a serious and very frequent sequel. Undoubtedly many of the chronic cases of cerebrospinal fever are due to it. The group of symptoms characterizing it, as outlined by Ziemssen[3] and others, consists of vomiting; severe pain in the head, neck and limbs; rigidity; great emaciation; increasing apathy; convulsions, and finally coma. This condition may alternate with periods of decided improvement in health. Hydrocephalus of an acute form may occur even early in the disease. This would account for some of the symptoms observed at that period. *Paralysis* may involve the eyes or the face; less often the limbs, in the latter case being either hemiplegic or paraplegic in type. It may be temporary or permanent. It generally does not develop until well on in the attack.

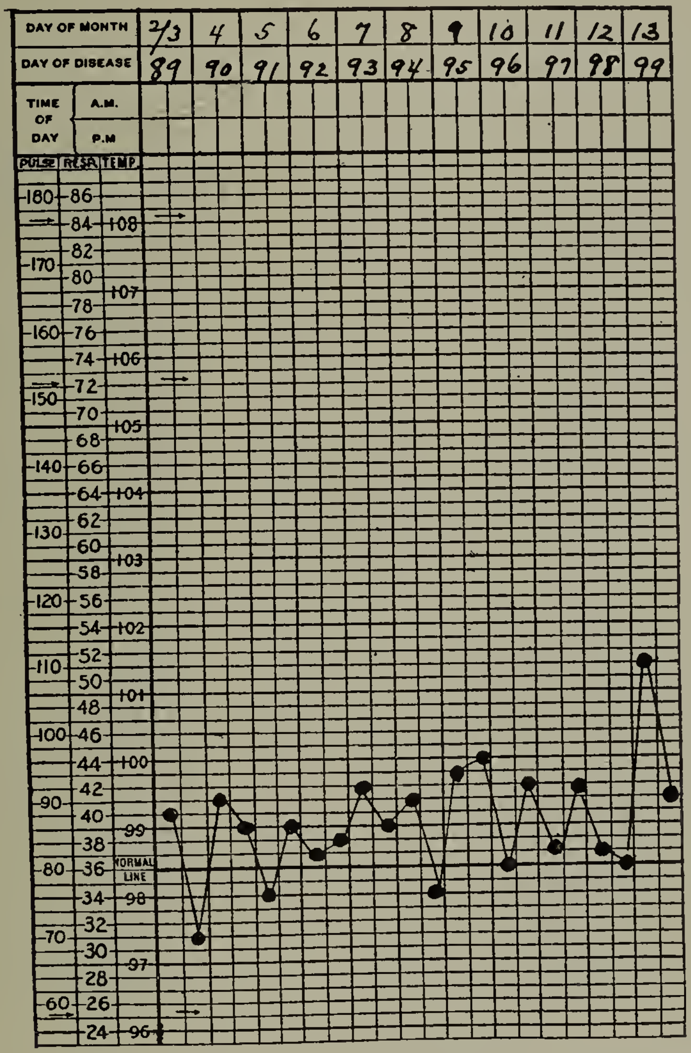

FIG. 122.—CEREBROSPINAL FEVER, CHRONIC FORM.

Willie R. (*Continued*). Chart of 89th to 99th day inclusive.

III. *Apparent Convalescence, Followed by Some Return of Symptoms.*—During last of December and January child constantly improving. Out of bed and walking about the room. Strabismus and slight rigidity of neck remained. Last of January frequent vomiting began. Occasionally slight convulsions. Child looked less well. Leucocytes increased from 14,350 on Feb. 1 to 17,050 on Feb. 13. Practically no fever.

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[1] Die Taubstummheit u. ihre Abhängigkeit von Cerebr.-sp. Mening., 1883. Ref., Councilman, Mallory and Wright, *loc. cit.*

[2] Nord. med. Ark., 1901, II, No. 4.

[3] Hand. spec. Path. u. Therap., Bd. II, Th. II, 683.

Inflammation of the pharynx, nasopharynx, and tonsils is a frequent complication occurring early in the attack, and sometimes antedating other symptoms. *Pneumonia* is frequently combined with meningitis. When it is the primary disease it is pneumococcic in origin and the meningitis is probably of the same nature and not to be classed as cerebrospinal fever. Pneumonia, however, is frequently seen as a complication secondary to cerebrospinal fever, and probably is produced by the meningococcus. *Nephritis* is a serious complication occasionally seen adding to the gravity of the case. Although not reported as so frequent by most writers, Steiner and Ingraham[1] found evidence of its presence in 28 out of 145 cases of the disease. *Arthritis* affecting a number of joints is a not uncommon complication or sequel. The fluid may be serous or purulent in nature and contains the meningococcus. In other cases the joint is red and swollen without evidence of effusion.

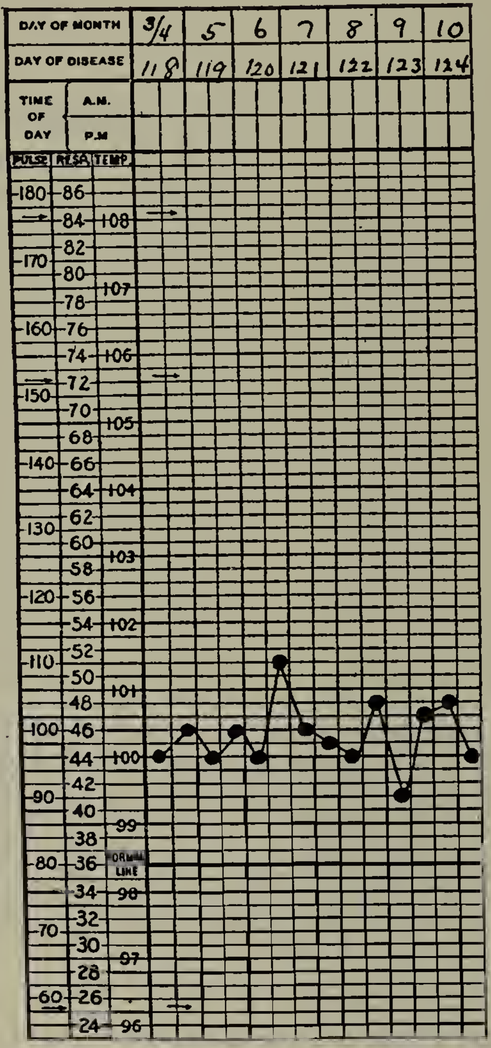

FIG. 123.

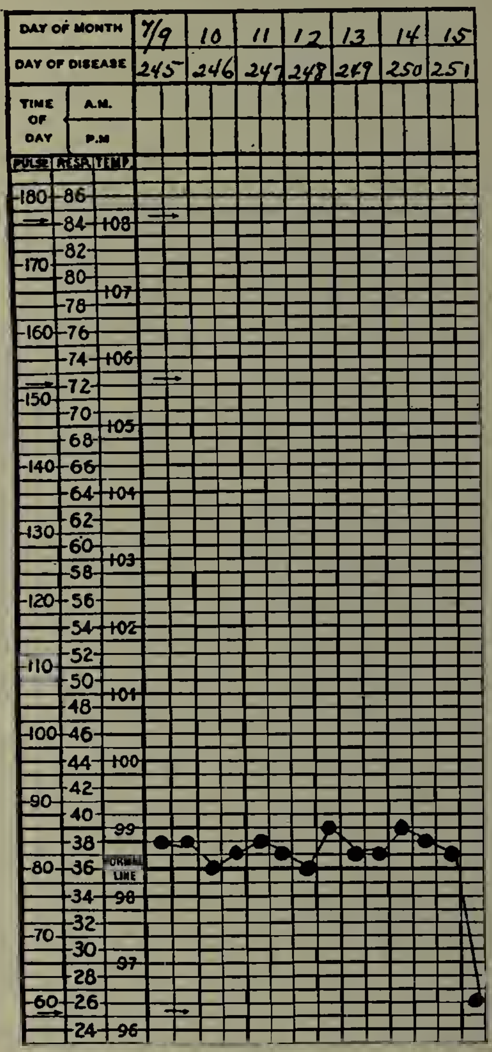

FIG. 124.

FIG. 123—CEREBROSPINAL FEVER, CHRONIC FORM.

Willie R. (*Continued*). Chart of 118th to 124th day inclusive.

IV. *Continuance of Subacute Symptoms.*—Mar. 1, child again confined to bed. Frequent distressing pain in head, neck, and limbs, emaciation decided, losing power in limbs, irritable and capricious but mind clear, slight febrile reaction. Sedatives constantly required.

FIG. 124.—CEREBROSPINAL FEVER, CHRONIC FORM.

Willie R. (*Continued*). Chart of 245th to 251st day inclusive.

V. *Gradual Increase of Hydrocephalic Symptoms.*—Slow but constant loss of strength continued, pain very frequent and distressing, emaciation extreme. In July, failed rapidly, rigid all over, head much retracted, periods of unconsciousness and finally persistent coma. Died July 15, on the 251st day of the disease.

Cerebrospinal fever may occasionally occur simultaneously with or as a sequel to *other acute infectious diseases*, among these being typhoid fever, scarlet fever, measles and diphtheria. Collins[1] reports an instance of the combination of the disease with malaria.

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[1] Amer. Jour. Med. Sci., 1908, CXXXV, 351.

Among other complications sometimes seen are pleurisy, pericarditis, endocarditis, peritonitis, parotitis, enteritis, subcutaneous abscesses, urticaria, pemphigus, and erythema.

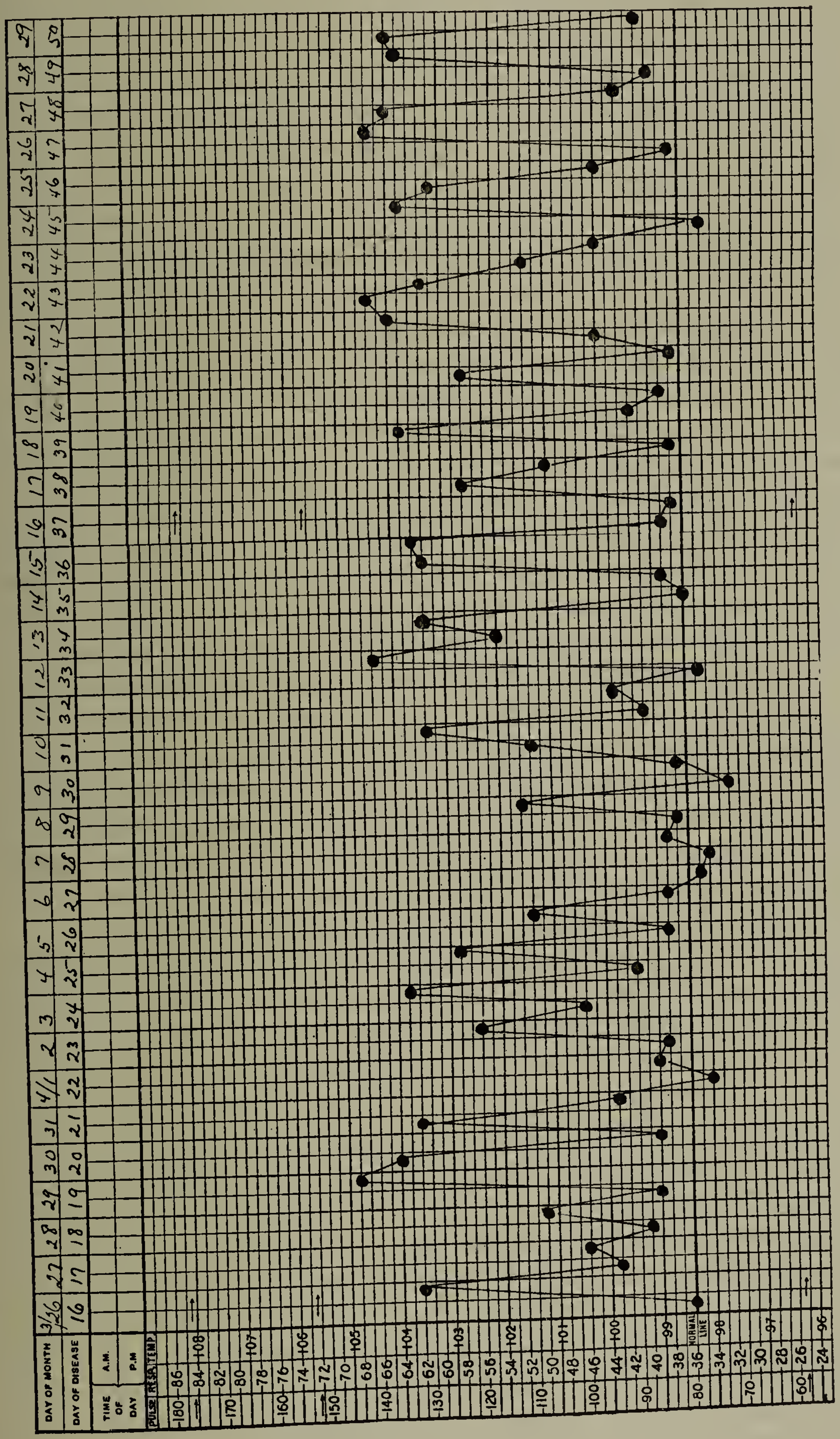

FIG. 125.—CEREBROSPINAL FEVER, INTERMITTENT FORM.

Regina J., aged 4 years. Portion of chart illustrating approach to the intermittent type of the disease. The rises of temperature were generally attended by renewal of projectile vomiting, and often by pain in the neck and elsewhere. Recovery.

Relapse.—In a disease with such an uncertain course and with such a natural tendency to recrudescence after convalescence is apparently beginning, it is difficult to reach any conclusion regarding the frequency of relapse. Indeed, the disposition to it may be called a part of the disease. It was observed by Netter[1] from 1 to 3 months after apparent recovery in 1.14 per cent. of 350 cases.

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[1] Bost. Med. and Surg. Jour., 1911, CLXV, 610.

Recurrence.—Little is known about recurrence. As a rule one attack appears to protect from subsequent ones, although instances to the contrary are reported.

Prognosis.—The disease is a serious one, the mortality varying according to Hirsch[2] from 20 per cent. to 75 per cent., and being oftener nearer the latter figure than the former. The actual number of cases dying during epidemics is often large, over 2500 having succumbed in New York City in the outbreak of 1904–1905 (Billings).[3] The mortality is especially high in early life. Friis[4] found it in the 1st year to be 77.7 per cent., from 1 to 5 years, 48.7 per cent., from 5 to 10 years 51.6 per cent., and from 10 to 15 years 21.4 per cent. Of 779 fatal cases collected by Hirsch[5] 208 were under 1 year of age, 337 from 1 to 5 years, 151 from 5 to 10 years, 41 from 10 to 15 years, 16 from 15 to 20 years, and 26 over 20 years of age. It is impossible to predict what the outcome will be in any given case, and the prognosis must always be very guarded. The fulminant cases are nearly always fatal. Sudden, severe onset; very exhausting and continued vomiting; persistent, unusually high temperature; the repeated occurrence of convulsions; rapid pulse and respiration, and the early development of coma or its long persistence are unfavorable symptoms. A drop in the number of neutrophiles in the blood and an increase of the lymphocytes is a favorable indication. The existence of no germs, or but a small number, in the spinal fluid is claimed also to be of favorable prognostic significance. Death takes place oftenest within the 1st week, and the outlook is consequently brighter when the 2d week is well under way and the symptoms seem to be ameliorating. To this, however, there are numberless exceptions, since a long-continued chronic form frequently develops which ends fatally oftener than in recovery. There is always, too, the very great danger of permanent sequels remaining. Epidemic influence upon the mortality is marked, the number of deaths being much greater in some years than in others. More fatal cases occur at the beginning of an epidemic than later.

What has been said above regarding the mortality-rate applies only to the condition before the introduction of serum-treatment. The lessened death-rate which followed will be discussed under Treatment.

Diagnosis.—This rests especially on the sudden, severe onset, vomiting, delirium, restlessness, intense headache, stiffness and pain in the neck, retraction of the head, pain in and rigidity of the muscles of the back and limbs, leucocytosis, herpes, the rapid development of the symptoms early in the attack, and the great tendency to variation in its course. All these are indications of the presence of some variety of meningitis, but in no other is there such a striking complex of symptoms. The spinal manifestations are generally more marked than in any other form. In the malignant cases the diagnosis may be impossible. This is true also of the very mild cases without suggestive symptoms. The existence of an epidemic is often of great service in reaching a conclusion, but the individual character of the epidemic is to be borne in mind. In that in Philadelphia in 1917 the diagnosis, although easy in many cases, was in others very confusing. In some instances the early symptoms were so entirely uncharacteristic that only the fact that the disease was prevailing led to the performing of lumbar puncture, and the discovery, based only on this, that meningitis existed. Percussion of the skull, recommended by Macewen, for the recognition of fluid in the ventricles is strongly urged by Koplik,[1] a slightly tympanitic note in the lateral regions indicating the presence of fluid. We have found this sign of little aid.

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[1] Bull. de la soc. méd. des hôp., 1918, XLII, 527.

[2] Die Meningitis cerebrospinalis epidemica, 1866, 33.

[3] Jour. Amer. Med. Assoc., 1906, XLVI, 1670.

[4] Ugeskrift for Laeger, 1891, Ref. Netter, 20th Cent. Pract. of Med., XVI.

[5] *Loc. cit.*, 121.

The character of the spinal fluid is most important diagnostically. The early purulent nature distinguishes it from tuberculous meningitis, in which the fluid is clear or only slightly opalescent. There is the contrast, too, between the numerous polymorphonuclear cells of cerebrospinal fever and the lymphocytic cells usually predominating in cases of tuberculous meningitis. In the more chronic cases the fluid may be clear, but by this time the distinction from tuberculous meningitis can generally be made readily in other ways. The distinguishing of the fluid of meningococcic meningitis from that of other acute purulent forms can be done only by bacteriological study. In some cases, such as those described by Woringer[2] and others, the infection is primary in the ventricles which become walled off, and the spinal fluid obtained by lumbar puncture will then be free from meningococci.

Cerebrospinal fever is to be differentiated from several other forms of meningeal inflammation. *Tuberculous meningitis* comes on, as a rule, very slowly and insiduously with only later a development of marked cerebral symptoms and finally of coma, and is without the rapidity of alteration in the symptoms to the degree characteristic of cerebrospinal fever. These diagnostic differences apply, however, only to typical cases of the disease; in others the differentiation based on symptomatology, apart from lumbar puncture, may be impossible.

Simple acute meningitis, in the sense of being due to germs other than the meningococcus, may simulate cerebrospinal fever so closely that diagnosis cannot be made except by lumbar puncture. Spinal symptoms, however, are generally less often present and the existence of some evident cause is often discoverable, since the disease is frequently a secondary one. It is always, too, of a severe form and generally fatal; and consequently the diagnosis from milder cases of cerebrospinal fever is usually made easily. It seems very probable, as Still[3] has maintained, that the posterior basic meningitis first described by Gee and Barlow[4] as occurring in infants is, in reality, cerebrospinal fever, since the microörganisms are practically identical.

Cerebrospinal fever may be confounded with several diseases which present no lesions of the meninges. Prominent among these is *typhoid fever* of the meningitic type. As a rule the passage of time will make the diagnosis easy, although there are certainly exceptions to this. The meningitic symptoms generally soon disappear in typhoid fever and other characteristics become evident, especially the absence of leucocytosis and the presence of the Widal reaction. The mere presence of leucocytosis, however, aids but little, since this may occur in typhoid fever as the result of some complicating conditions. Occasionally *grippe* exhibits symptoms resembling those of cerebrospinal fever so closely that only lumbar puncture can settle the diagnosis. Yet, as a rule, cases of this disease with meningitic symptoms exhibit a shorter course and no retraction of the head. The high degree of leucocytosis in meningitis is also of diagnostic value in excluding grippe. It is to be borne in mind that a true influenzal meningitis is sometimes observed. *Pneumonia* may be ushered in with symptoms simulating meningitis and herpes is common to both. The examination of the blood does not aid, since both conditions exhibit a high leucocytosis. The course of the case will make the diagnosis clear. It is to be remembered, however, that an actual meningitis may occur in combination with pneumonia, either of a serous or less often a pneumococcic nature. In suspected cases lumbar puncture may settle the question as to the presence of meningitis and the character of any germs found. *Autointoxication* of gastrointestinal origin may begin with vomiting and meningeal symptoms and be the cause of considerable uncertainty of diagnosis for a time. As a rule, however, the nature of the case soon becomes evident.

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[1] Amer. Jour. Med. Sci., 1907, CXXXIII, 547.
[2] Arch. d. méd. des enf., 1921, XXIV, 159; 553.
[3] Jour. Path. and Bact., 1898, V, 147.
[4] St. Barth. Hosp. Rep., 1878, XIV, 23.

Treatment. Prophylaxis.—With our ignorance of the method of extension of the disease, preventive treatment seems as yet almost impossible. The isolation of affected persons is desirable in spite of the lack of evidence of direct communicability. The employment of mild disinfectant nasal sprays in the case of those known to have been exposed is also to be recommended, in view of the possibly carrying of the disease by healthy persons.

Treatment of the Attack.—Except as regards the serum-treatment, this is largely symptomatic. The patient should be kept very quiet in a darkened room, in order to combat the excessive hyperesthesia. As the disease is a depressing one, the strength should be maintained by sufficient nourishment, not necessarily liquid, and often by alcoholic and other stimulants, given freely if need be. Although vomiting may render feeding difficult and a modification of the choice of food necessary, it is to be remembered that this symptom is cerebral in origin, not dependent upon indigestion. Comatose patients should be fed by gavage. Digitalis is frequently required. Strychnine is, in my opinion, better avoided, as it seems sometimes to increase the excitability. Bromides are often useful to quiet the patient, but the best drug for this purpose and for the relief of pain is morphine given hypodermically, taking care not to precipitate or increase a tendency to coma. Inunctions of mercurial ointment have been recommended. The employment of various preparations of silver, by injection or in other ways, have been advocated from time to time, but without any positive results. I have never been able to convince myself of any actual benefit being obtained.

Blisters to the back of the neck serve only to increase the discomfort. Warm baths at 100°F. (37.8°C.) are useful to quiet nervousness. An ice-bag may be applied to the head, remembering, however, the danger of depression by this treatment in the case of children. In the later stages the administration of iodides is recommended to favor absorption of meningeal thickening.

The removal of the exudate by lumbar puncture often gives surprising relief if the symptoms depend upon cerebral pressure. It should be employed in all such cases. Inasmuch as the inflammation often shuts off the cranial cavity from the spinal cord, lumbar puncture frequently fails to relieve the cerebral symptoms. In such cases tapping of the ventricles was advocated by Schultz[1] and is often serviceable.

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[1] Deutsch. Arch. f. klin. Med., 1907, LXXXIX, 547

Serum-Treatment.—Kolle and Wassermann,[1] Jochmann,[2] Flexner and Jobling,[3] Dopter[4] and others have prepared a serum for use in this disease. That made by Flexner and Jobling is obtained from horses which have been repeatedly inoculated with cultures of the meningococcus. After 20 c.c. (0.68 fl. oz.) or more of the exudate have been removed by lumbar puncture the serum, previously slightly warmed, is injected through the needle, which has been left in position. It may be given through a funnel and rubber tube, allowing it to enter by gravity, or a piston-syringe may be used cautiously. The injection should always be made very slowly. The amount injected should always be less than that of the exudate removed, in order to avoid increasing the intracranial pressure. Elevation of the hips favors the flow of the serum to the diseased region. The initial dose should be 10 to 15 c.c. (0.34 to 52 fl. oz.) for infants and not over 35 c.c. (1.183 fl. oz.) for older children. The injections should be made every 24 hours, sometimes oftener, during 3 or 4 days or more, beginning as early in the course of the attack as possible. The good results are sometimes immediate and surprising. The temperature in such cases may fall several degrees, the mental symptoms are greatly ameliorated, and the number of neutrophiles in the spinal fluid is much reduced, and of the meningococci likewise. In most cases, however, even where there is reason to believe that the serum is exerting a beneficial action, there is, in my experience, no immediate decided effect upon the temperature discoverable. The treatment appears to produce also a diminished tendency to the development of sequels. When no fluid can be obtained by lumbar puncture, the serum, in the case of infants, may be injected into the lateral ventricles after withdrawing exudate from this region.

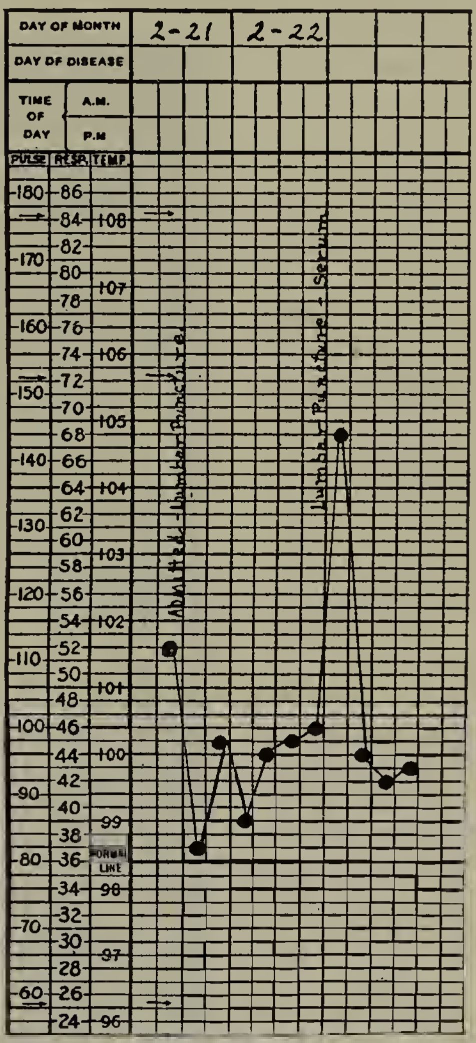

FIG. 126.—CEREBROSPINAL FEVER, WITH RISE OF TEMPERATURE AFTER INTRASPINAL INJECTION.

Frank F., 2 years old. Chart shows rise of temperature which sometimes occurs after serum-treatment. Children's Hospital.

As a rule the injection if properly made is without unfavorable results; but occasionally a considerable degree of prostration follows, and sometimes the symptoms become alarming, chiefly in the line of respiratory failure. Occasionally it has been necessary to employ artificial respiration combined with the administration of atropine hypodermically. Consequently a careful continued observation of the state of the respiration and the general condition of the patient should be maintained during the procedure and after it. It is frequently difficult to determine for how long a time the giving of injections shall be continued, and whether they are doing good or harm. If the fever and other symptoms improve, and the number of cells and of microörganisms in the spinal fluid diminishes, the injections can be stopped after 4 or 5 days, and after that perhaps given every other day for a few doses. If the symptoms persist, or if they return after injections have been stopped, the treatment must be continued. On the other hand, it sometimes happens that not only is there a decided reaction after each injection, with increased restlessness, nervousness and rise of temperature (Fig. 126 and history Fig. 117, p. 447), but that final disappearance of fever does not occur until the injections are stopped. It may be observed, too, that the polymorphonuclear cells seem to increase after each injection, instead of diminishing. These are indications for abandoning the treatment. After the first 4 days of treatment, therefore, which should be given to every case, the further continuance must be determined for the individual case by carefully judging the demands and the effects produced.

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[1] Deutsch. med. Wochenschr., 1906, XXXII, 609.
[2] Deutsch. med. Wochenschr., 1906, XXXII, 788.
[3] Jour. Exper. Med., 1908, X, 141.
[4] Ann. de l'instit. Pasteur, 1910, XXIV, 96.

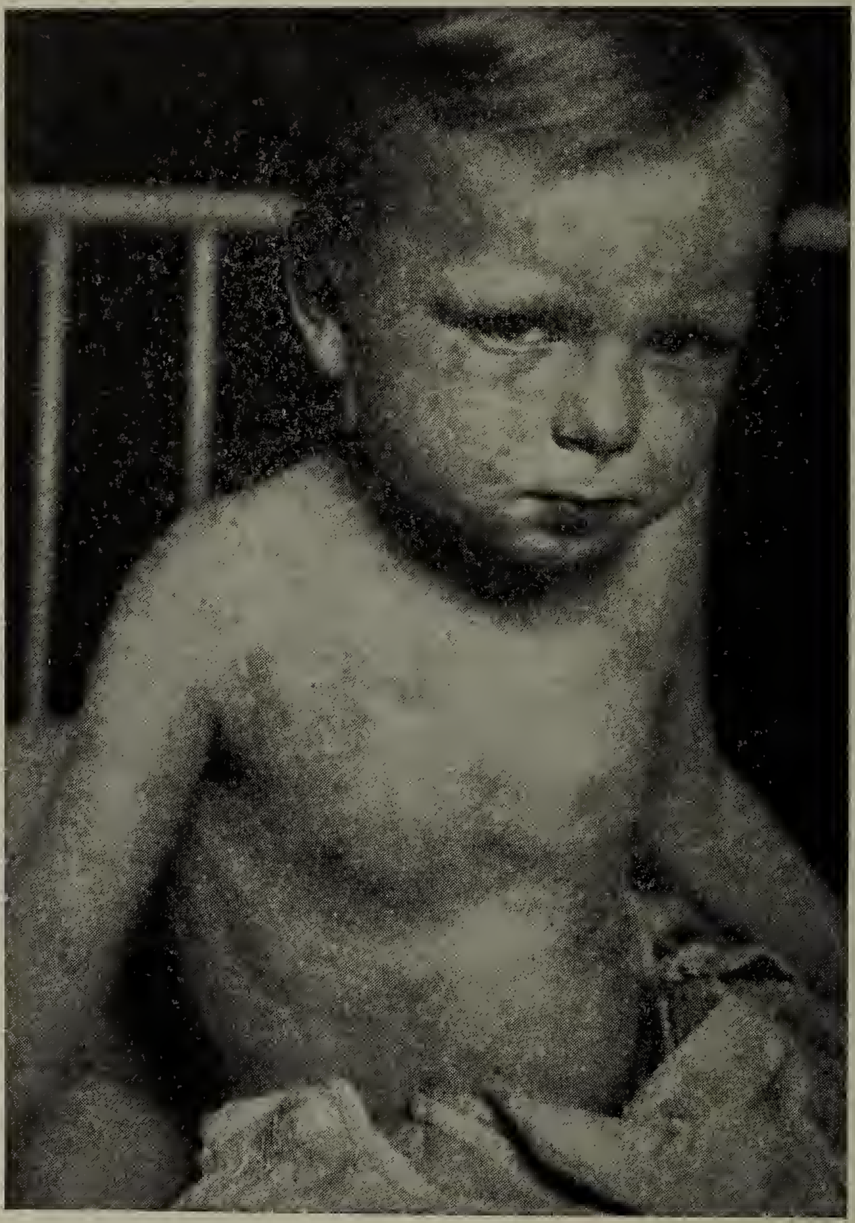

Fig. 127.—Serum-Rash in Cerebrospinal Fever.

Child of 3 years in the Children's Ward of the University Hospital, Philadelphia. Urticarial eruption appeared on the 4th day after the first intraspinal injection of serum; lasted 4 days. Photograph shows wide-spread eruption over the face and body.

As regards the general effect of the serum-treatment upon prognosis, Flexner[1] found that whereas the usual mortality in various parts of the world had reached as high as 70 per cent., in 1294 cases receiving this treatment it had been reduced to 30.9 per cent. The period of the disease at which the treatment is commenced is also of influence. Of Flexner's series it was begun in 199 in the first 3 days, with a resulting mortality of but 18.1 per cent. It is particularly in infancy that the good results are seen. Previously nearly all of this age died, especially subjects under 1 year old, whereas in 129 of Flexner's cases in the 1st year the mortality equalled 49.6 per cent. Between 5 and 10 years of age there were 218 cases with a mortality of 15.1 per cent. Not only is the mortality lessened by serum-treatment, but the attack is shortened and the complications and sequels are diminished also. That serum-treatment is so efficacious in some instances and of so little value in others depends doubtless in part upon the severity of the infection in the individual case, in part upon the promptness with which the treatment was commenced, and in part probably upon the strain of the meningococcus, to which reference has been made (p. 469). If the serum contains a strain which is not the one operative in the case, no benefit can, of course, be expected. For this reason the serum as at present procurable is polyvalent.

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[1] Jour. Exper. Med., 1913, XVII, 553.

It should be remembered that the serum used in the treatment of meningococcus-meningitis is bactericidal in nature and must be brought into contact with the organisms it is to destroy. As has been mentioned, a dry tap in the lumbar region is an indication for the injection of serum into the ventricles. There is evidence that meningococcus-infection of the subarachnoid space takes place through the choroid plexus, and that the inflammatory process develops first in the ventricles (Lewkowicz).[1] For this reason some authors, as Howell and Cohen[2] and Caussade and Remy,[3] recommend early intraventricular injections. Howell and Cohen also found that in certain cases serum introduced subdurally at the outer edge of the fontanelle would reach foci that could not be approached by ventricular or lumbar injections. Again it has been claimed that almost 70 per cent. of the cases of meningococcus meningitis show evidence of general infection, and that the mortality can be greatly reduced by combining intravenous with intraspinous treatment (Herrick;[4] Haden[5]). These injections are especially indicated in the cases of early general septic systemic infection, with or without many symptoms of meningeal involvement. Comparatively large doses should be employed, up to 150 c.c. (5.07 fl. oz.).

In addition to these different portals of entrance to the cerebrospinal system as just mentioned there is another method of approach which has been suggested and employed by Ayer.[6] (See also Wegeforth, Ayer and Essick.)[7] In this procedure the cisterna magna is entered through the foramen magnum. When the fontanelle is closed ventricular puncture can be done only after trephining. Therefore subarachnoid block and a consequent dry lumbar tap in an older child would indicate the use of cisterna-puncture. If it can be shown that the procedure is devoid of danger it would be the method of choice, inasmuch as most of the exudate in any meningitis is usually at the base of the brain and the bactericidal serum could thus be brought directly into contact with the causative organisms. We have used cisterna-puncture and serum-injections many times without ill effect. It is applicable at any age and may effect cure in subarachnoid block in apparently hopeless cases

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[1] Arch. de méd. des enf., 1921, XXIV, 329.
[2] Amer. Jour. Dis. Child., 1922, XXIV, 427.
[3] Paris méd., 1921, XI, 129.
[4] Jour. Amer. Med. Assoc., 1918, LXXI, 612.
[5] Arch. Int. Med., 1919, XXIV, 514.
[6] Arch. Neurol. and Psychiat., 1920, IV, 529.
[7] Amer. Jour. Med. Sci., 1919, CLVII, 789.

(Mitchell and Reilly).[1] The technic of all the procedures mentioned above are described. (See pp. 216 and 217.)

After the acute stage of the disease is over little benefit is to be expected from serum-treatment. Nevertheless it should by all means be tried, as occasionally good results follow. In chronic cases autogeneous vaccine may well be tried. To avoid mistakes in diagnosis it must be remembered that the injection of serum is naturally capable of producing the same symptoms, including the cutaneous eruption, which occur from the injection of antidiphtheritic serum (Fig. 127).

CHAPTER XI

ERYSIPELAS

Although it could well be classified as a form of sepsis, erysipelas has such distinct peculiarities that it deserves separate consideration. At one time a greatly dreaded affection, especially in hospital practice, better sanitary methods have diminished its frequency very greatly.

Etiology.—The disease is wide-spread, climate and locality having no influence upon it. It appears to be more common in the cold season of the year and particularly in spring. Although often occurring in epidemics, and reappearing with especial frequency in certain localities, such as old hospitals not well cared for and in infant asylums, sporadic cases sometimes develop in institutions under the best hygienic regulations.

Age offers no protection, yet the disease appears to be more frequent at certain periods of life. In 1568 cases studied by Roger,[2] 27 occurred in the first 2 years of life; 6 at from 2 to 5 years inclusive; 60 at from 6 to 14 years; 189 at from 15 to 20 years, and 239 at from 21 to 25 years. In rare instances it has been acquired before birth. There is undoubtedly an individual susceptibility, some persons being particularly liable to it, and an inheritance of this susceptibility seems certainly possible. By far the most important predisposing factor is the presence of a *wound.* Consequently infants with an unhealed umbilicus, eczematous areas, lesions of varicella, and the like, or those who have been recently circumcised or vaccinated, are especially predisposed. Sometimes the portal of entry is the mucous membrane, whence the disease spreads to the skin; yet erysipelas often develops without the slightest abrasion being discoverable anywhere. That there are not more individuals with wounds attacked indicates the lack of susceptibility in the majority of them.

Erysipelas is clearly an infectious disease, the germ being transmitted by direct contact or by clothing and the like, by auto-infection or by means of a third person. It is not diffused by the air to any extent. The *specific germ,* the *streptococcus erysipelatis,* first isolated and identified by Fehleisen,[3] has been found to be a form of the streptococcus pyogenes. Streptococci were found in ⅓ of 18 cases in which blood-cultures were

[1] Amer. Jour. Med. Sci., 1922, CLXIV, 66.

[2] Arch. gén. de méd., 1901, CLXXXVIII, 5.

[3] Deutsch. Zeitschr. f. Chir., 1882, XVI, 391.

made by Knox.[1] Tunnicliff[2] concludes that the hemolytic streptococcus found in erysipelas belongs to a distinct group; and thus is supported by the experience of Birkhaug[3] and Rivers.[4] The vitality of the germ of erysipelas is not great, since it lives in cultures not longer than 2 or 3 weeks. Occasionally other germs than the streptococcus appear able to produce the disease.

Pathological Anatomy.—The affected skin exhibits post-mortem an exudation of serum and of lymphoid cells in the cutis and subcutaneous tissue, with dilated and engorged blood-vessels, and with very numerous cocci in the lymph-channels; these being most numerous in the region last involved. Sometimes large bullæ containing serum are found. In the most severe cases evidences of suppuration are observed in the subcutaneous connective tissue. Often alterations are present in other parts of the body, among these being enlargement of the spleen, parenchymatous changes in the liver and kidneys, infarcts in the lungs, kidneys and spleen, septic endo-pericarditis and pleuritis, lymphatic swelling, peritonitis, and occasionally meningitis.

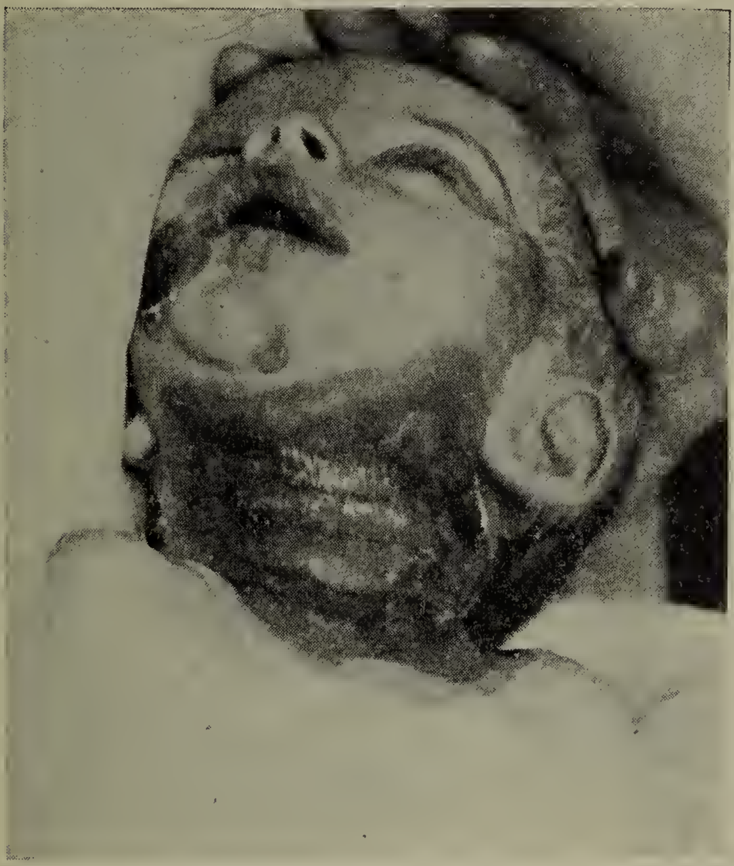

Fig. 128.—Erysipelas Beginning on the Neck

Child of 7 weeks, in the Children's Ward of the University Hospital in Philadelphia, suffering from chronic gastrointestinal disturbance. Improved slowly during 2 months. Then developed fever and eruption of erysipelas on the neck, with large bullæ. Died after an illness of less than a week.

Symptoms. Incubation.—The period of incubation is generally short, from 15 to 60 hours as proven by the inoculation-experiments of Fehleisen.

Symptoms of the Attack.—Initial symptoms may be absent, or consist of chilliness, coldness of the extremities, restlessness, prostration, vomiting, high fever, and occasionally convulsions. Simultaneously, or nearly so, redness, swelling and tenderness of the skin develop in the affected area. Roger's[1] statistics show that the face is much most frequently the starting point. This was true of 83 per cent. of the cases at all ages. In 66 children of from 2 to 15 years the disease began in the lower extremities in but 4 instances. The redness exhibits the uniform flush of a dermatitis, which it is; unlike that of scarlet fever. Its border is sharply defined, elevated, and either clean-cut or with irregular projections jutting out here and there into the healthy skin. When the process is severe vesicles or bullæ form upon the affected skin (Fig. 128). Within 2 to 3 days the inflammation begins to disappear in the part first involved, and desquamation follows, usually in fine scales, but coarser and in larger pieces if vesicles have been present. In the meantime the border of the infiltrated region has extended more or less rapidly, sometimes a considerable area becoming involved in a few hours, and sometimes the advance being very slow.

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[1] South. Med. Jour., 1921, XIV, 387.
[2] Jour. Amer. Med. Assoc., 1920, LXXV, 1339.
[3] Johns. Hopk. Hosp. Bull., 1925, XXXVIII. 307.
[4] Jour. Exper. Med., 1925, XLI, 179.

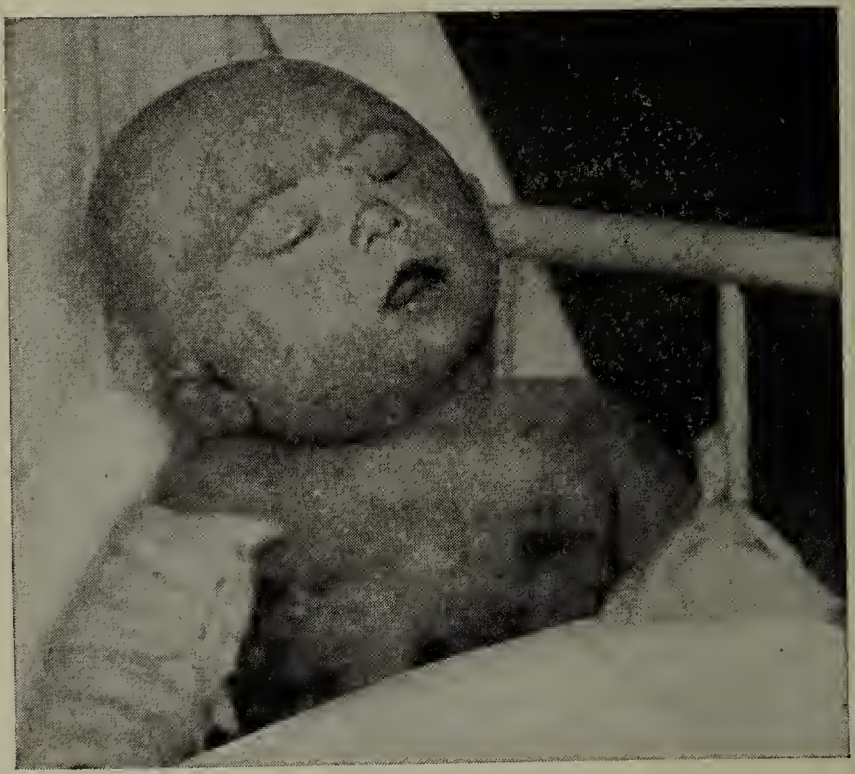

FIG. 129.—ERYSIPELAS WITH GREAT SWELLING OF THE HEAD.

Child of 14 months, in the Children's Hospital of Philadelphia. Disease began in the face, involved the head, closing the eyes, thence spread to the rest of the body. Death 9 days after the onset.

The manner of the spreading and the degree of swelling depend largely upon the locality. Where the skin is firmly adherent to underlying structures, as at the chin, the patellæ and the condyles, the disease often passes around, leaving these areas unaffected. Where the tissue is loose, as at the genitals and the eyelids, the edema is great. When the disease starts at the nose this organ swells rapidly and the rash generally quickly spreads to the cheeks in the well-known butterfly form. It often stops here, but it may involve the eyelids, closing them for days, or may extend over one or both sides of the face and to the ears (Fig. 129). The whole scalp may be finally involved. Under such circumstances the head and face seem twice their natural size and the child is entirely unrecognizable. The inflammation may cease here, or may spread to the body. Developing about a vaccine pustule or other lesion on the arm or leg, it may remain confined to this locality, or may extend rapidly over the whole limb and thence to other parts, but not so often to the head as elsewhere. In some cases the disease appears to start on the mucous membrane of the nose, throat, or mouth and spread to the face, the first symptoms being a severe angina or coryza. In other cases it first attacks the mucous membrane of the vulva and extends to the thighs and other regions.

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[1] *Loc. cit.*

The tendency of the rash to spread varies greatly. It may be very slight, the dermatitis reaching but little beyond the point of original appearance. On the other hand the eruption may wander more or less rapidly over the whole body (*erysipelas migrans*), returning and attacking again parts from which it had disappeared but a short time before. This spreading is often in the form of repeated short advances, with intermissions during which improvement in the general symptoms leads to the false hope that the attack is over.

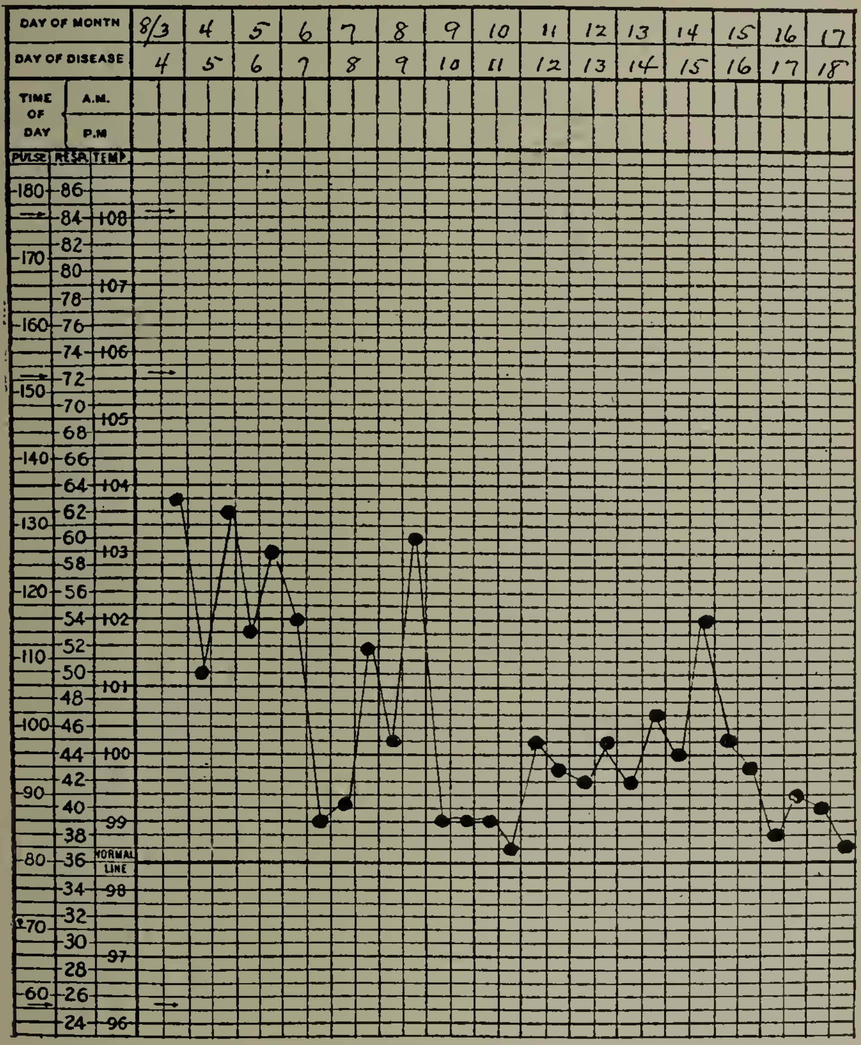

Fig. 130.

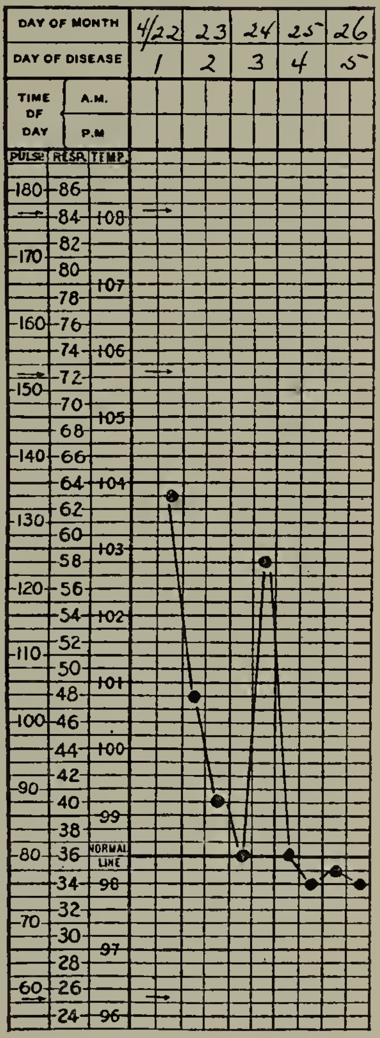

Fig. 131.

Fig. 130.—Erysipelas, Average Case.

Elsie S., aged 3½ years. Aug. 3, 4th day of disease, eruption spreading over left leg; Aug. 5, involving foot; Aug. 6, no extension; Aug. 8, rapid extension to buttock with increase of fever; Aug. 13, fresh extension, involving right leg and trunk; Aug. 14, development of bullæ, with increase of fever; Aug. 17, convalescing.

Fig. 131.—Mild Erysipelas with Recrudescence of Fever Attending Extension of Rash.

Edith S., aged 7 years. Apr. 22, red flush right cheek, extending over nose; Apr. 23, large blebs on right cheek, eye nearly closed; Apr. 24, extension to left cheek with renewed fever; Apr. 26, improving.

Symptoms Attending the Eruption.—The temperature as a rule rises very rapidly and remains at 104°F. (40°C.) or more, with but slight morning fall, as long as the spread of the rash is uninterrupted (Fig. 130). In very mild cases fever may be entirely absent or undiscovered. It generally is in proportion to the severity of the cutaneous manifestations. Each temporary cessation of the spreading of the disease, with subsequent recrudescence, usually is indicated by a fall of temperature followed by a rise (Fig. 131). An intermittent extension has therefore an intermittent temperature; a steady spread, a temperature more continuously elevated. The lymphatic glands in the neighborhood of the dermatitis are nearly always inflamed. The spleen is often enlarged. In severe cases the appetite is poor, the tongue dry, the pulse weak and respiration sometimes dyspneic. Restlessness, delirium or sopor not infrequently occur. The blood shows a leucocytosis most marked in the severe cases. Transient albuminuria is occasionally present. The complex of symptoms varies, being often influenced by the complications which are prone to occur (Figs. 132 and 133).

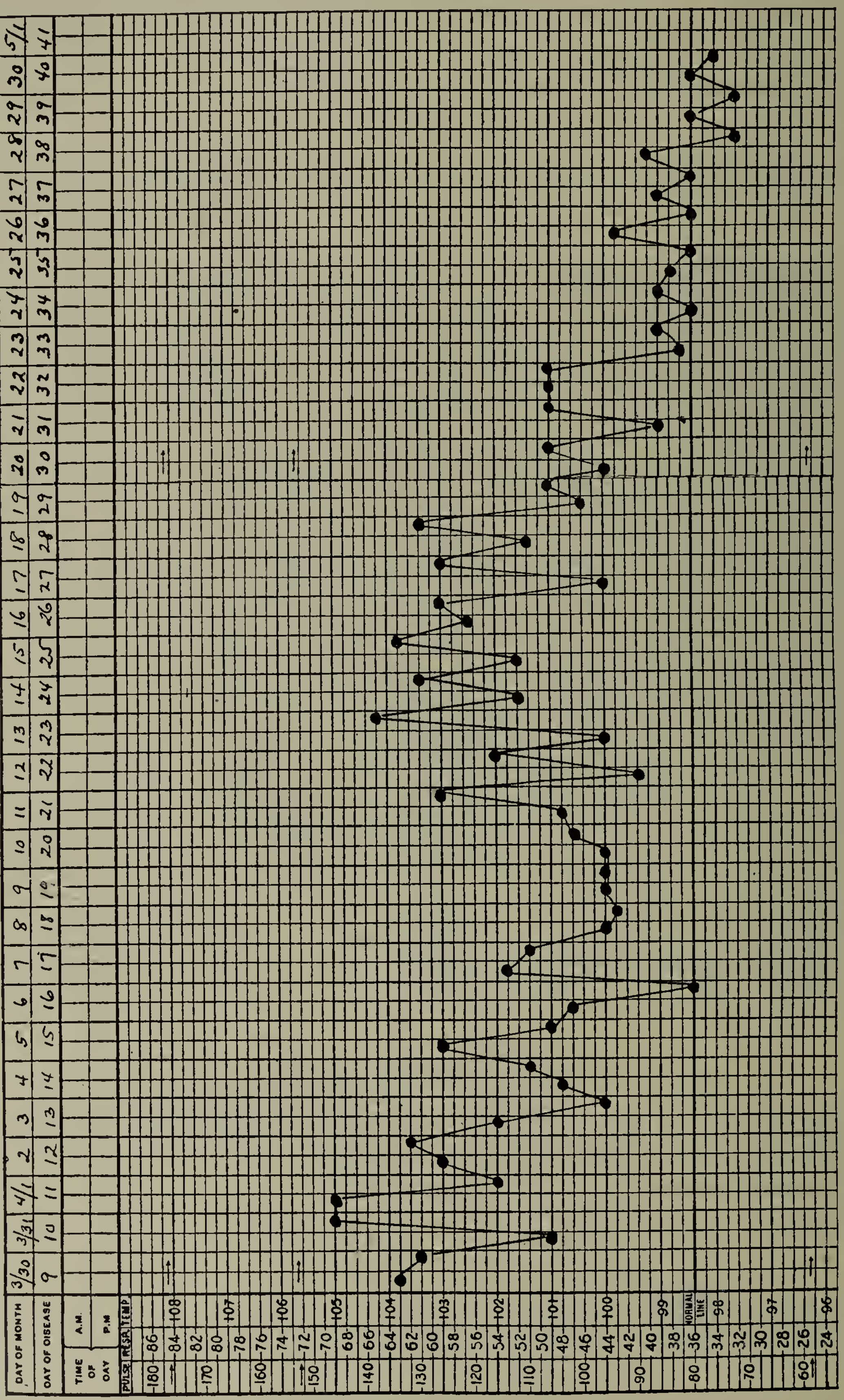

Fig. 132.—Erysipelas with Multiple Cutaneous Abscesses.

John B., aged 3 years. Onset with vomiting, fever, and redness and swelling of face. Then developed meningitic symptoms, with delirium, retraction of head, rigidity of limbs. Lumbar puncture negative. Moderate leucocytosis. First abscess formed on head on the 11th day. Eruption extended to upper extremities and trunk, with formation of numerous subcutaneous abscesses here, and fresh ones on the head. Pus contained streptococci. Emaciation and weakness great. Slow disappearance of meningitic symptoms. Final recovery.

The *duration* of the disease is extremely variable. As already stated the height of the affection in any one spot is reached in 2 to 3 days, and recovery in that region is rapid. An average duration of the entire attack is 7 to 9 days, but may last less than this or often much longer, and in cases of erysipelas migrans may continue occasionally even for months.

Erysipelas in Early Infancy.—This condition, and especially that denominated *Erysipelas neonatorum*, differs somewhat from the disease as seen later. The regions first attacked are oftenest the umbilicus, vulva, anus, and the lesions of circumcision or vaccination, rather than the head. The onset is more insidious, and the child is restless and fretful, but often without fever for some hours or even days, and sometimes at any period. When fever develops the spleen enlarges, vomiting and diarrhea are likely to occur and the dermatitis spreads widely. Loss of strength is rapid, septic symptoms appear, the pulse is very rapid and weak, food is refused, collapses upervenes, convulsions may appear, and

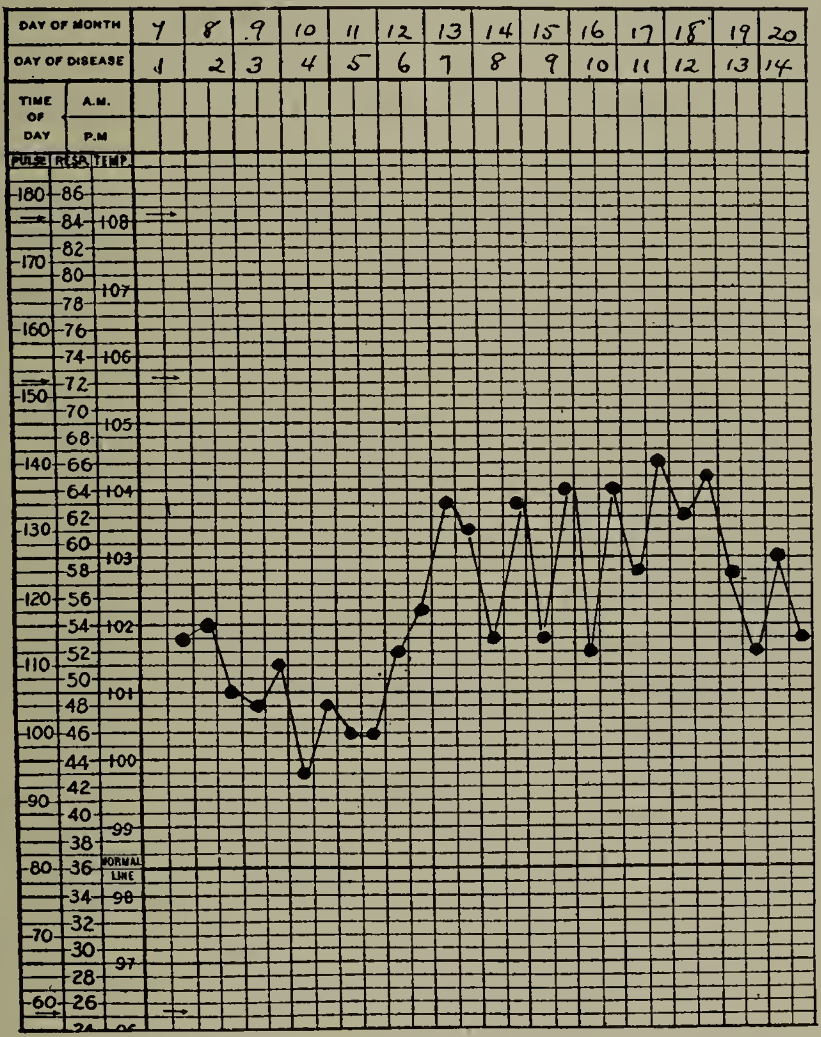

Fig. 133.—Erysipelas, Severe Case. Bronchopneumonia.

Adelaide C., aged 7 months. Feb. 7, swelling of left labium; Feb. 14, involvement of right labium; Feb. 16, bronchopneumonia; Feb. 20, rash has been steadily spreading, involving abdomen and lower extremities; severe toxic state; death.

death may follow within a week or even a day or two from the onset. There is a decided tendency for the disease to assume the wandering type if the infant lives long enough. The eruption differs little from that seen later in life, except that the swelling is liable to be greater and more tense, the redness less intense, the boundary less raised and desquamation more liable to occur. The cases which recover generally develop cutaneous abscesses.

Complications and Sequels of Erysipelas.—Suppuration in the subcutaneous tissues is not infrequent (Fig. 132), and necrosis of portions of the skin have been reported. Bronchitis, pneumonia (Fig. 133), peritonitis and endocarditis may occur, and edema of the larynx sometimes develops. Meningitis is an occasional complication, although many subjects exhibiting its symptoms show no such lesion at autopsy.

Nephritis is not common. Suppuration of joints or of lymphatic glands is sometimes seen.

Erysipelas may occur in combination with many other diseased conditions, especially, as indicated, with wounds. It may be a complication of other infectious disorders, as scarlatina, malaria, typhoid fever, cerebrospinal fever and diphtheria. In the last three it generally results from the development of a bed-sore or other open wound.

Relapse and Recurrence.—In addition to the frequent recrudescences with psuedo-crises so characteristic of the disease, true *relapse* may show itself after several days or even a few weeks. It probably owes its origin to the remaining of small infiltrated, infected foci in the skin. The relapse may be repeated a number of times.

Recurrence in the sense of a new infection is more common than relapse. In fact, one attack creates absolutely no immunity, but rather seems to predispose to later ones. Frequent recurrence is liable to result in permanent thickening of the affected areas, especially the scrotum and the eyelids.

Prognosis.—One of the most important factors is age. In the new-born erysipelas is almost always fatal and at any period of infancy it is serious, especially in the first 2 or 3 months of life. In childhood recovery generally takes place if the disease is uncomplicated and of moderate extent.

The situation involved is of importance as well. The prognosis is prone to be worse if the head is attacked. Wandering erysipelas has a graver prognosis than the more limited form, since its long duration tends to produce exhaustion.

Among unfavorable symptoms are very high temperature, weakness of the heart, and cerebral symptoms. All complications increase the gravity of the case and death may follow pneumonia and sometimes peritonitis, meningitis, and involvement of the kidneys.

Diagnosis.—This rests upon the combination of constitutional symptoms with a dermatitis of peculiar nature, often with vesicles and with marked tendency to extension. The latter features and the sharply defined border differentiate it from other forms of dermatitis. The rash of scarlet fever is distinguished by its punctiform character. Deep-seated phlegmonous inflammation lacks the characteristic border, as does also lymphangitis, which has, too, an entirely different outline, since it follows the course of the lymphatic vessels. Erythema infectiosum, (see p. 410) while having a distribution and color upon the face suggesting erysipelas, lacks the sharp outline and the infiltration, and exhibits a morbiliform rash on the trunk and limbs.

Treatment. **Prophylaxis.**—Especial care should be taken to insure asepsis of the umbilical wound of the new-born. Infants should be removed from their mothers if the latter are suffering from puerperal fever. All possible precautions should be employed in performing vaccination and circumcision, and all children with open wounds should be carefully separated from patients with erysipelas.

Treatment of the Attack.—So far as drugs are concerned, although the number tried is very great, none can be called specific. The internal administration of tincture of the chloride of iron in large doses has long been a favorite. There appears to be some evidence that it does good, and at any rate it can be said for it that it can do no harm. Quinine and salicylic acid in full doses are each favorite remedies with many. Locally the remedies most in favor are petrolatum, ointment of ichthyol (5 to

15 per cent. or stronger); ointment or glycerin-solution of resorcin (30 per cent. or more); powders of iodoform, oxide of zinc, or starch and salicylic acid; compresses or solutions of bichloride of mercury (1:2000), boric acid, lead water and laudanum, and alcohol and water. Of all of these the application of ichthyol ointment or of ichthyol collodion (10 per cent. or more) is perhaps one of the most popular, and the course of the disease often appears to be much abbreviated by the treatment. Collodion, either plain or with ichthyol, may be painted beyond the margin of the eruption and frequently and freshly applied, with the hope that the spread of the rash may be prevented by shutting off the lymphatic channels. Hütten[1] employed for this same purpose and with encouraging results the freezing induced by ethyl chloride. Subcutaneous or intramuscular injections of anti-streptococcic serum or of autogeneous vaccines have been employed, but the results have been inconclusive. I have seen prompt recovery follow their use, but could not determine that this was because of it. Erdman's[2] experience in 800 cases of erysipelas did not show any shortening of the attack in 95 instances in which vaccines had been used. The apparent causative relationship of a special hemolytic streptococcus offers hope for the development of a specific serum. Birkhaug,[3] Petenyi[4] and Zahorsky[5] have seen rapid improvement follow exposure to the quartz lamp. Jordan and Dustin[6] report favorable results with the employment of convalescent's serum, taken a week after the temperature has fallen to normal. The collection and preservation of the serum is the same as for the convalescent's serum of scarlet fever (see p. 376).

Other treatment is symptomatic. The general strength, and especially that of the heart, may require alcoholic stimulants and digitalis. Suitable nourishment must be given; excessive temperature may require warm or cool bathing; threatening nervous symptoms may be treated hydropathically or may need bromides, antipyrine, or occasionally, opium. Complications call for treatment appropriate to them.

CHAPTER XII

DIPHTHERIA

History.—Recognition of an infectious epidemic disorder characterized by the development of a membrane-like coating, generally situated in the fauces, nose or larynx, appears to date from very early times. Perhaps the first clear account was given by Aretaeus[7] in the 1st century. It ravaged Europe in the 16th and 17th centuries and was described in America by Douglas[8] in 1736 and very completely by Bard[9] in 1771

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[1] Beitrage z. klin. Chir., 1925, 1925, CXXXIII, 252.

[2] Jour. Amer. Med. Assoc., 1913, LXI, 2048.

[3] *Loc. cit.*

[4] Monatsschr. f. Kinderheilk., 1921, XXI, 269.

[5] Jour. Missouri State Med. Assoc., 1925, XXII, 61.

[6] Jour. Amer. Med. Assoc., 1924, LXXXII, 874.

[7] De causis et signis acut. morb. L. I, Cap. 9. Ref. Baginsky in Nothnagel's Handb. d. spec. Path. u. Therap., Diphtheria, 3.

[8] Pract. hist. of a new erupt. miliarial fever with angina ulcusculosa, Bost., 1736. Ref. Baginsky, *loc. cit.*, 6.

[9] Transac. Amer. Philosoph. Soc., 1779, I, 338.

Variously designated, it was first called "Diphtheritis" (διφθερα = a membrane or skin) by Bretonneau.[1] Since the discovery of the specific bacillus by Klebs the term diphtheria is properly applied only to the disease dependent upon this germ. The other pseudomembranous affections of the throat should be entitled pseudodiphtheria.

Etiology. Predisposing Causes.—The disease is of very frequent occurrence, and prevails in all climates and all civilized countries without regard to locality, although perhaps most common in cold, damp regions and in the cooler months of the year. Humidity is a decided factor, probably through its influence in favoring catarrhal affections, any acute or chronic catarrhal disorder of the mucous membrane of the nose or throat, as also the presence of adenoids and of enlarged tonsils, rendering the subject more susceptible. Ochsenius,[2] however, holds that humidity of the air predisposes to diphtheria, not by favoring the growth of the bacillus, but by lowering the resistance to disease. Poor sanitation of any sort, and impaired health in general, increase the tendency to diphtheria by diminishing the resisting powers. For this reason certain other infectious disorders, especially scarlet fever and measles, as well as influenza and pertussis, augment the susceptibility. It may follow tonsillectomy. The tendency to develop the disease is greater among children living in the less congested sections of a city than in the more crowded districts, since it is probable that repeated exposure to infection by the diphtheria-bacillus, in a dosage insufficient actually to cause the disease, results in the development of immunity (Zingher).[3]

Age is an important predisposing factor. The disease is most frequent up to 10 years, and especially from 1 to 5 years of age. Of 9011 cases admitted to the Philadelphia Hospital for Contagious Diseases (Welch and Schamberg)[4] 4076 were from 1 to 5 years old. It is less common in the 1st year and especially in the first 6 months. In 2600 cases reported by Rolleston[5] there were but 20 occurring in the 1st year; and in 2711 cases in children Baginsky[6] found only 15 (0.15 per cent.) under 6 months of age. This lesser occurrence in very young infants depends upon a natural immunity at this age, not upon the absence of exposure; since new-born infants exposed to the disease very readily become carriers yet without developing diphtheria. Yet new-born children are occasionally attacked (Jacobi).[7] (Riesman,[8] infant of 11 days; Stimson,[9] 10 days, primary nasal. Other cases are reported by Becker,[10] Rominger,[11] Brindeau,[12] Comby[13] and others.) Sex has some influence, statistics showing girls slightly more frequent sufferers than boys (Zingher). There exists often a very striking family predisposition, but apart from this the *individual susceptibility* is not very great and the disease much oftener limits itself to a single child in a family than is the case in some other infectious disorders, notably measles. The immunity (see p.

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[1] Des inflam. spéc. du tissu muqueux etc., Paris, 1826. Ref. Baginsky, *loc. cit.*

[2] Monatsschr. f. Kinderheilk., 1923, XXVI, 266.

[3] Amer. Jour. Dis. Child., 1923, XXV, 392.

[4] Acute Contagious Diseases, 1905, 611.

[5] Amer. Jour. Dis. Child., 1916, XII, 47.

[6] *Loc. cit.*, 52.

[7] 20th Cent. Pract. Med., XVII, 77, Article Diphtheria.

[8] Phila. Med. Jour., 1898, I, 423.

[9] New York Med. Jour., 1907, LXXXVI, 1123.

[10] Zentralbl. f. Gynakol., 1919, XLIII, 996.

[11] Zeitschr. f. Kinderheilk., 1920, XXVIII, 51.

[12] Bull. acad. de méd., Paris, 1921, LXXXVI, 191.

[13] Arch. de. méd. des enf., 1921, XXIV, 437.

516) may be overcome through various influences, and as a result diphtheria may appear at times in extensive epidemics in some years with but few cases in others. In large cities it is *endemic* to a varying degree, and it may develop sporadically in localities where its origin cannot be traced.

Exciting Cause.—This is now well recognized to be a specific bacillus first recognized by Klebs[1] in 1883, and shown to be the sole cause of the disease by Löffler[2] in 1884. Roux and Yersin[3] in 1888 proved that this germ was capable of occasioning in animals the same paralytic conditions as are seen in man, and demonstrated the production by it of a poisonous substance upon which the various symptoms of the disease depend. All later observations confirm the etiological relationship of the germ.

The microörganism is a non-motile, non-spore-bearing Gram-positive bacillus, averaging about the length of the tubercle-bacillus but thicker. It is straight or slightly curved and often somewhat club-shaped at the ends. It has the characteristic peculiarity of varying greatly in size, form and staining qualities, depending on its age and on the culture-medium employed; and stains well with alkaline methylene blue as recommended by Löffler, but always with unstained spots, the club-shaped ends being most intensely colored.

Life-history.—The bacillus is readily killed by a temperature of 58°C. (136.4°F.) but is not affected by cold. Under normal conditions it is very tenacious of life. In the great majority of cases it disappears from the throat in 2 to 4 weeks from the beginning of the disease, but it may sometimes persist for weeks or months in a virulent form in the throat or nose of those who have passed through an attack or who have merely been exposed, or it may live long in various objects entirely apart from the body. Abel[4] found it living for over 6 months on toys which had been kept in a dark place, Klein[5] for 18 months in cultures, Le Gendre and Pochon[6] for 15 months in the throat, and Valagussa[7] completely dry but living for 26 months. It occurs sometimes in the secretion from purulent otitis media or may occasionally be found in the tissues of the body or the blood and the urine. Thus in 209 fatal cases Councilman, Mallory and Pearce[8] discovered it in the heart's blood 12 times, in the liver 42 times, and in the spleen 26 times; frequently associated with the streptococcus, less often with the staphylococcus or the pneumococcus. Sommerfeld[9] observed it in the blood in 42 out of 320 cases (13.1 per cent.). It has also been found in the lungs, bone-marrow, kidneys and lymphatic glands. The diphtheria-germ is, however, as a rule not widely distributed in the body, being confined in most cases to the pseudomembrane and the surface of the mucous membrane.

The disease is *primarily a local one*, the germs being deposited and growing upon a mucous membrane which was not in a healthy condition. The symptoms resulting depend chiefly on the absorption of the poisonous principle produced by the bacillus, and perhaps partly upon the action of associated germs especially the streptococcus pyogenes and the staphylococcus pyogenes. There is no diphtheria without the specific germ, and it is equally true that the mere discovery of this on the mucous membrane does not indicate the presence of diphtheria.

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[1] Verhandl. d. Cong. f. inn. Med., 1883, II, 139.
[2] Verhandl. d. Cong. f. inn. Med., 1884, III, 156.
[3] Annal. de l'instit. Pasteur, 1888, II, 629.
[4] Centralbl. f. Bakt., u. Parasit., 1893, XIV, 756.
[5] Centralbl. f. Bakt., u. Parasit., 1890, VII, 492.
[6] Bull. soc. des hôp., 1895, XII, 815.
[7] Riv. di clin. pediat., 1909, VII, 332.
[8] Bact. and Pathol. of Diphtheria, 1901, 16.
[9] Arch. f. Kinderheilk., 1913, LX–LXI, 698.

Diphtheria-bacilli may lose their virulence, and such germs, although morphologically and in reality identical with the virulent ones, are without danger to those acquiring them. As a rule, the more virulent the germ, the worse the attack of the disease; but to this there are many exceptions, and the mildest cases may transmit very virulent germs and occasion a severe attack in another subject.

Another organism has been described, known as the pseudo-diphtheria bacillus, or the Hofmann-Wellenhof[1] bacillus, which is very similar to, but not identical morphologically and culturally with that of diphtheria, and is not capable of producing the disease. Whether this is, in reality, different from the avirulent Klebs-Löffler's bacillus, or only a variant, is not yet positively determined, but the latter seems very possible. The xerosis-bacillus likewise possesses a resemblance to that of diphtheria. The studies of Guthrie, Marshall and Moss,[2] in which experimental inoculations of the human throat were performed, have done much to clear up the differentiation of the true disease-producing diphtheria-bacilli from the pseudo-diphtheria organisms (diphtheroids).

Transmission.—The disease is a strictly *infectious* one and never develops independently. Indirect transmission is much the least frequent method of communicating the infection. It may occur through infected milk, or occasionally by domestic animals, or by books, toys, clothing and other inanimate objects. Litterer[3] has isolated virulent diphtheria-bacilli from fowls. This can occur without the fowls themselves being ill in any way (Spiegelberg).[4] Simmons[5] reported virulent bacilli in cats themselves ill, which had been associated with a diphtheria-patient. The belief in extension by means of cats is of long standing. Savage,[6] however, has analyzed the available reported cases, and found no positive evidence that the disease actually attacks these animals. Graham and Golaz[7] traced 52 out of 71 cases of diphtheria to infected milk.

In the large majority of cases the germs are communicated directly, either from an unrecognized case, or by a convalescent, or by a healthy person who has never had diphtheria but who has the virulent bacteria present in the throat or nose. Such convalescents and healthy persons are called "carriers." The remarkable persistence of the germs on the mucous membranes of convalescents or of healthy persons which is frequently seen, and their tenacity of life, account for cases which occur sporadically and without discoverable source. The existence of mild unrecognized nasal diphtheria is a very fruitful cause of dissemination of the disease in schools, hospitals, and elsewhere. Transmission may occur from the beginning of the attack or even before any symptoms appear. It continues possible as long as the bacilli remain virulent in the carrier. The difficulty in controlling the spread of the disease is shown by the investigations of the Massachusetts Board of Health,[8] according to which at least from 1 to 2 per cent. of all dwellers in cities have genuine diphtheria-bacilli on the mucous membrane of the throat, as have 8 to 50 per cent. of those who have been in any way exposed to the disease. Pennington[1] found that 10 per cent. of all the apparently healthy children examined in the public schools of Philadelphia exhibited diphtheria-bacilli, nonvirulent in about ½ of the cases. von Shoolly[2] discovered the germs in the throats of 5.6 per cent. of 1000 healthy school-children; and Schrammen[3] in 6.5 per cent. of 704 school-children, although at the time there was not a single case of diphtheria in the schools or in the families of the children. Doull and Fales[4] found that of 7790 school-children in Baltimore 1.75 per cent. carried virulent diphtheria-organisms.

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[1] Wien. med. Wochenschr., 1888, XXXVIII, 60.
[2] Johns Hopk. Hosp. Bull., 1921, XXXII, 369.
[3] South. Med. Jour., 1923, XVI, 514.
[4] Zeitschr. f. Bact., 1915, LXXV, 273.
[5] Amer. Jour. Med. Sci., 1920, CLX, 589.
[6] Jour. of Hyg., 1920, XVIII, 448.
[7] Jour. Amer. Med. Assoc., 1922, LXXIX, 1300.
[8] Jour. Mass. Assoc. Boards of Health, 1902, July. Ref., Welch and Schamberg, Acute Contagious Diseases, 1905, 619.

Pathological Anatomy.—The lesions of diphtheria may be divided into primary and secondary. The *primary lesion* consists in the production of the pseudo membrane. This is found oftenest upon the tonsillar tissue in the fauces and the nasopharynx; very frequently in addition upon the pharynx, uvula, larynx, trachea and bronchi; less often in the mouth, vulva, vagina, middle ear, conjunctiva, esophagus, stomach and intestine. It may also occur occasionally on wounds, as upon the penis after circumcision or on the umbilicus in the new-born. Macroscopically the pseudo membrane is a greyish-white or yellowish-white substance, tough or friable, covering a smaller or larger area. In places it can be removed rather readily, but for the most part only with injury to the tissues beneath. On the larynx it is firmly adherent; on the trachea easily detached. In severe cases it soon assumes a dark-greenish or blackish color or a gangrenous appearance. The surrounding tissue is deeply congested.

Microscopically the pseudomembrane consists of a degeneration and necrosis of the mucous membrane combined with an inflammatory fibrinous exudate from the underlying blood-vessels. This exudate may be in granular form, but oftener appears as a fine network which includes in its meshes leucocytes and degenerated epithelial cells as well as diphtheria-bacilli and micrococci. The blood-vessels and connective tissue beneath the pseudo membrane exhibit thickening and hyaline transformation. The diphtheria-bacilli do not reach the lowest layers of the exudate.

A distinct pseudo membrane is not necessarily present in diphtheria. Not infrequently there appear instead merely a few spots of yellowish, soft secretion in the crypts of the tonsils (*diphtheritic folliculitis*), while in other cases only a catarrhal condition of the mucous membrane of the throat is discoverable (*catarrhal diphtheria*). Nevertheless, even in the latter condition, the characteristic necrotic changes can be discovered in the superficial cells.

The primary lesion of diphtheria possesses little importance except when in the larynx, where by its mechanical interference it may cause suffocation. It is the *secondary lesions* which constitute the chief danger of the disease. These are the wide-spread degenerative changes in the various tissues of the body, the result of the action of the powerful toxin produced by the diphtheria-bacillus in the primary lesions and absorbed and distributed by the blood and the lymph. Other microbes present in the pseudo membrane probably aid in the destructive process, either by the local formation of toxins and the subsequent absorption of these, or by directly entering the blood-vessels and producing a septicemia; but the chief danger is from the toxin of the diphtheria-bacillus itself. The changes consist in cellular degeneration in all the organs. The cervical, bronchial, and mesenteric lymphatic glands are enlarged and congested and show leucocytic infiltration and often hemorrhages. Scattered bronchopneumonic areas are common, found in over ½ of the autopsies. A serous or sero-fibrinous pleurisy may occur. Small hemorrhages may be present in the skin, pleura, pericardium and endocardium and beneath the capsule of the liver and spleen. The cardiac muscle suffers degeneration in all long-continued severe cases, but, according to some investigators, this is probably not a specific myocarditis, but rather the cloudy swelling common to other acute infections. Nuzum[1] has observed an increased number of eosinophilic cells in diphtheritic myocarditis; a phenomenon not discovered in the myocarditis of other infections. In fatal cases there is always found hyalin degeneration and necrosis (Warthin).[2] The arteries sometimes exhibit proliferation in the endothelial lining. The spleen is increased in size, soft, and dark-red; its follicles enlarged, cellular degeneration prominent and hemorrhages frequent. The kidneys are larger than normal, soft, and exhibit changes varying from simple degeneration to a serious degree of acute nephritis, there being no type of nephritis peculiar to diphtheria (Councilman, Mallory and Pearce).[3] The interstitial and glomerular forms are more common in older children and in long-continued cases. The liver is slightly enlarged and soft and with necrotic alteration of the cells, especially in the interior of the lobules. The brain and spinal cord are generally little altered. Occasionally hemorrhages or a moderate fatty degeneration of the white substance is found. The cranial and spinal nerves exhibit a wide-spread degeneration.

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[1] Jour. Infect. Dis., 1907, IV, 36.

[2] Research Lab., Dept. of Health, New York City, 1905, I, 88; Jour. Infect Dis., 1907, IV, 337.

[3] Centralbl. f. Bakt., u. Parasit., Orig., 1912–13, LXVII, 423.

[4] Amer. Jour. Hyg., 1923, III, 604.

Symptoms.—The symptoms of diphtheria vary greatly with the locality affected, as well as with the intensity of the intoxication and the complications which may arise. A general description may be given of an average case of the commoner form, faucial diphtheria, as illustrating the ordinary type of the disease.

ORDINARY TYPE. FAUCIAL DIPHTHERIA. **Incubation.**—This is certainly short, varying generally from 1 to 4 days. The difficulty in determining it exactly depends on the uncertainty as to the length of time the bacilli may have been living upon the mucous membrane without producing local or general infection.

Invasion.—Symptoms of invasion are by no means characteristic. Although the process is primarily local, the first clinical manifestations noticed are more constitutional in nature, and consist in chilliness, slight fever, and loss of appetite; sometimes vomiting, headache, and swelling of the cervical glands, and occasionally convulsions. There may or may not be complaint of sore throat. Inspection shows only slight coating of the tongue, an irregular redness and swelling of one or both tonsillar regions, and often a dark-red coloring of the mucous membrane of the mouth.

Symptoms of the Attack.—Within 24 hours, however, the appearance is more characteristic and a small or larger yellowish-white or grayish-white deposit is seen, which resembles the secretion of ordinary follicular tonsillitis, but which is removable only with difficulty, as a rule, and appears to be of a more membrane-like character. New membrane generally forms rapidly after such removal. Sometimes the early appearance of the deposit is more gelatinous, the tonsil seeming to be covered by a slightly cloudy mucilaginous secretion.

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[1] Jour. Amer. Med. Assoc., 1919, LXXIII, 1925.

[2] Jour. Infect. Dis., 1924, XXXV, 32.

[3] *Loc. cit.*, 152.

The pseudo membrane spreads, and, if limited to one tonsil at the outset, extends to the other in from 2 to 5 days and often to the uvula, the pillars of the palate and the soft palate. Its color has, in the meantime, become a dirty grey, the odor from the mouth is very offensive, the swelling has much increased, and by the end of the 1st week the entire fauces, and to some extent the pharynx, may be covered by a thick pseudo membrane, sometimes almost closing the throat, the nose being also, as a rule, involved to some extent. (See Nasal Diphtheria, p. 504.) The degree of difficulty in swallowing, and the amount of pain in the throat are very variable and do not seem to bear any relation to the extent of the inflammation.

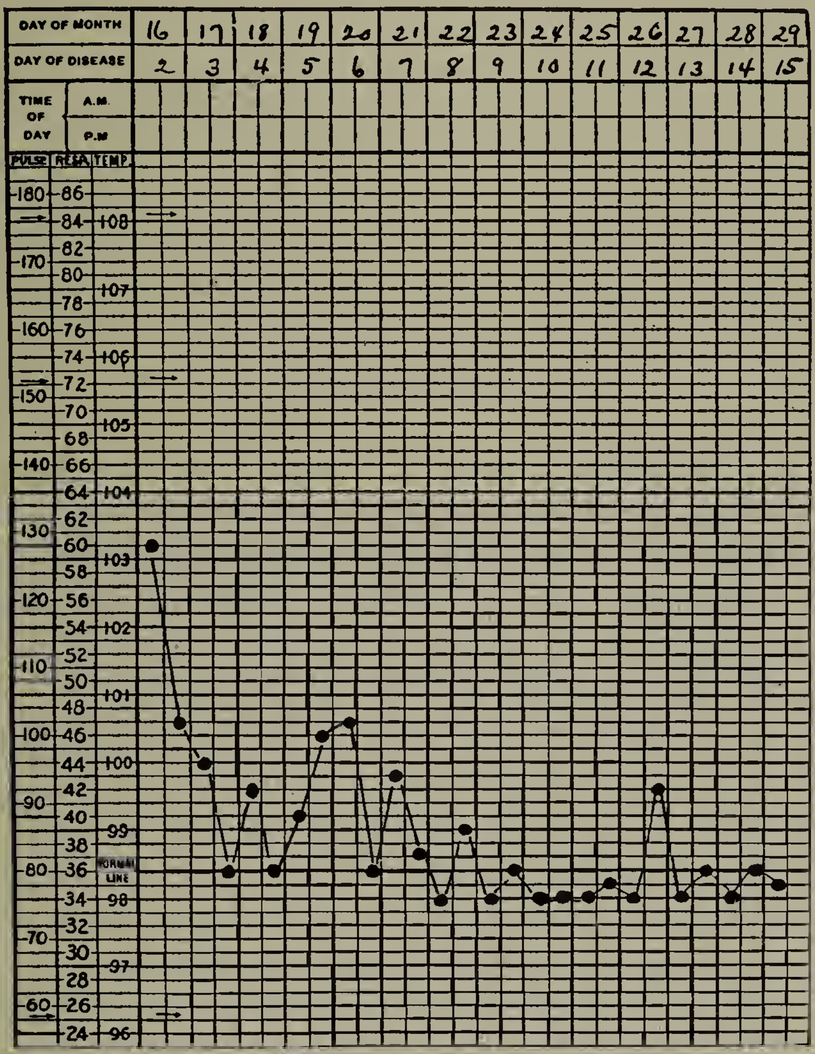

FIG. 134.—TONSILLAR AND NASAL DIPHTHERIA.

Mary K., aged 9 years. Feb. 15, onset with nausea, vomiting, sore throat, and fever; Feb. 16, tonsils and arches covered, nasal discharge; Feb. 18, great improvement; Feb. 21, throat clean. Given 7500 units antitoxin on 16th; 7500 on 17th; 6000 on 18th; 5000 on 19th. Philadelphia Hospital for Contagious Diseases. *Courtesy of Dr. B. F Royer*

Meanwhile the constitutional symptoms increase gradually in severity. The cervical glands grow larger, generally in proportion to the degree of faucial inflammation. The *temperature* exhibits no exact relation to the severity of the symptoms and is usually about 101° to 102°F. (38.3° to 38.9°C.); this comparatively low degree being characteristic of the disease (Fig. 134). Sometimes, however, it reaches 103° or 104°F. (39.4° to 40°C.) or more. It is generally highest at the beginning of the attack and then falls somewhat rapidly or slowly, although renewed rises may occur as the disease extends to new regions of the throat or as complications develop. Finally it reaches normal by lysis as the local symptoms improve. The *pulse* is generally weak and much more rapid than is in accord with the elevation of temperature, and the blood-pressure is low. This disproportion of the pulse-rate to the temperature is a very characteristic symptom. The appetite is lost, vomiting is common, constipation is the rule. The *urine* exhibits albumin in the majority of cases, although generally in not large amount, and epithelial cells and hyaline casts in many instances. The excretion of nitrogen is very greatly increased, as the result of the destruction of the tissue-cells (Howland).[1] The knee-jerks are often absent.

The *blood* shows a marked polymorphonuclear leucocytosis, which increases as the disease advances and diminishes during convalescence. The percentage of red blood-corpuscles and of hemoglobin is reduced in all severe cases. One of the most marked symptoms of the disease is the *prostration* which the diphtheria-toxin produces, frequently out of all proportion to the severity or duration of the other symptoms. Increasing anemia, loss of strength, and emaciation may be decided. Slight delirium may be present at the outset, and decided apathy, somnolence, or irritability later.

About the end of the 1st week, or frequently not until later, improvement may begin, indicated by softening and separation of the pseudomembrane and the gradual amelioration of symptoms. The disappearance of the pseudomembrane is often very rapid, especially under serum-treatment. Occasionally deep ulceration of the mucous membrane then becomes visible. Very often, however, the local and general symptoms continue a much longer time. The albuminuria generally persists until the end of the 2d or 3d week.

Convalescence is usually slow and debility long-continued. Weakness of the heart is a very prominent symptom even at this period and death may occur from this cause after the membrane has nearly or quite disappeared and the child has seemed to be out of danger.

Very frequently associated with faucial diphtheria is involvement of the nasopharynx. This decidedly modifies the course and duration of the disease. So also laryngeal diphtheria may result from a spread of the membrane from the fauces to the larynx. The symptoms of the two affections will be described separately.

Nasal Diphtheria.—Nasal diphtheria may be primary in the nose and either remain limited to this region or may spread to the nasopharynx, the throat, or the larynx. In other cases the nasal involvement is secondary to the affection in the fauces or nasopharynx.

Involvement of the nose and nasopharynx as a *secondary* affection is common and adds to the severity of the case and the discomfort of the patient. Respiration through the nose becomes difficult or impossible, and an offensive and very irritating discharge flows from the nostrils, causing excoriation and swelling of these and of the upper lip. Membrane may be seen lining the anterior nasal passages, and epistaxis is frequent. Constitutional symptoms in these cases are generally severe, and the duration of the attack is prolonged. The disease may extend to the ear, producing a purulent otitis media as a complication and resulting in a still longer continuance of the fever and of other symptoms.

Primary nasal diphtheria is commonest in infants, very frequently remains for some time undiscovered, and is a fertile source of contagion.

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[1] Arch. of Pediat., 1921, XXXVIII, 515.

When confined to this region either no general symptoms at all are noticed, or there is only slight fever and malaise. The child appears to have merely a cold in the head, but the character of the thin and very irritating nasal discharge is suspicious, especially if it is sanguinolent at times. Examination of the anterior nares will now often reveal the presence of pseudomembrane and of diphtheria-bacilli. These cases run a somewhat chronic course. In other instances the affection spreads to the nasopharynx and throat, and the constitutional and local symptoms are decided. Diphtheria *primary in the nasopharynx* is generally severe.

Nasal diphtheria, either primary or secondary, is a common form of involvement. In 1962 cases of diphtheria in the Boston City Hospital, Burrows[1] estimated that the nose exhibited the disease in about 40 per cent., and in 1200 cases of faucial diphtheria Rolleston[2] found involvement of the nose in 41.6 per cent. Occurring in the nose alone the latter investigator[3] discovered it in but 1.5 per cent. of 3000 patients.

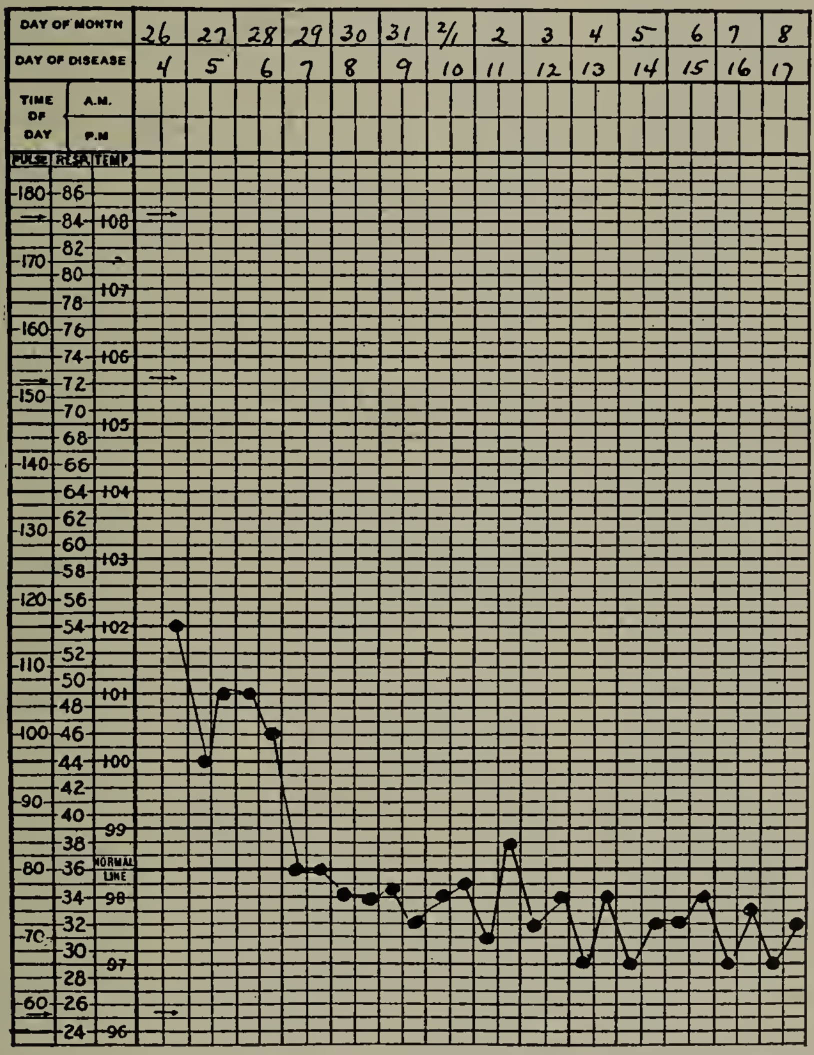

FIG. 135.—TONSILLAR AND LARYNGEAL DIPHTHERIA.

Joseph S., aged 3 years. Jan. 23, onset with fever and croup; Jan. 26, both tonsils covered, complete loss of voice, some stenosis; Jan. 27, less pseudo membrane and stenosis; Jan. 29, throat clean, a trifle croupy. Treatment, Jan. 26, 10,000 units; Jan. 27, 10,000 units; Jan. 28, 5000 units. Philadelphia Hospital for Contagious Diseases. *Courtesy of Dr. B. F. Royer.*

LARYNGEAL DIPHTHERIA.—This may properly be called *laryngo-tracheal diphtheria* since the pseudo-membrane may involve a portion of the trachea as well, or even extend into the bronchi. It is one of the most dangerous forms, a large proportion of the deaths in diphtheria depending upon this cause. The vast majority of cases of pseudo membranous laryngitis, or "true croup," are of a diphtheritic nature, and almost always secondary to lesions in the nose or the throat, although they often appear primary on account of these lesions being very slight, or transitory and overlooked. Diphtheria of the larynx develops about the 4th or 5th day of the disease. The first symptoms are hoarseness, a ringing cough and a very slightly noisy and prolonged respiration. Apart from the influence of any accompanying pharyngeal involvement there is little if any constitutional disturbance, and fever may or may not be present (Fig. 135). The symptoms are, in fact, local and are those of laryngeal stenosis. Generally they come on very rapidly, being fully developed by the 2d day of laryngeal involvement. The voice becomes very hoarse, whispering or absent; the cough peculiarly metallic, and dyspnea severe, both inspiration and expiration being prolonged and labored. The child has an extremely anxious expression; is restless and tossing; often sits upright in bed with head thrown back, and draws its breath only with difficulty and with the aid of all the accessory respiratory muscles and with spreading of the alæ of the nostrils. There is retraction of the epigastrium and the supraclavicular and suprasternal spaces and a lifting of the thorax high with every inspiration. Cyanosis is marked and the skin is clammy. Laryngoscopic examination reveals edema of the mucous and submucous tissues and the presence of pseudomembrane in the larynx; sometimes one predominating and sometimes the other.

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[1] Amer. Jour. Med. Sci., 1901, CXXI, 125.

[2] Report Metropolitan Asylums' Board, 1906.

[3] Brit. Jour. Child. Dis., 1917, XIV, 21.

The symptoms vary from time to time, now being much less marked; now increasing to the point of impending suffocation. The child then becomes livid, grasping at its throat or at the air with its hands, almost ceasing to breathe, and being apparently at the point of death. This change in the picture depends upon the varying degree of edema present. Occasionally relief is afforded by the coughing up of portions of pseudo membrane, but the deposit is soon reproduced and the threatening symptoms return. Before the days of antitoxin-treatment the majority of cases grew worse with varying rapidity, respiration becoming feeble, cyanosis increasing, the heart failing, a stuporous condition developing sometimes with convulsions, and death taking place after 24 to 48 hours or sometimes not for several days or a week. In other instances the child dies suddenly from lack of air in one of the suffocative attacks. In the cases which recover relief, at first partial and then permanent, may follow the coughing up of membrane, or the stenosis may disappear gradually.

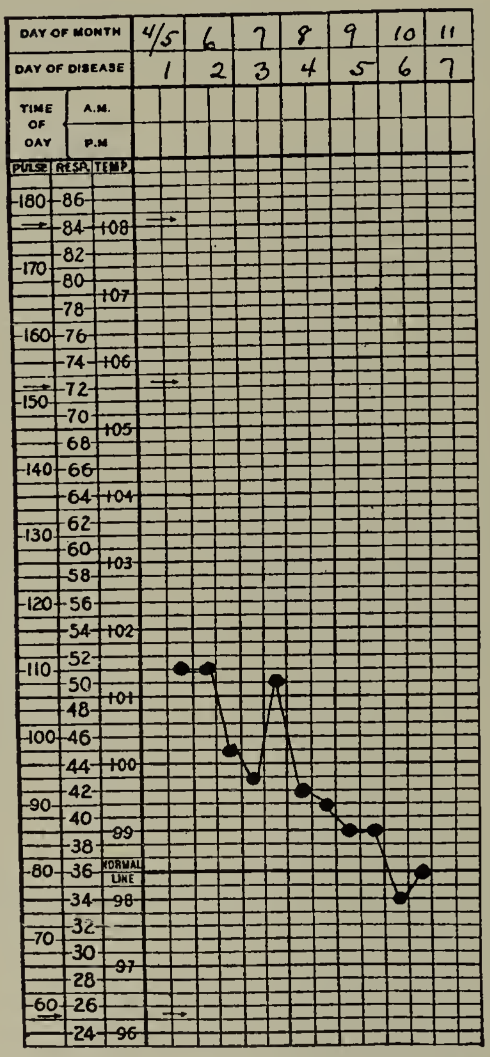

FIG. 136.—MILD TONSILLAR DIPHTHERIA.

J. A. S., aged 4 years. Deposit limited to tonsils, yellowish white, slight submaxillary swelling. General condition good. Slight albuminuria. 3000 units antitoxin given although symptoms already improving.

Laryngeal diphtheria is a frequent form of the disease. In the 1962 cases of the Boston City Hospital, reported by Burrows,[1] laryngeal stenosis occurred in 17 per cent.

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[1] *Loc. cit.*

Diphtheria may be classified also according to the character of the symptoms, the severity, and the degree of constitutional infection.

Mild Diphtheria.—Not infrequently the disease is of so mild a nature that it is either entirely overlooked or a positive diagnosis made possible only by bacteriological examination. Constitutional symptoms are nearly or entirely absent. As already stated primary nasal diphtheria is often of this nature, for unless membrane can be discovered—and it is not always present—the diagnosis is entirely bacteriological, since the profuse nasal discharge, although severe, could readily depend upon some other affection.

In other cases there are only the symptoms of a lacunar tonsillitis, the child suffering from sore throat and some degree of fever, but not from any constitutional depression (Fig. 136). Should an examination of the throat chance to be made some of the tonsillar crypts are found filled with secretion exactly as in ordinary lacunar tonsillitis. This deposit may disappear in 24 hours or less and leave the patient feeling completely well. Diphtheria-bacilli will, however, be found in the throat on bacteriological examination. Sometimes these cases persist as apparently simple lacunar tonsillitis for a few days and then develop membrane and run the ordinary course of diphtheria. In other cases a rapid recovery of the tonsillitis is followed in a few days by the development of laryngeal diphtheria, and in still others the diphtheritic lacunar tonsillitis is rather persistent and is accompanied by a degree of constitutional depression out of all proportion to the lesions seen in the throat, although it may perhaps not be sufficient to make the child desire to be in bed.

Catarrhal Diphtheria.—This term is applied to the mild cases in which there is an entire absence of membrane in either the nose or throat, the lesions being apparently simply those of a catarrhal inflammation. Constitutional symptoms are absent or insignificant. Diphtheria-bacilli are, however, found on bacteriological study, and histological examination shows the characteristic pathological changes in the epithelium of the mucous membrane. As in lacunar diphtheria the disease may give rise to laryngeal involvement.

Severe Faucial and Nasopharyngeal Diphtheria.—As laryngeal diphtheria is always a most dangerous affection, only the cases of severe faucial diphtheria are considered in this connection. Many of these are well called "*septic diphtheria,*" the general symptoms being those of profound sepsis, streptococci appearing to share with the diphtheria-bacilli in the poisoning of the patient. Others owe their severity entirely to the action of the diphtheria-toxin. These grave cases may begin as ordinary ones and then steadily grow worse, or the onset may be sudden and severe. The membrane is wide-spread; the tonsils and adjacent parts greatly swollen, often assuming a gangrenous appearance; the mucous membrane of the nose shares in the process and secretes an abundant offensive mucopurulent fluid. Hemorrhage may take place from the mouth and nose; the lips are swollen, fissured and sometimes exhibit membrane, as does occasionally the lining of the cheeks; the tongue is thick and dry; the eyes are sometimes involved in the process; the cervical lymphatic glands are greatly swollen and often the periglandular connective tissue also. Respiration through the nose is much obstructed. Swallowing may be difficult and food regurgitated through the nose. The face is peculiarly pale and more or less cyanotic, and the surface of the body pale or sometimes exhibiting petechiæ or a dusky redness; the extremities are cold; the pulse feeble and rapid, irregular or occasionally unusually slow. The heart shows evidence of increasing dilatation and loss of muscular tone, the sounds being weak and murmurs sometimes present. The temperature is variable, sometimes low, often high, and subject to marked fluctuations. Abdominal pain, vomiting, and diarrhea are common and prostration extreme. The mind may be clear or there may be restlessness or delirium or, oftener, profound apathy or stupor. The urine is frequently scanty and contains tube-casts and albumin in large amount; the spleen is enlarged.

The attack lasts a variable time. The development of the membrane reaches its height in 4 or 5 days, and no change in it may be apparent for several days more. At the end of about the 1st week it begins to disintegrate in favorable cases, and may disappear in a few days more. Sometimes, however, it persists for 2 weeks or longer. After its disappearance the mucous membrane is left raw-looking and sometimes ulcerated. The toxic symptoms reach their height with the maximum growth of the pseudomembrane, and although usually ameliorating as this disappears, by no means always do so, and in any event improve only slowly. A complicating laryngeal diphtheria is not likely to develop after the 1st week. The course is not usually much longer than a week in fatal cases, the child dying generally from increasing debility or bronchopneumonia; or more unexpectedly from uremic convulsions, heart-failure, or involvement of the larynx. Sometimes the fatal ending occurs only after improvement in local symptoms has commenced and convalescence seems assured; and may even be delayed until the 3d week of the disease or later.

Malignant diphtheria is a term applied to the very worst of the severe cases. The symptoms described are seen in a specially marked form. The pseudomembrane is wide-spread and the process sometimes necrotic or gangrenous; the toxemic symptoms are usually severe; the involvement of the cervical glands and cellular tissue great, and purpuric patches and hemorrhages from various mucous membranes may be present. Death occurs in 24 to 48 hours from the onset.

UNUSUAL LOCALIZATIONS.—Membrane may appear in more unusual regions, either primarily, or secondarily to diphtheria of the throat or nose. In very severe cases it may spread to the buccal cavity, lips, or the adjacent portion of the face. In 1 instance I observed it on the lining of the cheeks before it could be discovered in the throat, the primary lesion in this instance appearing to be on a circumcision-wound. Diphtheria of the conjunctiva is rare. The disease may be primary or secondary upon the penis or the scrotum, producing great swelling, or upon the vagina and vulva. In the last the genitals are much infiltrated and a thin, irritating discharge excoriates the neighboring skin. Any cutaneous wound, especially that of tracheotomy, or the unhealed umbilicus or even an eczematous area may be attacked by the diphtheritic process, especially in much debilitated subjects. Occurring primarily on the skin it is rare, Billings[1] finding only 27 recorded cases, and adding 2 of his own. Exceptionally it may be primary in the bronchi and spread thence to the larynx. Secondary involvement of the trachea and bronchi is common (Fig. 137); that of the tongue, esophagus, stomach and intestines rare. On these unusual situations toxin-absorption may not be great, and the condition may be a chronic one. In some instances, however, especially when an open wound is affected, the symptoms may be acute and severe.

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[1] Annals of Surg., 1915, LXII, 343.

Complications and Sequels.—One of the most frequent complications of diphtheria is *bronchopneumonia*. This is particularly liable to occur when the larynx is involved; in severe septic cases, and in infancy. It depends not only upon the presence of the diphtheria-bacillus, but upon associated microörganisms, especially the streptococcus and the pneumococcus. It may develop at any time during the course of the disease and may be found at autopsy in over ½ of the cases. Croupous pneumonia is only an accidental complication.

Cardiac failure is one of the most dreaded of complications or sequels. It results from degeneration of the cardiac muscles or ganglia, or an involvement of the pneumogastric nerve. Alteration of the bundle of His may or not be present. Farr[1] has collected the published instances of diphtheritic heart-block in children. In other cases cardiac failure is produced by the formation of a heart-clot. The mechanism of this does not seem to be the same in every instance and the subject has not been conclusively settled. McCulloch[2] has studied the heart in diphtheria with the electrocardiogram, and has found evidence of damage both to the intrinsic conducting system and to the heart-muscle. Marvin's[3] studies indicate that myocarditis is the important lesion. Heart-failure may occur at any time during the course of the disease; even upon the 1st day in malignant cases; but is witnessed most frequently as a sequel in the 3d or 4th week during convalescence, and generally after severe attacks of diphtheria. If developing during the attack it takes place oftenest while the disease is at its height, or after the membrane has nearly or quite disappeared. The evidences of heart-failure may come on gradually, lasting 3 or 4 days or less, and consist in abdominal pain; obstinate vomiting; dyspnea; weak, compressible pulse which is usually rapid and irregular, sometimes slow; feeble heart-tones sometimes with faint "accidental" murmurs or a gallop-rhythm, enlargement of the liver, and the percussion-evidences of cardiac dilatation. The surface of the body is pale and the tissues may be dropsical. In other cases heart-failure is abrupt, the child dying suddenly when raised or when moving itself in bed, when emptying the bowels, or

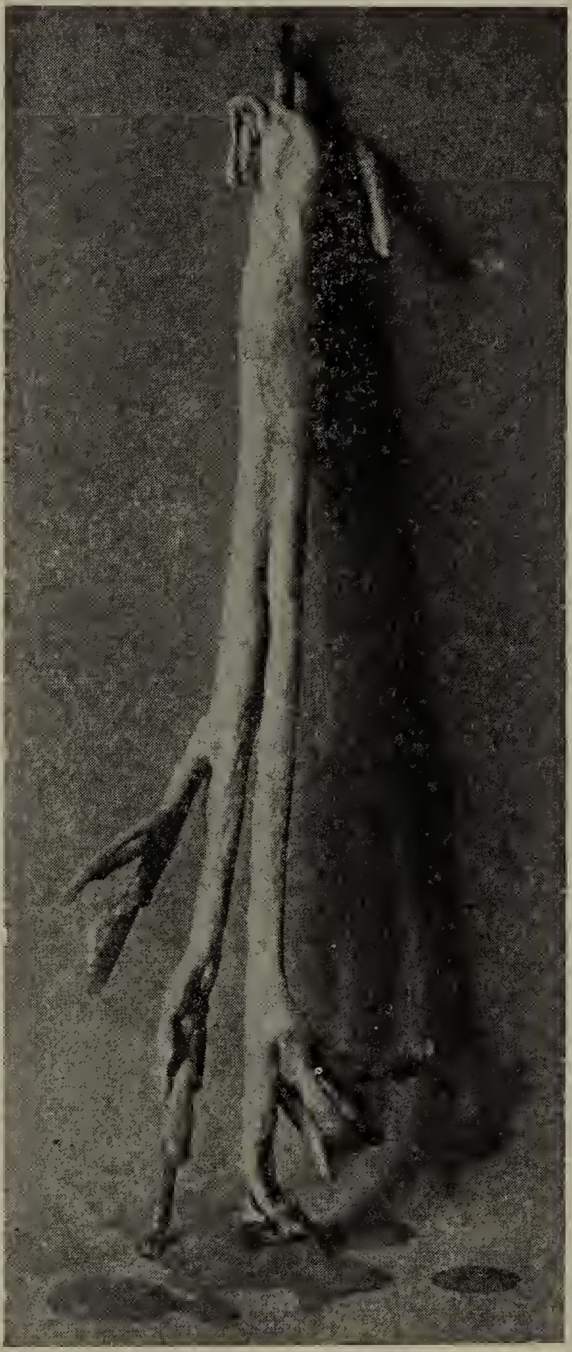

FIG. 137.—TRACHEO-BRONCHIAL CASTS IN DIPHTHERIA.

Boy of 5 years with laryngeal diphtheria, in the Philadelphia Hospital for Contagious Diseases. Intubation no benefit. Tracheotomy performed and casts removed with forceps through the opening. *Courtesy of Dr. N. A Christensen.*

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[1] Penna. Med. Jour., 1920, XXIII, 633.
[2] Amer. Jour. Dis. Child., 1920, XX, 89.
[3] Amer. Jour. Dis. Child., 1925, XXIX, 433.

undergoing an examination of the throat, or, during convalescence, when walking about the room or even after undue excitement.

Sometimes repeated attacks of impending heart-failure occur extending in all over a considerable period, to be followed finally by recovery or by a fatal ending. Accompanying this condition there is generally emaciation and loss of appetite and strength. Moderate disturbance of the heart, as shown by irregular and rapid pulse, faintness, and shortness of breath, is very common in diphtheria. White and Smith[1] found cardiac symptoms in 878 out of 946 cases of diphtheria. Occasionally permanent valvular lesions persist as sequels Bullowa[2] concludes from the study of a large number of cases that the oculo-cardiac reflex is absent in severe diphtheria, and that this denotes the existence of danger from the side of the cardiac action.

Nephritis is a frequent complication of diphtheria, evidenced by the occurrence of moderate albuminuria in from ⅓ to ⅔ of all cases, and in nearly every case at all severe. Baginsky[3] found albuminuria 417 times in 993 cases of diphtheria, and in 256 of these there were morphological elements present also. It depends upon degeneration of the renal epithelium through the action of the toxin. The urine may be somewhat diminished in amount and epithelial cells, leucocytes, and tube-casts, generally hyaline, are present, but seldom any blood. Dropsy is absent. Recovery is generally rapid. Nephritis is seen usually in the 1st or 2d week, and is, as a rule, much less dangerous and persistent than that connected with scarlet fever. In some severe instances a sudden, acute nephritis may develop, characterized by great diminution in the amount of urine, a high degree of albuminuria and epithelial casts, yet only exceptionally by dropsy and symptoms of uremia. Complete anuria is sometimes observed. I have known it to last at least 3 days before the fatal issue. Chronic nephritis is an unusual sequel.

Diphtheritic Paralysis.—Observed as a complication during the attack diphtheritic neuritis manifests itself in the production of heart-failure, and occasionally, in severe cases, of an early palatal paralysis. Occurring in some form, as a complication or a sequel, it was seen in 20.7 per cent. of 2300 cases of diphtheria observed by Rolleston.[4] As a sequel it is a cause of cardiac failure, but is especially seen in the "*postdiphtheritic paralysis*" affecting various regions of the body. It is observed oftenest after severe attacks in those past the period of infancy, but to this there are numerous exceptions, and not infrequently it occurs when the diphtheritic affection of the throat has been completely overlooked. The most frequent and usually the first seat is the soft palate, which exhibits paralysis most frequently 3 or 4 weeks or more after the onset of the attack of diphtheria. Baginsky[5] found this symptom 68 times in 993 cases of diphtheria. The speech becomes nasal and fluids pass into the nose when attempts at swallowing are made. The palate is relaxed and does not move with phonation and the palatal reflex is abolished. With this paralysis is generally associated a loss of the patellar reflex, even in cases where no sign of neuritis is discoverable beyond the palate. Indeed the patellar reflex may be abolished without any palatal involvement. Early in the attack the paralysis may extend in some cases from the palate to the pharyngeal and laryngeal muscles, with the result that liquids readily enter the trachea when swallowing is attempted, and that respiration may be interfered with by the laryngeal involvement.

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[1] Bost. Med. and Surg. Jour., 1904, CLI, 433.
[2] Arch. of Pediat., 1923, XL, 306.
[3] Nothnagel, Spec. Path. u. Therap., II, 226.
[4] Arch. of Pediat., 1913, XXX, 335.
[5] *Loc. cit.*, 213.

Next in frequency and in order of development, although much less common, is paralysis of the ocular muscles, producing strabismus, loss of accommodation, dilatation of the pupils and ptosis or sometimes other evidences of neuritis of the 3rd nerve. The nerves of the face may exceptionally be attacked, and not uncommonly those of other parts of the body, especially the lower extremities. In the latter event there appear paresthesia, pain, and difficulty in walking owing to lack of power or of coördination. The condition is that seen in multiple neuritis of any sort and the electrical reactions are the same. Less often the arms share in the ataxia and loss of power, and occasionally the head drops forward from involvement of the muscles supporting it. In bad cases the child may be almost powerless and respiration may become difficult through paralysis of the abdominal and thoracic muscles and of the diaphragm. Mixsell and Giddings[1] report 8 instances of the last in 4259 cases of diphtheria, and Rolleston[2] 16 instances in 864 cases of paralysis. Death in these severe cases may result from inability to swallow, interference with respiration, or the cardiac paralysis already referred to.

In the majority of cases, however, the paralysis affects only the palate and the patellar reflexes and often the ocular muscles as well, and recovery begins in about 2 weeks and advances rapidly. When the affection is more wide-spread, convalescence is much slower. Rarely there occurs a cerebral paralysis of the hemiplegic type. Dynkin[3] was able to collect 72 reported cases, and Rolleston[4] 80 cases.

Digestive disturbances are frequently seen in severe cases. The vomiting which attends heart-failure has already been referred to. Diarrhea may occur, dependent upon inflammation other than of a pseudomembranous nature.

Prolonged anemia and *debility* are frequent sequels after severe cases and to some extent after all. Chronic *rhinitis* is a common sequel and *otitis media* is often seen, although less frequently than after other infectious diseases. It arises generally by infection through the Eustachian tube. Rolleston[5] reported it in 4.10 per cent. of 5076 cases in the Metropolitan Asylums Board's Hospitals.

Various *cutaneous eruptions* may occur, among these being a diffuse multiform erythema. Herpes is sometimes seen (4 per cent. Rolleston).[6] Purpuric eruptions may be observed in malignant cases. Various urticarial and erythematous eruptions develop after the use of antitoxin. (See Treatment.)

Other infectious diseases may be associated with diphtheria, prominent among these being scarlet fever and measles. Chicken-pox, smallpox, whooping cough and typhoid fever may occur in combination with diphtheria.

Among more unusual complications may be mentioned pleurisy, endocarditis, pericarditis, cutaneous emphysema—the result of necrosis in the pharynx—arthritis, chorea, meningitis, and thrombosis or embolism in the brain or extremities, in the former localization hemiplegia perhaps resulting; in the latter gangrene sometimes developing. Ransome and

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[1] Jour. Amer. Med. Assoc., 1921, LXXVII, 590.

[2] Arch. of Pediat., XXX, 340.

[3] Jahrb. f. Kinderheilk., 1913, LXXVIII, Ergänzungsh., 267.

[4] Clin. Jour., 1913, XLII, 12.

[5] Brit. Jour. Child. Dis., 1915, XII, 18.

[6] Brit. Jour. Dermatol., 1907, XIX, 375.

Corner[1] could collect but 9 cases of gangrene from medical literature, including 1 of their own; and Robbins[2] 25 occurring in children. I have seen it in 1 unreported instance, and others have been published by Gunson[3] and Jacobs.[4]

Relapse.—This is not as rare as often supposed, Rolleston[5] finding it in about 1.5 per cent. of 2560 cases. It consists in the reappearance of the symptoms of the disease within a few weeks after the onset of the first attack and before the germs have left the system. The occurrence of measles during convalescence from diphtheria is liable to be followed by a re-development of the diphtheritic process.

Recurrence.—Recurrence is frequently seen, protection given by one attack appearing to continue but a very short time, probably at the most from 1 to 2 years, after which there is no certain immunity, and some individuals even seem predisposed to repeated attacks. Yet recurrences as compared with the total number of cases of diphtheria are not very common and are prone to be less severe than the first attack. It is uncertain to what extent this depends on an acquired natural immunity which increases as the individual grows older. (See p. 516.)

Prognosis.—The mortality of diphtheria, always high, has varied much in different periods, even before the introduction of antitoxin-treatment. Epidemics have differed greatly in their severity, depending probably on varying degrees of virulence of the germ. Since the last portion of the 19th century the disease as a whole has certainly become less serious, although this diminishing severity has been affected in part by the employment of antitoxin, and in part by the application of bacteriological diagnosis, by which very many mild cases are recognized which would otherwise have passed undetected.

Yet the prognosis in individual cases is always most uncertain. Unfavorable symptoms may readily develop in cases which at first appeared to be mild, and unexpected complications and sequels may add very greatly to the danger. The favorable influence of the employment of antitoxin is certainly very great. This has been so uniform an experience that it no longer demands proof. A few statistics may, however, be given by way of illustration. Burrows[6] found that for 15 years prior to the introduction of antitoxin the mortality in Boston had been 30.8 per cent., whereas in 1962 cases treated in the Boston City Hospital in a single year after this period, the mortality had fallen to 12.23 per cent., or, if those moribund on admission were deducted, 9 per cent. The statistics of the New York Board of Health (Northrup)[7] showed a mortality of 34.9 per cent. in 27,210 cases treated without antitoxin, and only 15 per cent. in 56,425 cases treated with it. The report of the Metropolitan Asylums Board's Hospitals for London (Herringham)[8] gave a mortality of 30.25 per cent. in 11,704 cases before the introduction of antitoxin, and from 22.5 to 9.29 per cent. in the years following this. Most of the published statistics are, to a large extent, from hospital practice, and are virtually alike in the diminished mortality shown. In purely private practice the results with antitoxin are still better. In 1610 such cases in St. Louis, collected by Zahorsky,[1] the mortality was but 1.5 per cent.

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[1] Lancet, 1911, I, 94.
[2] Med. Rec., 1918, XCIV, 620.
[3] Brit. Jour. Child. Dis., 1916, XIII, 237.
[4] South. Med. Jour., 1923, XVI, 602.
[5] Brit. Jour. Child. Dis., 1907, IV, 332.
[6] *Loc. cit.*, 125.
[7] Nothnagel's Encyclopedia, American Ed., Diphtheria, 143.
[8] Allbutt and Rolleston's Syst. of Med., I, 1030.

The favorable effects of antitoxin-treatment have been especially marked in laryngeal cases. According to the investigations of the American Pediatric Society,[2] before the employment of antitoxin about 73 per cent. of cases of laryngeal diphtheria died, while in 1704 cases treated with antitoxin in private practice the mortality was but 21.12 per cent. In 15,148 laryngeal cases occurring in the practice of New York City physicians, as quoted by Biggs and Guerard,[3] all treated with antitoxin, the mortality was but 16.6 per cent. Moreover the employment of antitoxin certainly appears to have diminished greatly the necessity of operative treatment. The report of the American Pediatric Society showed that about 60 per cent. of the laryngeal cases thus treated did not need intubation, while previously about 90 per cent. required it. The mortality also of cases requiring intubation has diminished under the influence of antitoxin. In 639 such cases without antitoxin reported by McCollom[4] there was a mortality of 82.49 per cent., while 1478 intubated cases receiving antitoxin had a mortality of only 41.4 per cent. Siegert's[5] 37,000 collected cases of tracheotomy and intubation showed a mortality of 60.55 per cent. before the employment of antitoxin and only 35.70 per cent. after this period.

The *promptness* with which the serum-treatment is commenced is most important. This is dependent upon the remarkable affinity of the diphtheria-toxin for the tissue-cells; the serum being able to neutralize the toxin only before it is thus absorbed. Its value, then, is preventive of further damage; not curative of that which has already taken place. In the first report of the American Pediatric Society[6] the mortality in cases injected on the 1st day of the disease equalled 4.9 per cent. This increased steadily to 22.9 per cent. in cases first treated on the 4th day and to 38.9 per cent. after the 4th day. Biggs and Guerard[7] published the following table illustrative of this fact.

TABLE 78.—COMPARISON OF DATE OF TREATMENT AND MORTALITY

Date of treatment	Cases	Mortality, per cent.
1st day of the disease	1415	3.5
2d day of the disease	2640	8.0
3d day of the disease	2340	12.8
4th day of the disease	1458	23.6
5th day or later	1912	35.0

Various factors other than treatment influence the mortality. *Age* is prominent among these. The disease is much more fatal in infancy, largely on account of the danger of laryngeal involvement and the development of bronchopneumonia, and becomes progressively less dangerous as age advances. New-born infants are especially likely to succumb. The great majority of deaths are in subjects less than 5 years old. In

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[1] Med. News, 1903, LXXXIII, 1085.
[2] Med. News, 1897, LXX, 632.
[3] Med. News, 1896, LXIX, 677.
[4] Royer, Proceedings Phila. County Med. Soc., 1905, XXVI, 80.
[5] Jahrb. f. Kinderheilk., 1900, LII, 56.
[6] Transac. Amer. Pediat. Soc., 1896, VIII, 21.
[7] *Loc. cit.*, 728.

Burrows[1] 1962 cases, all treated with antitoxin, the mortality was divided as follows: 1st year 40.40 per cent.; 2d year 33.9 per cent.; 3d year 23 per cent.; 4th year 15.60 per cent.; 5th year 14.60 per cent. The total mortality from birth to 5 years was 21.30 per cent., from 5 to 10 years 8.40 per cent., and from 10 to 15 years 3.10 per cent. In 17,889 fatal cases in the statistics of the New York Board of Health[2] 14,554 (81.36 per cent.) were under 5 years of age. Favorable *social* and *hygienic conditions* improve the prognosis decidedly. The *situation of the membrane* and the rapidity of its extension influence the prognosis in individual cases. An abundant and rapidly spreading or deeply penetrating membrane in the throat makes the prognosis grave. Involvement of the larynx, as already stated, increases the mortality greatly. Primary nasal diphtheria is generally of little danger to the patient, although sometimes serious by its extension to the larynx. In diphtheria of the nasopharynx, on the other hand, the prognosis is unfavorable. The development of marked *septic symptoms* is an unfavorable prognostic indication as is also evidence of *cardiac weakness*, although even cases of heart-block may occasionally recover. The Boston City Hospital Cases showed that the mortality was often directly in proportion to the rapidity of the pulse. The development of *hemorrhagic conditions* makes the prognosis extremely serious. The absence of an increase of the polymorphonuclear cells in the blood has been considered of bad import, although this is not universally admitted.

Among serious complications are bronchopneumonia and an unusual degree of albuminuria, of anemia, or of lymphatic enlargement. Post-diphtheritic paralysis generally results favorably if the heart and respiratory muscles escape. Ronaldson's[3] studies confirm Rolleston's[4] earlier observations, that the more marked the antitoxin-reaction the better is the prognosis. This applies particularly to the serum-rash, a well-marked secondary rash being a very good prognostic sign. The combination of measles with diphtheria or the development of one after the other greatly increases the gravity of the case. Scarlet fever occurring as a complication is unfavorable and the case may be readily fatal. If the order is reversed, diphtheria being the complication, the prognosis is not so often affected. The association of diphtheria and typhoid fever is dangerous.

Diagnosis. **Clinical.**—Even without bacteriological examination the diagnosis of diphtheria is usually easy in typical and well-developed cases. Early in the disease or in atypical attacks it may be a matter of great difficulty. It rests, in general, in the case of tonsillar diphtheria upon the rapid development of pseudomembrane which is removed only with difficulty and which leaves a bleeding surface beneath; the reforming of the membrane after removal; its tendency to spread beyond the tonsils; constitutional depression out of proportion to the local symptoms; the frequent presence of albuminuria, and of glandular enlargement in the neck, and the common occurrence of paralysis as a sequel. The mode of onset and the temperature-curve are too variable to be of much diagnostic assistance.

Mild attacks of diphtheria are most liable to be confounded with *follicular tonsillitis* especially when the separate foci of secretion in the latter disease fuse and cover the tonsil. The onset of this condition is generally more sudden, the fever higher; the throat feels sorer and is of a deeper red; the early swelling greater; the secretion is easily removed, and there is no spreading beyond the tonsils. The patient often has the history, too, of having been susceptible to repeated similar attacks. It is impossible, however, to make a positive diagnosis between the ordinary follicular tonsillitis and diphtheritic folliculitis without a bacteriological examination. Severe and even fatal *streptococcic inflammation of the tonsils* may occur, and can be distinguished from diphtheria only by bacteriological examination. Such cases are seen, for instance, in scarlet fever. Similarly a severe pseudomembranous inflammation is sometimes dependent upon *pneumococcic involvement.*

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[1] *Loc. cit.*

[2] Northrup, *loc. cit.*, p. 24.

[3] Brit. Jour. Child. Dis., 1923, XX,129.

[4] Practitioner, 1905, LXXIV, 660.

Primary nasal or nasopharyngeal diphtheria is often unrecognized because unsuspected. It is especially in infants and young children, when free nasal and often blood-stained discharge is combined with excoriation of the lip and nostrils and perhaps the presence of fever, that the secretion should be studied bacteriologically and an examination for membrane made.

Laryngeal diphtheria must be distinguished from stenosis depending upon other laryngeal conditions. The persistence of stenosis by day as well as by night usually indicates the existence of something more than *false croup,* yet not necessarily so. The further differential diagnosis is considered under Respiratory Diseases. The severe laryngeal stenosis which sometimes accompanies measles may or may not be diphtheritic in nature. It may be dependent upon a subglottic laryngitis. Only a laryngoscopic examination, when this can be made, or a bacteriological study can finally determine the question.

The diagnosis is sometimes difficult between diphtheria and *scarlet fever,* but only in anomalous cases. The former disease may exhibit a scarlatiniform erythema, while in the latter the eruption may be absent, undiscovered, uncharacteristic, or late in appearing. The onset of scarlet fever, however, is usually more abrupt and the inflammation of the throat at the outset more severe. Only a bacteriological study can determine the diagnosis in some instances. The combination of the two diseases may be especially difficult to recognize. Here the sequence of symptoms is the best guide. *Thrush, aphthæ* and *ulcerative stomatitis* offer little practical difficulty in diagnosis. The first two are different in situation and appearance, and the last could suggest diphtheria only when this is limited to the oral cavity; an occurrence of great rarity. *Vincent's angina* may resemble diphtheria early in the attack, and a bacteriological examination may be required to distinguish them. The deposit in Vincent's angina is more liable to involve the tongue, cheeks, and gums. When on the tonsils it is usually confined to one side, is slower in developing, more necrotic than pseudomembranous, and tends to produce a deeper and more punched-out ulcer.

Bacteriological.—Although, therefore, the clinical diagnosis of diphtheria can be made with positiveness in many cases of the disease, the bacteriological diagnosis, as already indicated, is always of value and often indispensable. In all suspicious cases cultures should be taken as early as possible. The secretion is obtained by rubbing the affected area, or the posterior portion of the pharynx when the larynx is involved, firmly with a swab of sterilized cotton or a platinum wire loop, and then transferring the germs in the same manner to the blood-serum culture-medium in a test-tube. After about 6 hours, or with more certainty after

12 hours, this may be examined for the presence of the diphtheria-bacilli. The culture should not be taken immediately after an antiseptic application has been made to the suspected region. It can also not be depended upon if made late in the course of the disease. Since the surface of a thick membrane may fail to give a positive culture, inasmuch as the bacilli here are dead, a lower layer should be reached if possible. This may yield positive results. A single negative culture is not sufficient in suspicious cases. A test of the virulence of the germs is sometimes necessary, effected by injecting them into guinea-pigs. Sometimes the diagnosis of diphtheria can be made by the immediate staining of smear-preparations from the throat or nose. The failure to find the germs under these conditions is not, however, a proof that the disease is not diphtheria.

Regarding the diagnostic value of the diphtheria-bacillus, while this is undoubtedly very great, reliance should not be placed on it alone. A case which is clinically diphtheria should be treated as such in spite of the failure to find the specific germ. On the other hand the mere presence of diphtheria-bacilli in healthy throats does not constitute diphtheria (see p. 500), and there is no reason why the same accidental presence should not be found in persons whose throats exhibit a catarrhal or even a lacunar tonsillitis. Nevertheless, the combination of diphtheria-bacilli with catarrhal pharyngitis or rhinitis generally indicates the existence of diphtheria and the provisional diagnosis of this disease should be made.

As to the diphtheroid bacilli other than the virulent Klebs-Löffler germ, there is much difference of opinion. There may be found in the throat an organism which is in reality the diphtheria-bacillus although non-virulent to guinea-pigs. There is also described the pseudo-diphtheria bacillus of Hofmann-Wellenhof already referred to (p. 500). Neither germ is capable of producing diphtheria, and neither is common in cases which are apparently clinically this disease; but whether they are in reality but variants of the diphtheria-bacillus, capable under favorable circumstances of again becoming virulent, is not certainly determined. The difficulty in making a diagnosis of diphtheria solely upon the cultural findings is therefore great. Van Riemsdijk[1] found bacteria resembling diphtheria-bacilli in 50 per cent. of children examined in a region where there had been no diphtheria for 10 years, and Kolmer[2] reported diphtheria-bacilli, although avirulent, in 40 per cent. of cultures from the penis of 100 healthy boys.

Treatment. PROPHYLAXIS. **Natural Immunity.**—Bearing closely upon the necessity of immunization is the question of natural immunity. It had been recognized that infants in the early months of life were less susceptible to diphtheria than after this period and during early childhood; and that through later childhood there was a slowly increasing return of immunity. The investigations of Schick[3] have established these facts upon a scientific basis. He found that the intracutaneous injection of $\frac{1}{50}$ of the minimum dose of toxin lethal for a guinea-pig, diluted with 0.05 to 0.2 c.c. of normal salt solution, will produce in non-immune persons an erythema, while in those immune there is no reaction whatever. In making the test a small, accurately graduated syringe is employed, with a very fine special needle; the skin pinched into a fold; and the needle inserted into the *skin*, not beneath it. If no antitoxin is present in the patient's circulation, a red, slightly edematous spot of erythema,

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[1] Nederl. Tijdsch. v. Geneesk., 1914, LVIII, 1066

[2] Arch. of Pediat., 1912, XXIX, 94.

[3] Münch. med. Wochenschr., 1913, LX, 2608.

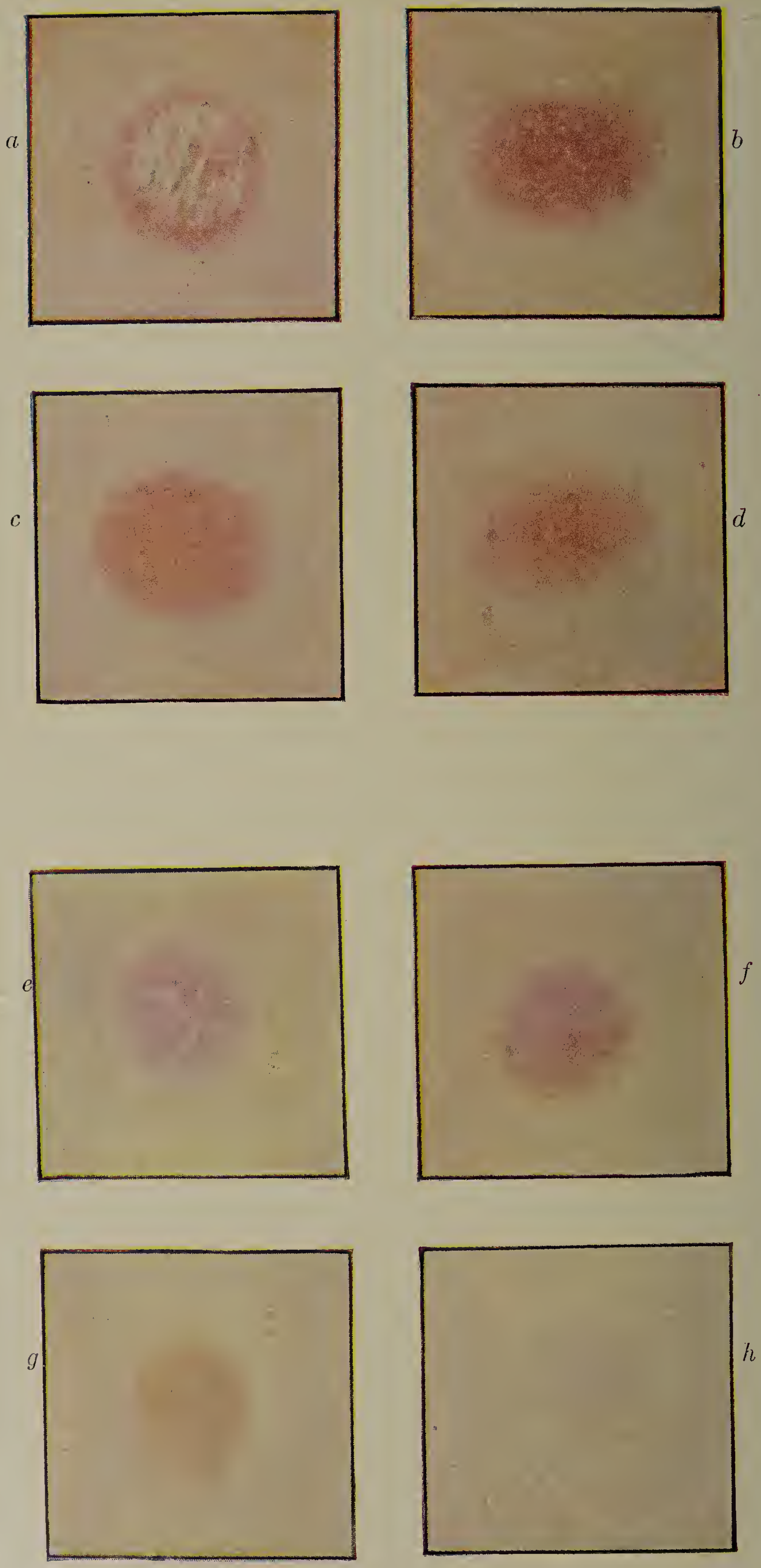

Fig. 138.—The Schick Reaction in Diphtheria.

(*a*) to (*d*) Typical positive reactions 48 hours after test: (*a*) strongly positive reaction, with vesiculation of the surface layer of the epithelium, which is seen occasionally in individuals who have practically no antitoxin; (*b*) and (*c*) positive reactions; (*d*) a moderately positive reaction; (*e*) fading reaction 1 week after test; show redness, scaling and beginning pigmentation; (*f*) after 2 weeks; (*g*) after 3 weeks; (*h*) faint pigmentation after 4 weeks. (*Zingher, American Journal of Diseases of Children, April,* 1916.)

0.5 to 2 cm. in diameter, with a brownish tinge, appears within 24 to 48 hours (Fig. 138). It begins to disappear within from 7 to 10 days, leaving a brownish pigmented area with superficial desquamation, the pigmentation persisting for months. Pseudo-reactions appear earlier, are less sharply defined, more infiltrated and disappear in 48 hours, and there is no subsequent pigmentation. The value of this test has been confirmed by other investigators, among them in the United States being Moody,[1] Park, Zingher and Serota,[2] Kolmer and Moshage,[3] Shaw and Youland,[4] Bauer[5] and others. An excellent bibliography is given by Moffett.[6] A comparison of the statistics given by these writers, the figures being approximations only, shows that a positive reaction, *i.e.*, an absence of immunity, was present in the new-born in 7 per cent. (Schick); in the 1st year in 35 to 45 per cent.; from 1 to 2 years in 55 to 60 per cent.; from 2 to 5 years in about 66 per cent.; from 6 to 8 years in 35 to 45 per cent.; and from 8 to 15 years in 25 to 30 per cent.

The most complete study on the susceptibility to diphtheria as determined by the Schick test is that by Park, Zingher and their associates no over 150,000 children in New York City. The following table[7] will show the results of their work:

Table 79

Age, years	Total no. of children tested	Schick-positive reactions	Schick-negative reactions	Percentage of Schick-positive reactions
5–6	4,809	2,902	1,907	60.3
6–7	13,754	6,944	6,810	50.4
7–8	16,180	7,042	9,138	43.5
8–9	17,126	6,278	10,848	36.6
9–10	18,065	5,825	12,240	32.2
10–11	18,057	5,308	12,749	29.3
11–12	17,994	5,075	12,919	28.2
12–13	16,258	4,327	11,931	26.6
13–14	14,138	3,271	10,867	23.1
14–15	9,650	1,906	7,744	19.7
15–16	4,861	869	3,992	17.8
16–17	369	68	301	18.4
Total	151,261	49,815	101,446	32.9

The practical value of the Schick test is great, in that it enables us to determine what subjects are already immune. Those who give a negative reaction do not need antitoxin or toxin-antitoxin injections. This is a matter of moment from an economical point of view when considerable numbers of patients are to be immunized, and is important, too, when an anaphylactic reaction is feared in those who have previously received antitoxin. Since about 40 to 50 per cent. of children from 1 to 15 years are naturally immune (Kolmer and Moshage), it is evident that artificial immunization is required in only ½ of the children who have been exposed. In the first 6 months of life there appears to be in most cases a natural immunity. Kuttner and Ratner[8] have shown that the Schick test may not be reliable in the new-born infant. They suggest performing the test upon the mother, a negative reaction indicating that the baby also is immune, since there is placental transmission of diphtheria antitoxin.

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[1] Jour. Amer. Med. Assoc., 1915, LXIV, 1206.
[2] *Loc. cit.* Also Arch. of Pediat., 1914, XXXI, 481.
[3] Amer. Jour. Dis. Child., 1915, IX, 189.
[4] Trans. Amer. Pediat. Soc., 1916, XXVIII, 329.
[5] Penna. Med. Jour., 1921, XXIV, 471.
[6] Ann. Clin. Med., 1923, I, 372.
[7] Amer. Jour. Dis. Child., 1923, XXV, 392.
[8] Amer. Jour. Dis. Child., 1923, XXV, 413.

Immunization.—Upon the development of a case of diphtheria, all susceptible members of the household, proven by the Schick test to be so, should receive an immunizing injection of diphtheria-antitoxin. When for any reason the test cannot be carried out, certainly all the children of the family should be immunized, and it is no doubt safer to treat the older members in the same way. The dosage employed should be from 500 to 1000 units, the latter amount being used at all ages except the 1st year of life. The protection afforded is very complete yet but of short duration; not over 4 weeks and often not much longer than 10 days. Consequently children in hospital-wards should have the immunization repeated every 3 to 4 weeks, if a repetition of the Schick test gives a positive result.

In the effort to produce a more lasting immunity v. Behring[1] has urged repeated subcutaneous injections of a mixture of toxin and antitoxin, as first suggested for use in children by Theobald Smith[2] in 1909. It is sometimes of advantage to combine with the toxin-antitoxin mixture a vaccine of dead diphtheria-bacilli. Park and Zingher[3] found that v. Behring's method of immunization greatly increased the amount of antitoxin in the blood of persons already immune; while in the non-immune the treatment combined with the vaccine produces sooner or later an immunity in 40 out of 50 individuals treated. The use of toxin-antitoxin mixtures for the prevention of diphtheria is far beyond the experimental stage, and it seems more than probable that its universal employment would wipe out the disease, as has been done so largely for small-pox by vaccination. Continued studies by Park,[4] Zingher[5] and many others have demonstrated that about 85 per cent. of susceptible children will produce sufficient antitoxin, after 3 hypodermic injections of toxin-antitoxin spaced 1 to 2 weeks apart, to develop a negative Schick test within from 3 to 6 months. Marked, if not absolute, protection against diphtheria will be present, which will last, in at least 90 per cent. of the children, for more than 6 years and probably for the remainder of life. Infants under 6 months of age, because of the high percentage of natural immunity and also because of the possibility of more severe reaction, should not be injected. From 6 months to 6 years of age the toxin-antitoxin may be given without the preliminary Schick test if so desired, inasmuch as at this time of life it is usual to find a positive reaction.

Three to 6 months after the toxin-antitoxin has been injected a second Schick test should be performed, and if it proves positive a second series of toxin-antitoxin injections should be administered. Zingher[6] observed no instances of anaphylactic shock during such second series. A child who develops clinical diphtheria within 6 months after the toxin-antitoxin injections should be treated with antitoxin in the usual way, just as though no attempt had been made to establish immunity. A certain amount of local and general reaction with fever and body-pains may follow the toxin-antitoxin injections. This is less likely to occur in children than in adults. Toxin-antitoxin is an unstable product and it may become quite toxic if frozen, because prolonged freezing dissociates the combination and lessens the antitoxic strength.[1] It must be carefully prepared in order that not too much toxin is present; otherwise the reaction following its injection may be severe. The presence of horse-serum in the mixture renders its use inadvisable in asthma or in other conditions in which anaphylactic or allergic reactions may be feared. A number of modifications of toxin-antitoxin treatment have been recommended. Larson and Eder[2] have employed a 2 per cent. sodium ricinoleate solution to detoxify the toxin, thus avoiding the employment of antitoxin with its horse-serum content.

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[1] Deutsch. med. Wochenschr., 1913, XXXIX, 873.

[2] Jour. Exper. Med., 1909, XI, 241.

[3] Bureau of Laboratories, City of New York, 1914–15, VIII, 104.

[4] Jour. Amer. Med. Assoc., 1922, LXXIX, 1584.

[5] Jour. Amer. Med. Assoc., 1921, LXXVII, 835; 1922, LXXVIII, 1945; 1923, LXXX, 456.

[6] New York State Jour. of Med., 1923, XXIII, 6.

Quarantine.—Persons suffering from diphtheria should be isolated until at least 2 successive negative cultures show that the bacilli have disappeared from the mucous membranes. Persons merely suspected of having the disease ought to be quarantined until a bacteriological study can be made and the diagnosis determined positively. Other children in the family should be kept from school, and, if possible, removed from the house; and should not be allowed to mingle with others until bacteriological examination shows that no diphtheria-bacilli are present on the mucous membranes. The nurse or members of the family in constant attendance upon the patient should employ disinfectant gargles and sprays, and should refrain from association with others. The Schick test may be used to determine the presence of immunity in those persons in contact with the patient. When cultures for any reason cannot be made—and this is now exceptional—the quarantine of the patient should continue for at least 3 weeks after the mucous membranes are free from deposit. This ensures the disappearance of the bacilli in the very large majority of instances, since bacteriological examination shows that in probably 80 per cent. of cases there are none of these germs to be found in 1 week, and in 90 per cent. in 2 weeks, after the pseudomembrane has left the throat. In 882 cases recorded in the Department of Health of Philadelphia the average duration of quarantine from the onset of the disease to the 2d consecutive negative culture was 15.9 days (C. Y. White).[3] Other details in connection with quarantine and disinfection are those appropriate to infectious diseases in general.

Management of Carriers.—The procedure for children of the family and for those in constant close contact with the patient has been referred to in the preceding paragraph. What is to be done with carriers of other sorts; *i.e.* those who without such known exposure are found to have diphtheria-bacilli on the mucous membranes, is one of the most perplexing of problems. Theoretically it would be advisable to quarantine all such; but the studies of Pennington and others, already referred to (p. 501), showed the manifest impossibility of this procedure. Certainly when cases repeatedly develop in schools or hospitals, the only proper course is to make cultures from every individual, and to institute the proper treatment. (See Treatment of Carriers, p. 530.)

TREATMENT OF THE ATTACK. **General and Hygienic.**—The selection and the care of the sick-room are those described under the Management of Infectious Diseases (p. 345). Fresh air and sunlight are of great importance. The patient should be confined absolutely to bed in a recumbent position no matter how mild the attack. The diet should be liquid, preferably milk. The difficulty in swallowing and the distaste for food which sometimes develop later in the attack, often occasion such active resistance on the part of the child that the curtailing of the frequency of feeding may be necessary. This applies also to all local treatment in this disease, since exhaustion is such a prominent symptom. The question of the course to be pursued must be determined for each case individually. Feeding through a stomach-tube or nasal tube is frequently a great aid in such cases. Very high fever may require hydrotherapy in the form of warm or cold packs carefully applied, with due regard to the avoidance of exertion or excitement on the part of the patient.

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[1] Jour. Amer. Med. Assoc., 1924, LXXXII, 552, 567.

[2] Jour. Amer. Med. Assoc., 1926, LXXXVI, 998.

[3] Personal communication.

The unusual prostration characteristic of diphtheria renders *stimulation* necessary in all but the mildest cases. It is usually better to begin this early rather than to wait for visible evidences of exhaustion to manifest themselves. Alcohol for many years was a standby, and, despite the discussion regarding it, it is, in our opinion, of undoubted benefit if properly used. It is tolerated in relatively large amounts in this disease. Morphine has been highly recommended in cases of laryngeal and tracheal stenosis. It does not, of course, remove the actual stenosis, but it modifies the cough and dyspnea and produces much needed rest. In addition, digitalis, camphor, caffeine, strychnine, adrenaline and nitroglycerine are frequently required to combat circulatory disturbances. If heart-block, occurs the administration of digitalis is contraindicated and that of other cardiac stimulants is usually useless, and absolute rest and morphine are then the only helpful measures.

Serum-treatment.—Of the numerous remedies formerly in use little is now heard since the introduction and wonderful success of the *serum-treatment.* (See Prognosis, p. 512, for statistics.) This method, first brought prominently before the medical profession by von Behring and Wernicke,[1] consists in the injection into the circulation of the patient of the serum of an animal—the horse being relied upon for this purpose—which has received repeated injections of the diphtheria-toxin in increasing doses, and which, as a result, has developed in its blood a body, perhaps a globulin, powerfully antagonistic to the toxin, and hence called the antitoxin. This neutralizes the toxin already in the patient's blood, checks the growth of the membrane and prevents the formation of more toxin by inhibiting further development of the bacilli. The strength and dosage of the serum is measured by "antitoxin-units," such a unit being the amount of serum sufficient to neutralize the effect of 100 times the dose of diphtheria-toxin which would kill in 4 days a guinea-pig weighing 250 grams (8.82 oz.). The antitoxin-serum is preserved in hermetically sealed glass vessels. It should be used promptly after it is opened.

Method of Employment.—The antitoxin can be given with a special syringe made for the purpose and thoroughly sterilized just before using. Makers now frequently supply the serum in a glass tube, which can itself be used as a syringe, a glass piston and hypodermic needles already sterilized accompanying this. A region should be chosen which is not pressed upon in lying, the flank, the anterior of the thigh, and the abdomen being favorable situations. The skin should be thoroughly scrubbed with diluted alcohol or painted with tincture of iodine and the hands of the operator cleansed. The air should be expelled before the needle is thrust under the skin, or, better, into the muscles, since the absorption is more rapid here. In severe cases or those seen late the serum should be given intravenously; since of all things it is important to check further toxic action as promptly as possible (see p. 513), and the intravenous method is the best for accomplishing this (Park).[1] Local anesthesia is not necessary. A small piece of adhesive plaster or a pledget of cotton afterward painted with collodion may be applied after the needle is withdrawn.

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[1] Zeitschr. f. Hyg., 1892, XII, 10.

Dosage.—For immunizing purposes the amount to be given varies from 500 units in the 1st year of life up to 1000 units for older children. In average cases of the disease over 2 years of age seen early 6000 to 8000 or more units should be given. If no improvement follows, this may be repeated within 6 to 12 hours and the treatment continued perhaps in larger doses until improvement is seen. It is questionable, however, whether, if a first large dose is not beneficial, later doses will have any effect, for if the first treatment was of sufficient size, the antitoxin remains in the blood in large enough amount to neutralize any further toxin produced. In all severer cases in which the deposit has extended beyond the tonsils, and in all instances where involvement of the larynx is threatened, the dose should be at least 10,000 units after 2 years of age, often best given intravenously. Serum used in this way should be of clear color and highly concentrated, and if no serum has been given previously or not within 6 days, a preliminary small intramuscular injection should be administered or a skin-test done to determine the presence of anaphylaxis. If the interval has been less than 6 days, no preliminary test is needed (Osgood).[2] Owing to these possible dangers from the intravenous route, it is better to reserve this for the serious cases or those seen late in the disease. Platou[3] recommends the intraperitoneal route for the giving of antitoxin, since within an hour after the injection it is sufficiently absorbed to neutralize the circulating toxin. The method should be of use in small infants with collaped veins. (For the technic of intraperitoneal injection see p. 260.)

Time of Administration.—The earlier in the disease the antitoxin is administered the more certain and powerful the action and the smaller the amount required to be effective. This is due to the fact, already stated, that the antitoxin stops the further destructive action of the diphtheria-toxin, but cannot regenerate the cells which have already been injured by it. The comparative results of treatment begun on different days is very strikingly shown in the statistics detailed under Prognosis (p. 512). When given after the 3d day of the disease much less good can be expected. Nevertheless, as there are exceptions to the rule, no case should be considered to have lasted too long to seek benefit from serum-treatment, and the dose should be large; while on the other hand very severe cases may be already too far advanced, even upon the 1st day, to be capable of being benefited. Even in the mildest and the doubtful cases, by far the wisest course is to administer antitoxin immediately without waiting for confirmation of the diagnosis by bacteriological examination.

Results of Antitoxin Treatment.—Favorable results following the injection are seen within 12 to 24 hours and sometimes earlier. They consist in a softening and separating of the pseudomembrane at its edges, a diminution of the glandular swelling and of nasal discharge, and an improvement in the general condition. In many cases the membrane disintegrates and disappears with surprising rapidity and the throat is clear in from 3 to 4 days. Complications of diphtheria depending on damage already done by the toxin or upon septic conditions are in no way benefited by antitoxin. Thus the serum-treatment unless given early and in large dosage does not appear to diminish the number of cases of paralysis, since the damage done to the cells of the nervous tissue takes place very early in the attack. In favorable laryngeal cases there is a rapid lessening of the stenosis. The good effects of antitoxin are particularly evident in the reduction of the number of tracheotomies and intubations now necessary, as well as in the mortality where operation has been required. (See Prognosis, p. 512.)

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[1] Jour. Amer. Med. Assoc., 1921, LXXVI, 109.

[2] Jour. Amer. Med. Assoc., 1923, LXXXI, 1406.

[3] Arch. of Pediat., 1923, XL, 575.

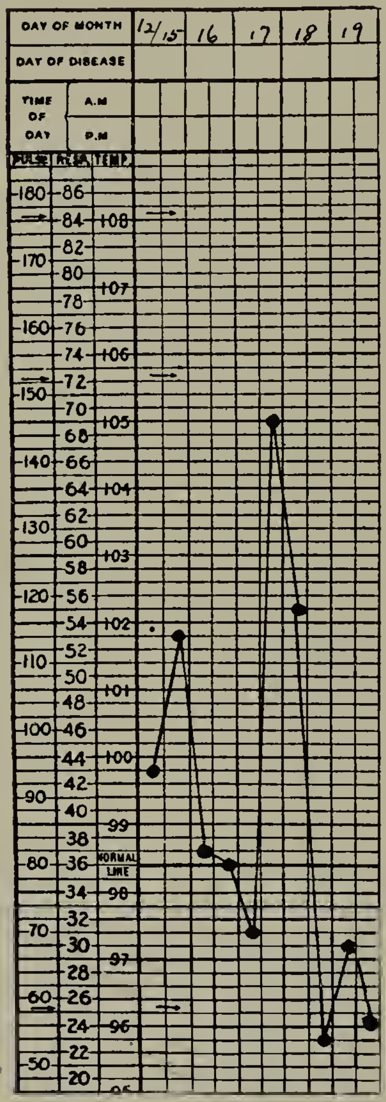

FIG. 139.

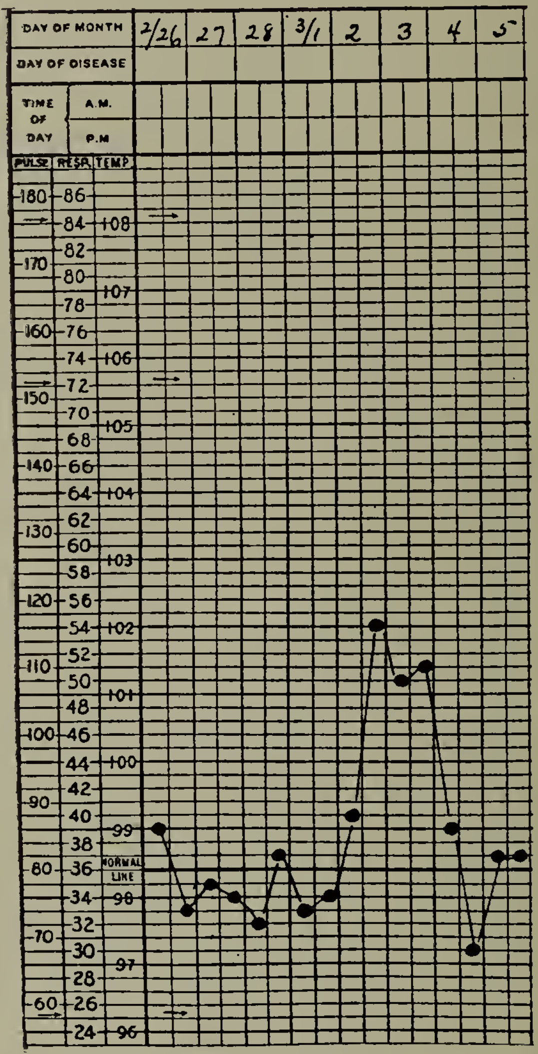

FIG. 140.

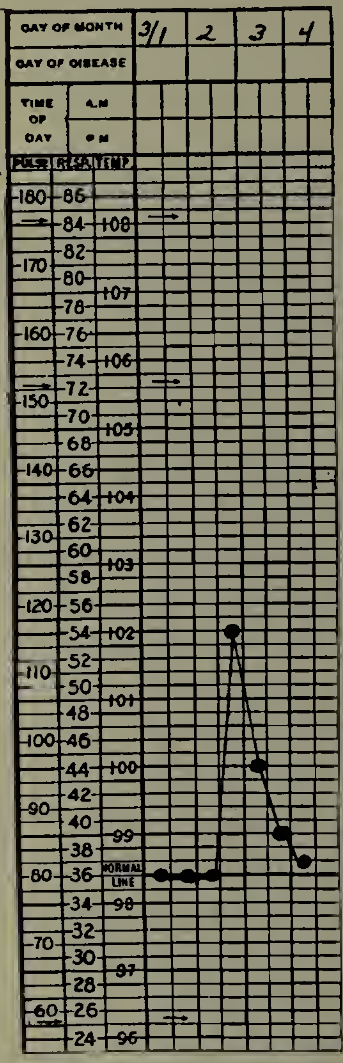

FIG. 141.

FIG. 139.—RISE OF TEMPERATURE PROMPTLY AFTER PROPHYLACTIC ADMINISTRATION OF DIPHTHERIA-ANTITOXIN.

Harry M., aged 1 year, 4 months. Recovering from grippe. 1200 units of antitoxin. given on the morning of Dec. 17.

FIG. 140.—FEBRILE REACTION PROMPTLY DEVELOPING AFTER DIPHTHERIA-ANTITOXIN.

Chester E., 2 years old. Convalescent from bronchopneumonia. 1235 units of antitoxin given on the afternoon of Mar. 1.

FIG. 141.—SERUM SICKNESS OCCURRING 7 DAYS AFTER THE ADMINISTRATION OF ANTITOXIN.

Dorothy McC., 7 years old. Immunizing dose of diphtheria-antitoxin (2500 units) given on Feb. 23; throat been slightly red and cultures positive; Feb. 25, cultures being still positive. antitoxin repeated; Mar. 2, wide-spread eruption of urticaria which is giving pain; Mar. 3, still wide-spread typical antitoxin rash; Mar. 4, rash about gone.

Unfavorable Effects of Antitoxin.—A group of symptoms to which the title "Serum-Disease" has been applied, not infrequently follows the administration of antitoxic serum. As this condition is due to the introduction of a foreign blood-serum independently of its content of diphtheria-antitoxin contained, it would seem that the smaller the amount of serum used the better. Consequently concentrated sera are always to be preferred as less liable to produce unpleasant after-effects and less painful at the time of injection. It is not, however, certain that the quantity of serum used bears any real relationship to the production of symptoms. These symptoms are seen in from 5 to 30 per cent. of the cases, and occur anywhere from the 1st to the 17th day, although oftenest from the 5th to the 9th (Sturtevant).[1] They consist in erythema of a scarlatiniform or rubeloid type, urticaria, edema, fever (Figs. 139, 140, 141), vomiting, painful articular swelling, albuminuria, malaise, headache, and general aching. Some or all of these may be present, urticaria, often wide-spread and lasting 2 to 3 days, being the most frequent. (See p. 488, Fig. 127.) Instances of sudden death following the injection have occurred, but these have appeared, certainly in some cases, to be due to the mere hypodermic puncture in individuals suffering from lymphatism. (See Sudden Death, p. 243.) In other patients grave symptoms or death have apparently been due to an unusual susceptibility to the action of an alien serum, such as that of the horse. (See Allergy, Vol. I, p. 750.) It does not appear proven that the result is produced by the antitoxin itself. The symptoms in these cases consist of alarming dyspnea and cyanosis developing promptly after the injection, and either passing away gradually or terminating fatally in a few minutes. It is true that repeated injections of antitoxin at intervals separated by months or longer may produce an increasing sensitiveness to the serum, but the majority of cases of sudden death have taken place after the period of childhood, after the first injection, and in individuals with a decided asthmatic tendency, especially produced by approach to a horse. Whatever the cause, the numbers of instances of dangerous symptoms or fatal result are so extremely few, as compared with the frequency with which antitoxin has been employed, that they must be ignored for practical purposes. As the serum has no action, either good or bad, upon complications, it should be given regardless of their presence. In persons with a positive asthmatic history or otherwise sensitive it may be well to omit the administering of immunizing doses, or, in those with the disease, to defer it if the attack is mild. In those positively requiring the use of serum it should be given in a subcutaneous injection of 0.1 c.c. (0.0034 fl.oz.) with another injection of .2 c.c. (0.0068 fl.oz.) following in ½ to 1 hour, and so on in increasing dosage until the required amount is administered. A number of writers have claimed that the value of the serum through its antitoxin content is no greater than that of simple horse-serum. This has not found general acceptance. The subject has been reviewed by Calhoun,[2] Ratnoff,[3] Feer[4] and others.

Local Treatment.—Local treatment is of value in so far as it destroys or removes the bacilli from the affected mucous membrane, or retards their growth or checks their dissemination by producing conditions of cleanliness. It is harmful when it exhausts the patient; and inasmuch as exhaustion occurs so readily in this disease local treatment must be employed with great discrimination. The chief object is that of cleansing. For this purpose douching of the nose and throat with warm alkaline antiseptic solutions, such as a diluted liquor sodii boratis comp.

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[1] Arch. Int. Med., 1916, XVII, 176.
[2] Amer. Jour. Dis. Child., 1921, XXI, 107.
[3] Berl. klin. Wochenschr., 1923, II, 440.
[4] Münch. med. Wochenschr., 1919, LXVI, 343.

(Dobell's solution) may be employed, or a normal salt or a 1 or 2 per cent. boric acid solution. The injection is preferably given with a fountain-syringe or a soft-rubber nasal syringe (p. 266, Fig. 39). The child should lie well on one side and the fluid, if used in the nose, be made to enter the upper nostril and flow from the other. In some cases an atomizer may well be employed, but the syringe is generally more effective. Local treatment every 3 or 4 hours is enough in mild cases, and in severe ones every 2 hours. Spraying or swabbing the throat with a much diluted and non-acid solution of peroxide of hydrogen is often efficacious in dissolving the membrane. Application of solutions of more powerful drugs, such as preparations of iron, nitrate of silver, and the like, have largely gone out of vogue. They are usually painful and cause distress to and resistance by the patient. An ice-bag applied externally over the position of the tonsils, and small pieces of ice placed frequently in the mouth, tend to relieve the pain and difficulty in swallowing.

Extension of the disease to the larynx is largely prevented by the early and free use of antitoxin. When, however, laryngeal symptoms are threatening, the use of the croup-tent (p. 264) is often of great benefit. The patient should, however, not be kept in the tent over 20 or 30 minutes at one time, as the effect may be depressing or the amount of oxygen too greatly diminished. Sometimes allowing a stream of oxygen to enter the tent at the same time with the vapor is of benefit. Girou[1] advised intratracheal injections of antitoxin in infants in whom tracheotomy for croup had been necessary. Most observers, however, fail to obtain any beneficial effect from this local use, and this is in accord with the fact that the diphtheria-bacillus grows without difficulty in antitoxin.

Operative Treatment.—When cyanosis is clearly increasing in laryngeal cases, stenosis is decided, and the accessory muscles of respiration in full play, operative measures are indicated, and it is important that they be not unduly delayed. These consist respectively in *tracheotomy* and in *intubation*. The latter has largely supplanted the former in most countries. Tracheotomy, however, may quickly become necessary after intubation if no relief is obtained.

The technique of *tracheotomy* is so strictly surgical that no attempt will be made to describe it in this connection. The after-treatment of tracheotomy consists principally in keeping the tube clean of mucus and pseudomembrane, and in maintaining the air saturated with warm water-vapor produced by the boiling of water, the slaking of lime, the plunging of hot iron into water, or in other ways.

Intubation.—This was first successfully put into practice by O'Dwyer[2] and has been the means of saving many lives. Inasmuch as favorable results depend to a large extent on the skill and training of the operator, I append the following description kindly prepared for me by Dr. Henry R. Wharton, formerly Senior Surgeon to the Children's Hospital of Philadelphia.

"*Instruments Required for Intubation.*—The instruments required for intubation are *intubation-tubes*, and an apparatus to insert them. The tubes for children are usually six in number, of different sizes adapted to the ages of from 1 to 12 years. The sort now generally employed consists of a metallic cylinder which bulges near its center, with a collar or head to rest upon the vocal cords. The tubes are gold plated or of hard rubber with a metallic lining, and are provided with an obturator which has a blunt extremity; and through the edge of the collar on each tube there is a small perforation into which a strand of fine braided silk is passed. This serves to remove the tube, if in its introduction it should have been passed into the pharynx or the esophagus instead of the larynx, or if it has to be hurriedly withdrawn, owing to sudden obstruction of breathing.

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[1] Rev. de laryngol. d'otol. et de rhinol., 1923, XLIV, 709.

[2] N. Y. Med. Jour., 1885, XLII, 145.

"*The Introducer.*—This instrument consists of a handle and a staff, bent to a right angle at its extremity, having a screw that attaches it to the obturator, and a sliding gear for detaching the obturator from the tube when it is placed in the larynx (Fig. 142).

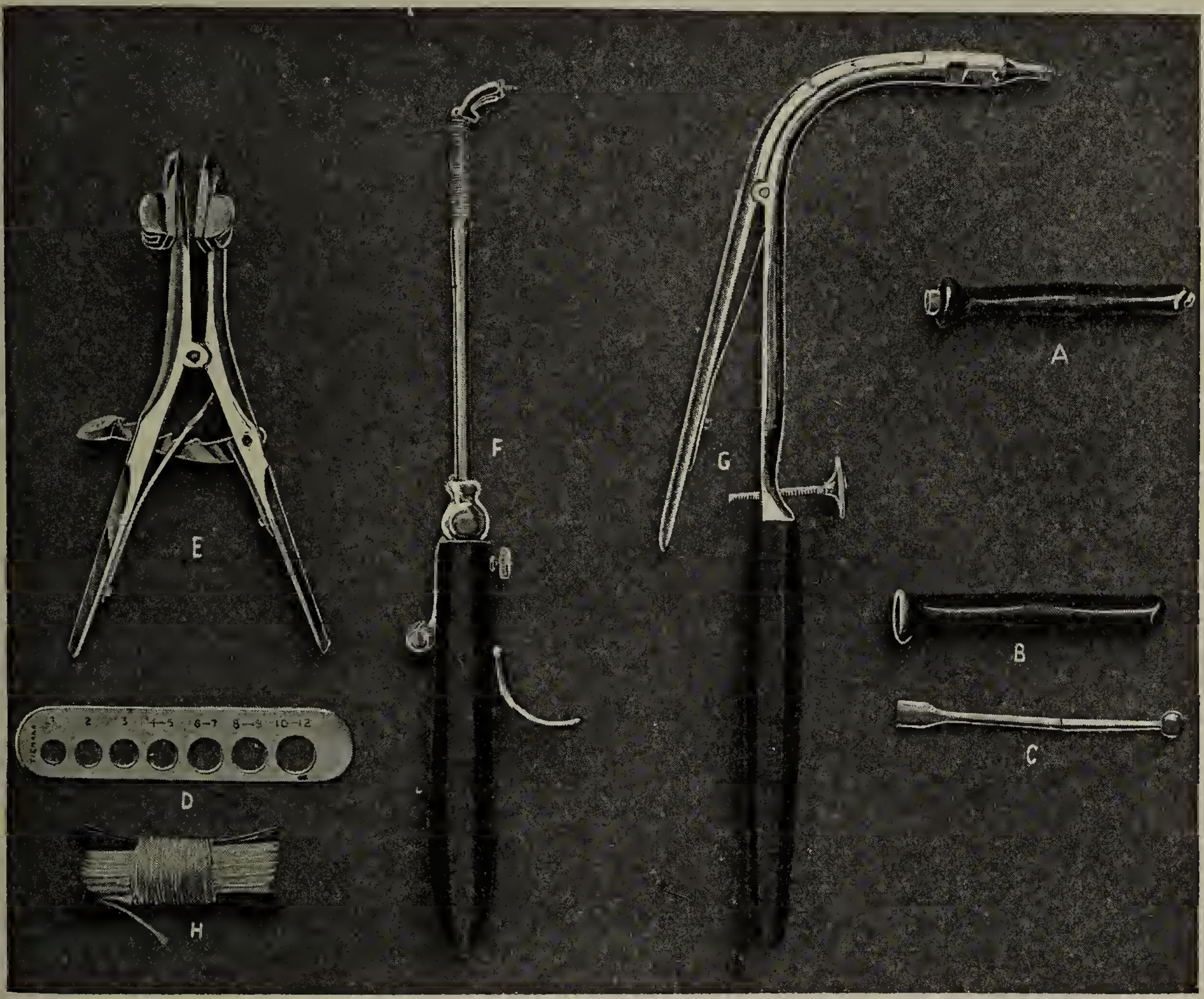

Fig. 142.—O'Dwyer's Intubation Instruments.

A, Tube with obturator; B, tube; C, obturator; D, metal gage; E, mouth-gag; F, introducer; G, extractor; H, silk cord. (*Fowler.*)

"*Mouth-gags.*—Mouth-gags of various kinds may be employed. The one generally used is a self-retaining instrument (Fig. 142), and the portion inserted between the teeth is covered by pieces of rubber-tubing to prevent injury.

"*Extractor.*—The extractor is also bent at a right angle and has at its extremity a small forceps with duck-bill blades which are made to separate and apply themselves to the inner surfaces of the tube with sufficient firmness to withdraw it (Fig. 142).

"*Preparations for Intubation.*—It should not be forgotten that when an intubation-tube enters the larynx breathing is arrested until the obturator is removed, and therefore the manipulations should be as rapid as is consistent with accuracy. The surgeon should select a tube of suitable size for the age of the patient, pass a strand of fine braided silk about 2 feet in length through the opening in the collar of the tube, and knot the ends together. The tube is then attached by means of the obturator to the introducer. To prevent the patient from biting the finger, in case the mouth-gag should slip, the surgeon should protect the index-finger of the left hand in the region of the second joint by wrapping it with a piece of rubber plaster, or by slipping over it a metal shield.

"*Operation.*—This may be performed while the child is in the sitting posture, or while it is recumbent. The former position I prefer. The child should be placed upon the lap of the nurse or assistant, and covered by a blanket loosely thrown around it. The nurse grasps the child's elbows from outside of the blanket and holds them firmly, but should not press them against the chest in such a way as to embarrass the respiratory movements. At the same time the legs of the patient should be secured by being held between the knees of the nurse. The head of the patient is held firmly between the open hands of the assistant, placed on either

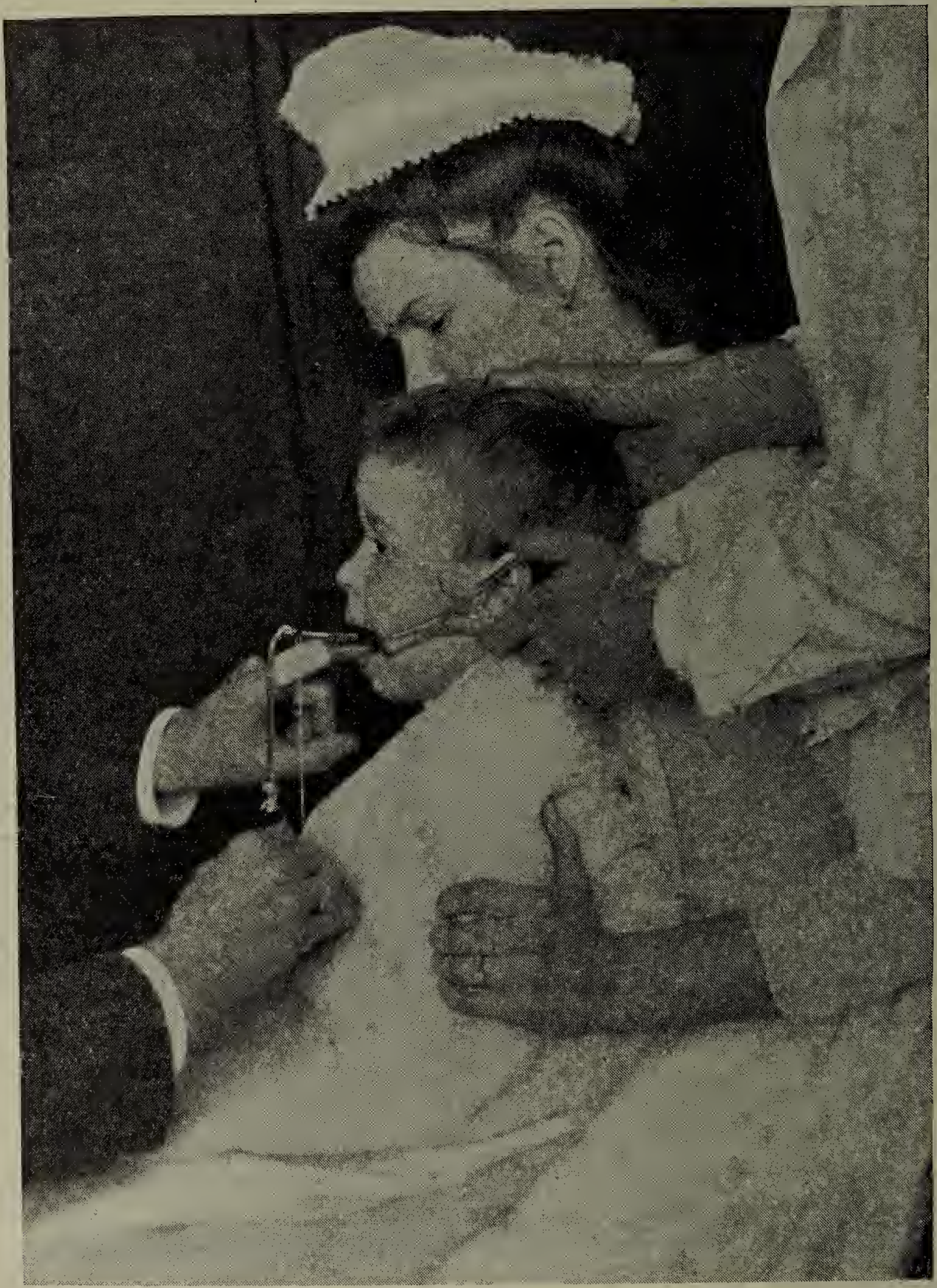

FIG. 143.—INTUBATION. INSERTING THE TUBE.
(*Wharton, Starr's Amer. Text-book of Diseases of Children*, 1894, 314.)

side of the head and cheeks. The left hand of the assistant may also be used to steady the mouth-gag after it has been introduced (Fig. 143). As before stated, the tube may be introduced with the child in the recumbent posture. This I have done when, from the condition of the circulation, I did not think it advisable to lift the patient to the sitting posture. The mouth is opened and the blades of the mouth-gag introduced between the molar teeth upon the left side, and the jaws opened by this as widely as possible. The surgeon next passes the index-finger of the left hand into the pharynx and feels for the epiglottis, hooking this forward by the end of the finger. The tube attached to the introducer held in the right hand is next passed into the mouth and carried back to the pharynx, the operator being careful to see that it hugs the base of the tongue in the middle line, that the handle is depressed well upon the child's chest, and that the silken loop is free. When the extremity of the tube comes in contact with the end of the finger resting upon the epiglottis, the handle of the instrument should be raised as the tube enters into the larynx and descends into that organ, and when in position, the finger is placed upon the head of the tube to prevent its being withdrawn with the obturator. The trigger is next pressed, and the introducer and obturator withdrawn from the mouth by depressing the handle upon the chest. Before removing the finger it is well to push the tube well into the larynx. As soon as the obturator is removed there is generally a violent expiratory effort with coughing, accompanied by a gush of mucopurulent matter or membrane, and after this escapes the breathing is usually satisfactorily established. If the operator has passed the tube into the pharynx or esophagus, no improvement in the respiration takes place, and it should then be withdrawn by the silken loop and another attempt made to introduce it correctly. The mistake which inexperienced operators make in attempting to introduce the intubation-tube consists in not hugging the posterior surface of the tongue closely, as a result of which the tube passes over the epiglottis into the pharynx. The most serious complication which is apt to occur during the introduction is in the pushing of a mass of membrane in front of the tube into the trachea. If this is too large to be expelled through the tube, the breathing is suddenly arrested. The tube should then be removed at once, and if the mass of membrane does not escape upon the expiratory efforts of the patient, the trachea should be rapidly opened. So much do I dread this accident, which has occurred to me in one case only, that I never introduce an intubation-tube without having at hand the necessary instruments for a rapid tracheotomy.

"Another accident which is said to have occurred, of which I have no personal experience, is the pushing of the intubation-tube through the wall of the larynx into the cellular tissue. This is not likely to happen unless undue force has been used. The production of a false passage is recognized by the fact that, although the tip of the tube can be felt to enter the larynx, it does not descend, but projects above the epiglottis. Some operators keep the silken loop attacked to the tube during the time it is retained in the larynx, so that by drawing upon it the nurse or attendant is able to withdraw the tube instantly if it should become obstructed with membrane, or be coughed up and pass into the pharynx or esophagus. I generally allow the loop to remain in place for 10 or 15 minutes. At the end of this time I introduce the finger into the mouth and feel that the tube is in its proper place, and while the tip of the finger rests upon the edge of the tube, divide the silk loop and withdraw it.

"*After-Treatment.*—After intubation, so far as the tube itself is concerned, no treatment is required. The patient should be kept in a warm room in which a certain amount of moisture is maintained by the use of boiling water or by a steam-spray. If there is but little tendency to expectoration through the tube, soda solution, which consists of carbonate of soda, 1 to 2 dr. (3.9 to 7.8); glycerine, 1 fl.oz. (30); and water, 6 fl.oz. (177) applied by means of a steam-atomizer, may be used with advantage. One of the greatest troubles after intubation of the larynx is the satisfactory feeding of the patient and the administration of liquid medicines. Liquids, as a rule, are not swallowed well, a portion of them passing into the tube and producing violent coughing. Cases are, however, occasionally met with in which the swallowing of liquids does not seem to be specially interfered with by the presence of the intubation-tube. Nursing

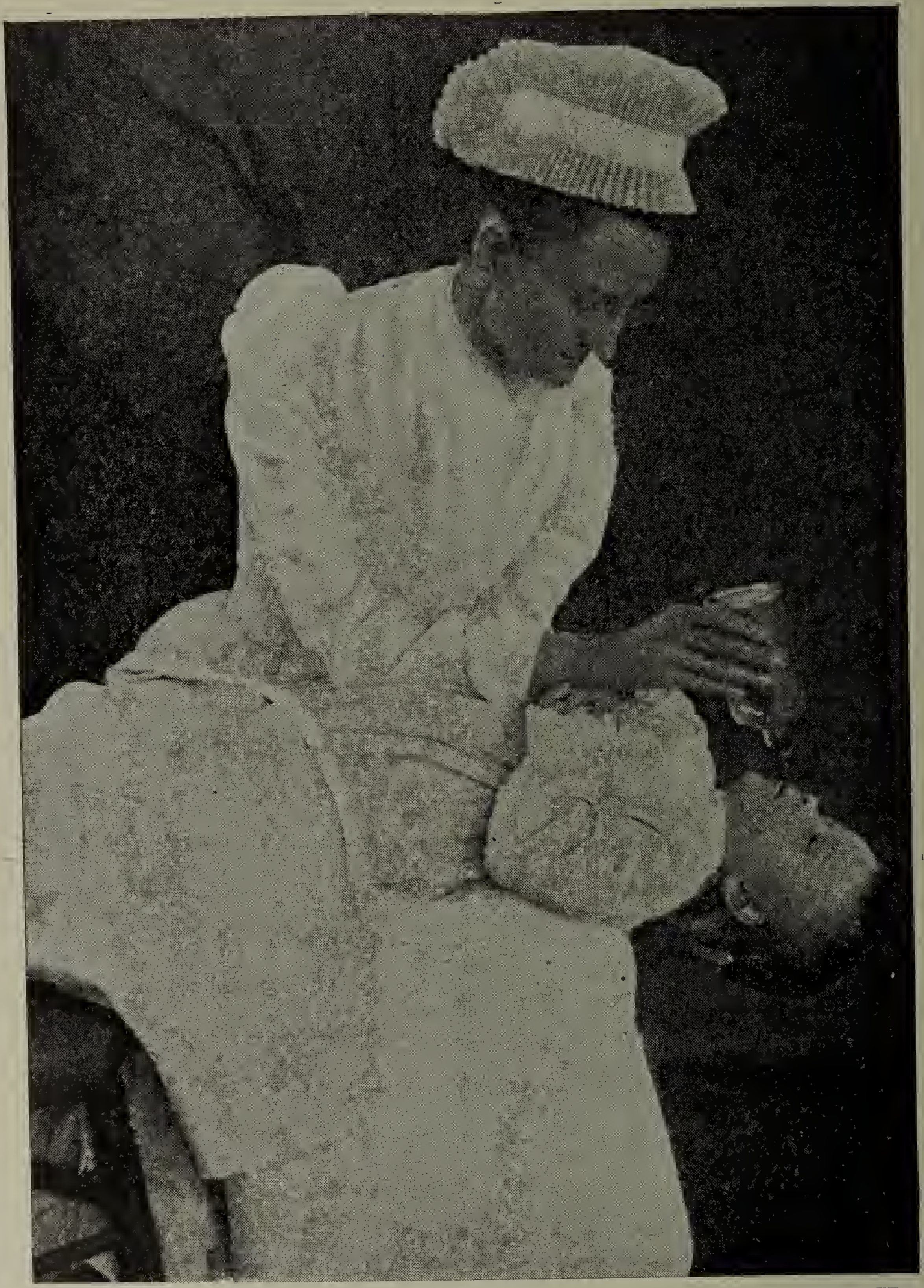

FIG. 144.—METHOD OF FEEDING INFANT AFTER INTUBATION, WITH THE HEAD LOWER THAN THE BODY.

(*Wharton, Starr's Amer. Text-book of Diseases of Children,* 1894, 316.)

infants may sometimes continue at the breast after the operation. I usually order a diet of semisolids, such as corn-starch, soft boiled eggs, mush, and junket. The taking of a sufficient quantity of water often causes trouble, and in such cases the child may be allowed to swallow small pieces of ice, or water may be regularly administered by the rectum. In cases where there is difficulty in swallowing even this form of diet, it may be necessary to resort to introduction of liquids into the stomach by means of a feeding-tube passed through the nostril into the esophagus. In young patients in whom a liquid or milk diet is essential, if the head is dropped a little lower than the body during the act of deglutition it will often be found that fluids are swallowed without difficulty (Fig. 144).

"*Removal of Intubation-tubes.*—The intubation-tube usually remains in place about a week. I usually remove it within 3 or 4 days, and if the breathing is satisfactorily carried on for half an hour, and no dyspnea appears, its reintroduction may not be necessary. If, however, after it has been out a few minutes, dyspnea returns, it should be promptly reintroduced and its removal should not be attempted for 3 or 4 days. In many cases the tube is coughed out within a week from its introduction, and its reintroduction is not often required in these cases. It can usually be permanently dispensed with in from 5 to 10 days, although I have had cases in which it could not be permanently removed until the 15th day. Cases have been reported in which it had to be worn for many months. After an intubation-tube has been coughed up or removed, the patient should be carefully watched from 12 to 24 hours, for the dyspnea may return at any time within this period and require replacement of the tube. The intubation-tube may be coughed up and swallowed, entering the stomach. This accident need cause no anxiety, as in my experience these tubes usually pass safely through the intestines. After intubation of the larynx very decided hoarseness often persists for several weeks, but after this time usually entirely passes away.

"*Retained Intubation-tubes.*—If an intubation-tube has been worn for a long time there is sometimes great difficulty in removing it permanently. Its removal may often be accomplished by introducing at intervals tubes of gradually increasing size."

In some cases of chronic tube-retention it is necessary to do a tracheotomy and then gradually dilate the larynx.

Treatment of Complications and Sequels and of the Convalescence.—The exhaustion and *cardiac weakness* following severe cases of diphtheria require especial attention. Rest in bed in the recumbent position must continue so long as there are any evidences of decided weakness of the heart, shown by rapidity, irregularity, or slowness of the pulse, or by weak heart-sounds. Excitement and physical exertion of all sorts must be carefully avoided. Even after mild attacks the patient should be kept in bed for at least a week after the membrane has disappeared and only cautiously allowed to sit upright, although the heart may appear to be entirely normal. General tonic treatment is indicated, especially with alcohol, strychnine and sometimes digitalis. Adrenaline chloride (1:1000) has been used to prevent cardiac failure, the dose being 5 to 10 m. (0.31 to 0.616) according to age. Pituitrin has also been employed with success.

The *anemia* which often remains demands a long course of iron or arsenic with abundant nourishment. Frequently cod-liver oil is of benefit, and the continued use of alcoholic stimulants may be needed.

Nephritis remaining as a sequel requires treatment appropriate to it. It may necessitate a continuance of liquid diet, especially milk, longer than one would wish in view of the importance of nourishment from other points of view.

The treatment of *post-diphtheritic paralysis* depends to some extent upon the part affected. Paralysis of the extremities is to be aided by massage. That of the muscles of deglutition may occasionally require feeding by gavage, but this is uncommon. In any form of paralysis the chief aid is to be sought in electricity and the free use of strychnine combined with abundant nourishment. There has been much discussion as to the value of antitoxin in preventing or relieving paralysis. As has been shown by Kleinschmidt[1] and others, there appears to be no relationship whatever between the development of paralysis and the presence of antitoxin in the blood. Nevertheless, since good results have been claimed by many, among them Heubner,[2] from the employment of large doses as a curative measure, the treatment may well be given a trial. Labbe[3] claims to have cured a paralysis of the soft palate, which had been present for 4 years, by antitoxin injections. Marriott[4] reports a unique case in which a child suffocating because of paralysis of the respiratory muscles was kept alive for 5 days with artificial respiration by a Gesell-Erlanger apparatus. Complete recovery followed.

Treatment of Carriers.—Finally, there is often difficulty in ridding the patient of the Klebs-Löffler bacilli, which may persist long after all symptoms have disappeared, the individual thus becoming a carrier. The treatment is the same for healthy individuals who have not had the disease, but who have been proven to be dangerous carriers. The difficulty in dislodging the bacilli in these cases is often very great. This is doubtless because in many instances they may remain indefinitely in the crypts of the tonsils or adenoid growths or in the nasal sinuses. Various measures have been recommended. A very weak (1:10,000) solution of bichloride of mercury may be employed by syringing the nose or as a gargle; or a weak solution of boric acid or of liquor sodii boratis comp. may be used. Gray and Meyer[5] were able to render 88 of 90 carriers free from diphtheria-baccilli in a comparatively short time, by local applications of 0.5 to 2.0 per cent. mercurochrome; Saurman[6] recommends spraying the nose and throat 4 to 5 timses a day with a 2 per cent. aqueous solution of gentian violet, and Kellogg,[7] obtained the test results by painting the throat with iodine in glycerine. Hand[8] applied with success a strong solution of nitrate of silver (60 gr.:1 fl.oz.) (4:30) to the throat. Many report success with the spraying of the nose and throat several times daily with a boullion-culture of the staphylococcus pyogenes aureus, as first recommended by Schiøtz.[9] A rapid disappearance of the diphtheria-bacilli may follow, these being crowded out by the other germs. (See articles by Lorenz and Ravenél,[10] Rolleston,[11] Lake[12] and others.) Good results have also been claimed from the application of cultures of lactic acid bacilli applied in a similar manner (Nicholson and Hogan).[1] Vaccine-treatment with killed diphtheria-bacilli has also been recommended, but the results as reported by Park and Zingher[2] have been disappointing. In every case of persistence of the bacilli an inoculation-test in guinea-pigs should be made. If the germs are proven to be non-virulent, quarantine is no longer necessary. In other cases of unusually long persistence of virulent bacilli, often the best means of treatment is the removal of the tonsils and adenoids, which is usually followed by prompt disappearance of the organisms (Weaver).[3]

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[1] Jahrb. f. Kinderheilk., 1917, LXXXV, 261.

[2] Münch. med. Wochenschr., 1916, LXIII, 128.

[3] Arch. de méd. des enf., 1921, XXIV, 612.

[4] Jour. Amer. Med. Assoc., 1920, LXXV, 668.

[5] Jour. Infect. Dis., 1921, XXVIII, 323.

[6] Jour. Amer. Med. Assoc., 1924, LXXXIII, 249.

[7] California State Jour. of Med., 1915, XIII, 150.

[8] Phila. Med. Jour., 1898, II, 432.

[9] Ugeskr. f. Läger, 1909, LXXI, No. 49. Ref. Jour. Amer. Med. Assoc., 1910, LIV, 422.

[10] Brit. Jour. Child. Dis., 1913, 298.

[11] Jour. Amer. Med. Assoc., 1912, LIX, 690.

[12] New York Med. Rec., 1912, LXXXI, 1228.

CHAPTER XIII

GRIPPE

(Influenza)

Grippe, or influenza, was described clearly in the 12th century, and its epidemic, infectious character recognized since the 16th century. It first appeared in the United States in 1627. Whether the cases observed in these earlier wide-spread outbreaks were etiologically identical in nature with those which have been encountered in more recent years cannot be positively determined; but from a clinical point of view no sharp distinction can be drawn. One of the difficulties in tracing the analogy between the present grippe or influenza and older epidemics is the difference in nosology. Various names such as Sandfly fever and Three-day-fever have been given to wide-spread epidemics of febrile conditions which probably were influenza, and the disease is said to have been mistaken for dengue and malaria. It is evident that the possibility of separation of sporadic grippe and epidemic grippe or influenza is not possible at the present time, and will not be until more is known of the etiology of these conditions. Some authors, however, as Jordon,[4] believe that the so-called influenza of interepidemic years is in whole or in part different from the influenza of the great epidemics. From a bacteriological standpoint a distinction may, it is true, be made, and it is to those cases depending upon the influenza-bacillus that the title "influenza vera" has been applied, others resembling these clinically but being produced by other germs having been called pseudo-influenza, or "influenza nostras." It has also been proposed to designate as influenza all the diseases depending upon the influenza-bacillus; a course very similar to that resulting should we call "pneumonia" all lesions produced by the pneumococcus. It seems best, therefore, to class together all those cases showing symptoms which have usually been regarded as evidences of grippe or of influenza respectively, and in which no sharp clinical distinction can be made, and to designate these "grippe," whatever the active germ may be. Certainly the mere existence of symptoms of a severe febrile cold does not warrant the diagnosis of grippe. Certainly, too, the epidemic contagious

[1] Jour. Amer. Med. Assoc., 1914, LXII, 510.
[2] *Loc. cit.*
[3] Jour. Amer. Med. Assoc., 1921, LXXVI, 831.
[4] Amer. Jour. Hygiene, 1922, XI, 325.

condition to which the title "grippe" is rightly applied is as often dependent upon other germs as upon the influenza-bacillus. The subject of grippe in early life has been exhaustively studied by Risel[1] and by Moffett[2] with extensive bibliography, and an excellent critical review of the subject of the etiology of grippe is given by Zinsser.[3]

Etiology. Predisposing Causes.—Climate, race, locality, sex, and social conditions, exert no influence. The previous health is also not a factor, except that affections of the respiratory tract increase the susceptibility. Consequently all causes are important which render the mucous membrane of the respiratory tract sensitive, such as bad weather, exposure to cold, insufficient ventilation, and the like.

All periods of life are susceptible, and the disease may occur even in the new-born. Strassman[4] observed 8 cases at this time of life, and Cannata[5] has seen 22 infants aged between 5 and 20 days suffering from grippe. Abt.[6] described a case which appeared to be of congenital origin. In Comby's[7] statistics of 218 cases in children, 48 occurred from birth to 2 years, 1 being but 17 days old; 76 at from 2 to 5 years, and 94 at from 5 to 15 years. Achard[8] reports 32 cases below the age of 2 years. The *epidemic character* is more marked than perhaps in any other affection. The disease at first occurred in epidemics, generally wide-spread and separated by decades. The great epidemic of 1889 appeared first in Turkestan after an interval of many years, and extended rapidly over the greater part of the earth. In less than 6 months it had reached the United States. In 1890–91 there was another serious outbreak most marked in England and America. The disease then became endemic to a limited extent, with occasional larger and more wide-spread outbreaks, at first regardless of season, but later usually in the cooler months of the year; until the greatest and most serious epidemic of it ever experienced, which occurred in 1918. This apparently arose in the Orient[9] and spread rapidly over Europe and America, millions of individuals being attacked. Of the cities of the United States, Philadelphia suffered very heavily, about 150,000 cases having occurred in the course of 2½ months.[10] In the years 1918–1919 1,000,000 cases are said to have been reported in Pennsylvania alone.[11] The *individual susceptibility* is extreme, the large majority of persons exposed contracting the disease. This was especially true in the epidemics of 1889–90 and of 1918. In the latter children were somewhat less frequently and decidedly less severely attacked than adults.

The relationship of grippe to epidemic encephalitis is discussed elsewhere. (See p. 612.)

Exciting Cause.—The disease is a distinctly infectious one. The investigations of Pfeiffer[12] revealed an extremely small, non-motile bacillus in the sputum and the nasal secretion, at first free in the mucus and later in the pus-cells. It is less often present in the blood and it has been recovered from the cerebrospinal fluid by lumbar puncture. It generally disappears with the return of health, and it lives outside of the body in infected nasal or bronchial mucus for only about 14 days. Exceptions occur, however, and in more chronic cases or even in those entirely convalescent it can sometimes be found in the secretions or in the pus of complicating conditions even for months. According to Lord[1] it could be discovered in the sputum years after recovery from the disease. Although the influenza-bacillus appears to have been that most frequently present in the early epidemics, as time passed it was more and more replaced by other germs, and up to the year 1918 this bacillus could not be found in the large majority of cases. In the 1918 epidemic the opinions regarding the exciting cause were most divergent; in some regions the influenza-bacillus being reported in the majority of cases; in others its occurrence being comparatively uncommon and other organisms of various sorts being apparently the agents. Wollstein and Goldbloom,[2] at autopsy on 18 infants under 2 years of age, always found the influenza-bacillus but never in pure culture. Somewhat suggestive of the etiological relationship of the influenza-bacillus are the positive inoculation-experiments on monkeys by Blake and Cecil[3] and on man by Davis.[4] Influenza-bacilli have been found in conditions having little resemblance to influenza, or as harmless saprophytes in measles, pertussis, etc. (Zinsser).[5] Wollstein and Spence[6] observed the germ in about 50 per cent. of 1000 cases of all kinds of inflammation of the respiratory tract, and consider that it is probably an early secondary invader and a cause of complications. Among the other germs recorded are the micrococcus catarrhalis, pneumococcus, staphylococcus, bacillus mucosus capsulatus, streptococcus mucosus capsulatus, streptococcus hemolyticus, diplococcus mucosus and pneumococcus mucosus. These germs may occur in practically pure culture or often associated in various ways. Yet it is uncertain whether the influenza-bacillus or any of these are the actual cause of grippe, and whether the disorder is not produced by a microörganism still undiscovered. Indeed, the influenza-baillus may be regarded as a part of the flora of the normal respiratory tract, as shown by Lord, Scott and Nye[7] and others. Nicolle and Lebailly,[8] Gibson and Connor[9] and others have performed inoculation-experiments on animals and man with a filterable virus, and obtained at times positive results. The studies of Olitsky and Gates,[10] Olitsky and McCartney[11] and Lister,[12] with a minute anaerobic filterable body (Bacterium pneumosintes) suggest that this is either the cause of grippe or at least prepares the field for the invasion of secondary organisms. The whole subject, then, is an unsettled one which needs considerable further study. There is sufficient resemblance between the cases occurring in different epidemics and certain of the interepidemic cases to suggest a common cause for all of them. Any difference may be due either to variation in virulence of the causative agent, or to the different secondary invading organisms.

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[1] Ergebn. der inn. Med. u. Kinderheilk., 1912, VIII, 211.
[2] Arch. of Pediat., 1919, XXXVI, 578.
[3] Medicine, 1922, I, 213.
[4] Zeitschr. f. Geburtsh. u. Gynák., 1890, XIX, 39.
[5] La Pediat., 1923, XXXI, 1137.
[6] Jour. Amer. Med. Assoc., 1919, LXXII, 980.
[7] Bull. de la soc. méd. des. hôp., 1890, VII, 67.
[8] Brit. Jour. Child. Dis., 1919, XVI, 78.
[9] U. S. Publ. Health Service, 1918, Suppl. No. 34, Sept. 28.
[10] Monthly Bulletin, Dept. of Health, 1918, III, Nos. 10–11, 23.
[11] Jour. Amer. Med. Assoc., 1920, LXXIV, 742.
[12] Deutsch. med. Wochenschr., 1892, XVIII, 28; Zeitschr. f. Hyg., 1893, XIII, 357

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[1] Bost. Med. and Surg. Jour., 1905, CLII, 537.

[2] Amer. Jour. Dis. Child., 1919, XVII, 165.

[3] Jour. Amer. Med. Assoc., 1920, LXXIV, 170.

[4] Jour. Amer. Med. Assoc., 1919, LXXII, 1317.

[5] *Loc. cit.*

[6] Amer. Jour. Dis. Child., 1920, XIX, 459.

[7] Jour. Amer. Med. Assoc., 1919, LXXII, 188.

[8] Ann. de l'Inst. Pasteur, 1919, XXXIII, 395.

[9] Brit. Med. Jour. 1918, II, 645.

[10] Jour. Amer. Med. Assoc., 1920, LXXIV, 1497; 1921, LXXVI, 640. Jour. Exper. Med., 1922, XXXVI, 685.

[11] Jour. Amer. Med. Assoc., 1923, LXXXI, 744.

[12] South African Med. Rec., 1922, XX, 434.

Grippe is very contagious. In the large majority of instances transmission is direct through the infectious secretion from the respiratory tract. Usually an outbreak among the children of a family arises from one of the adult members sick with the disease. The air may also spread the disease by disseminating the dried infected mucus, but probably only to a limited distance. Indirect transmission by a third healthy person or by clothing, and the like, is of doubtful occurence, but it is possible that there are healthy "carriers." The entrance of the germs into the body is by way of the respiratory tract.

Pathological Anatomy.—Apart from the presence of a catarrhal condition of the respiratory and alimentary mucous membranes the lesions are those chiefly of the complications which may arise. In certain epidemics broncho-pneumonia is almost invariably found, and capillary hemorrhages in practically all of the viscera (Wollstein and Goldbloom).[1] Subcutaneous emphysema of the neck, chest and shoulders may occur, and a peculiar degeneration of the recti muscles with hemorrhage has been described by Bakwin[2] as a frequent finding. Bakwin has also observed that a nasal sinusitis is very frequently present. This has been noted by others, particularly by Hurley,[3] who claims that a mucous nasal sinusitis due to the influenza-bacillus is invariably present, and that this is the real nature of the disease called influenza.

Symptoms.—The clinical manifestations are most varied and depend to a large extent upon the epidemic. Thus in the epidemic of 1889, as observed in the United States, neuralgic symptoms were by far the most prominent, while in that of 1918 pulmonary manifestations prevailed. We may, however, recognize a *typical form* of grippe, and a number of *modified forms*, marked by the predominance of certain groups of symptoms. Some one or another of the variations has oftener been witnessed than the type itself.

TYPICAL FORM. **Incubation.**—This is short, lasting from 1 to 3 days and is generally unattended by symptoms. Occasionally malaise, irritability, vague pains and loss of appetite are witnessed.

Symptoms of the Attack.—In typical grippe there is exhibited a fair balance between the various groups of symptoms which characterize the different varieties. The onset is usually sudden, with chilliness, high fever and sometimes convulsions. In older children pain in the limbs, trunk and head may be complained of; in younger ones there is clearly discomfort, the nature of which cannot be determined. The temperature does not remain persistently high, but runs an irregular and characteristic course, varying from 100° to 105°F. (37.8° to 40.6°C.) and falling by lysis or crisis in 3 to 4 days (Fig. 145). The respiration and pulse are accelerated in proportion to the temperature. Vomiting sometimes occurs and may be obstinate; diarrhea may develop; loss of appetite is marked. Prostration, out of proportion to the other symptoms, is one of the most characteristic manifestations. A varying degree of inflammation of the nose and throat and less often of the trachea and bronchi is present, yet not of a nature sufficient to account for the general symptoms. Albuminuria is seen in a small proportion of cases; enlargement of the spleen is not infrequently discoverable. Bunting[4] has carefully studied the leucocytic picture in uncomplicated grippe and has found that there may be an early polymorphonuclear leucocytosis which is followed by a leucopenia, the lymphocytes being relatively increased. Kinsella and Broun[1] note that the clotting time is delayed and the platelets reduced, the latter observation being also made by Bunting. Leucocytosis develops with secondary infection. In the 1918 epidemic leucopenia was a very characteristic feature. The *duration* of the acute attack is generally only from 3 to 5 days, but decided debility, anorexia and often neuralgic pain may remain for several weeks. The symptoms in early life are generally not so severe as in adults, but to this there are numerous exceptions. Schiff[2] found the Pirquet reaction absent in all of 64 cases in children, even although returning later.

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[1] *Loc. cit.*

[2] Amer. Jour. Med. Sci., 1920, CLIX, 435.

[3] Med. Rec., 1921, XCIX, 821.

[4] Amer. Jour. Med. Sci., 1921, CLXII, 1.

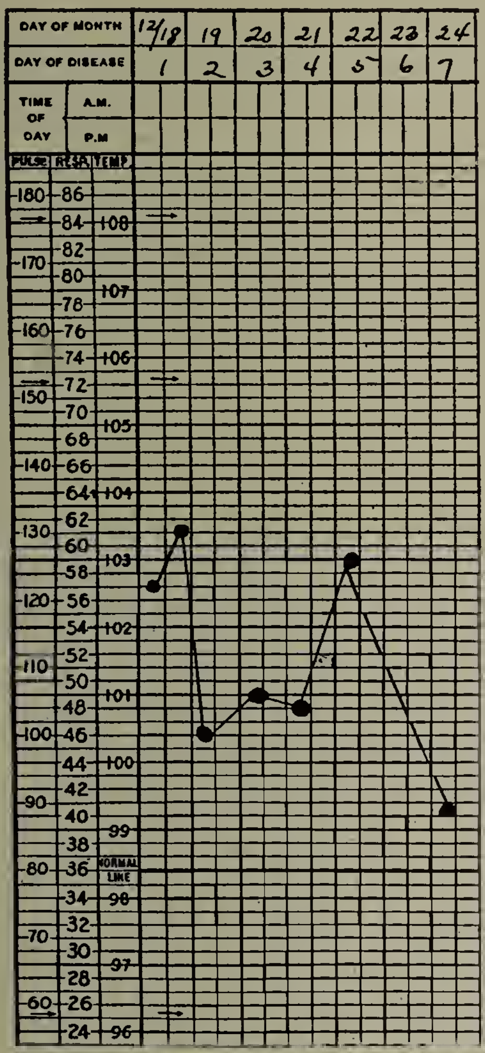

FIG. 145.

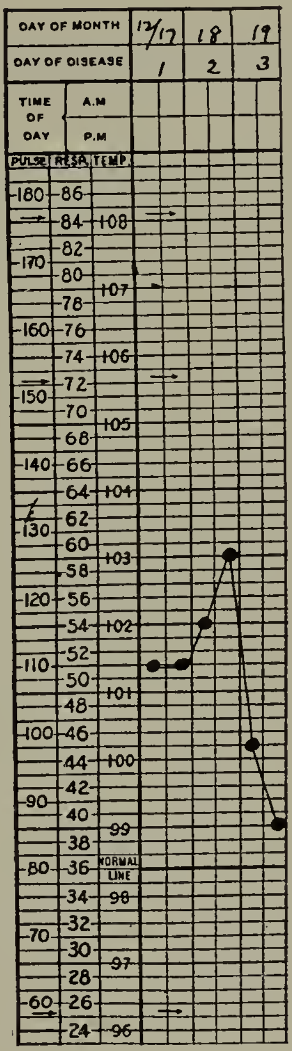

FIG. 146.

FIG. 145.—GRIPPE, TYPICAL FORM.

Anna S., aged 10 months. Cough, running eyes, fever, debility, frequent loose stools, vomited once. Slight preponderance of gastrointestinal symptoms. Epidemic prevailing.

FIG. 146.—GRIPPE, TYPICAL FORM, MILD.

Hannah E., aged 4½ years. Vomited occasionally, headache, slight sore throat, loose cough, no marked prostration. Child bright and very little ill. Epidemic prevailing.

This ordinary type may be either *mild* or *severe*, and between the two extremes all grades of severity are seen. In the *mild cases* local manifestations may be nearly or entirely absent and there may be only a very moderate degree of prostration with slight fever (Fig. 146). The child may not feel ill enough to go to bed, and in a day or two is entirely convalescent. This is a form quite common in early life. In the *severe cases* the temperature may be high or only slightly elevated, but in any event it runs no regular course and is uncharacteristic. The prostration is very decided, appetite is completely lost and some of the localizing symptoms may be marked. Sometimes the course is protracted for weeks or even months, with debility and in older children more or less pain. In infants especially the attack may be very severe, vomiting troublesome, loss of appetite absolute, prostration very great, and respiration rapid. The patient may seem to be overwhelmed by the poison of the disease without any evidence of local lesions and collapse may follow. The severe cases last from a few days up to 2 or more weeks, and convalescence is very tedious (Fig. 147).

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[1] Jour. Amer. Med. Assoc., 1920, LXXIV, 1070.
[2] Orvosi Hetilao, 1917, No. 7. Ref. Monatsschr. f. Kinderheilk., XX. 237.

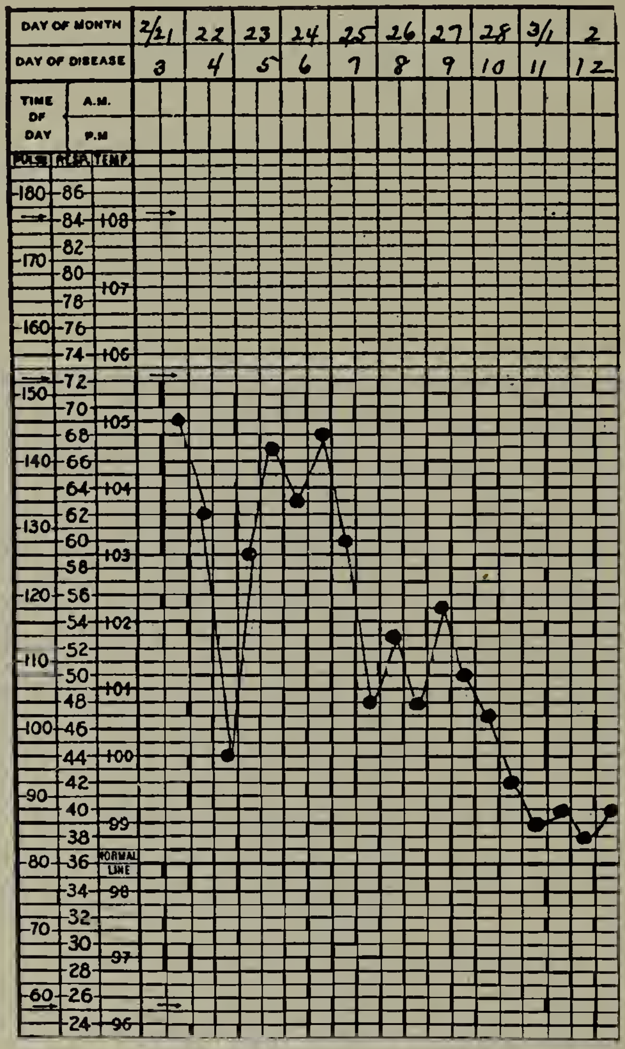

Fig. 147.

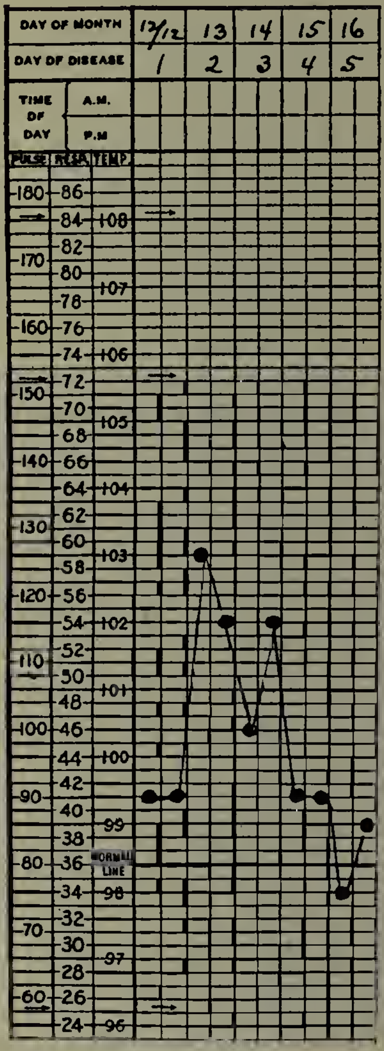

Fig. 148.

Fig. 147.—Grippe, Severe Respiratory Type.

William McC., aged 11 months. Feb. 19, for some days cold in head and moderate bronchitis, no fever; Feb. 21, temperature rose in afternoon, croupy cough; Feb. 22, cyanosis; Feb. 23, many râles, with cyanosis and oppression suggesting asthmatic bronchitis; Feb. 15, seems very ill, cyanosis and râles continuing; Feb. 27, râles nearly gone, general condition improving. Brother ill at the same time with grippe.

Fig. 148.—Respiratory Form of Grippe.

William R., adult. Headache, general aching, severe coryza, harassing cough, congested frontal sinuses requiring local treatment, marked prostration. Decided debility as a sequel.

The different variants from typical grippe depend on the prominence of symptoms of a certain class, and the lesser development of those of other classes. Not infrequently the symptoms of two or more classes may be equally prominent. As a rule the nervous and gastrointestinal forms of grippe are most frequent in infancy and early childhood. The respiratory is that most often seen in later childhood. Yet to this there are numerous exceptions, and all sorts of combinations of symptoms of the different forms may be seen.

Catarrhal or Respiratory Form.—In this form coryza is decided, the pharynx is red, the tonsils swollen with the follicles sometimes engorged; stomatitis may occur; there is annoying cough depending upon involvement of the larynx, trachea and bronchi; and fever and prostration are present to a degree in no way explained by the local manifestations (Fig. 148). The severity of the attack generally diminishes in 3 or 4 days. In severe cases of this type the course is longer and the process readily and rapidly advances to the production of bronchopneumonia which is much more severe, extensive and fatal than in any other group of cases of this disease (Wollstein and Goldbloom).[1] Pulmonary edema and marked cyanosis are prominent features of the toxic cases (Fig. 147). There is also a special tendency to the development of otitis and of cervical adenitis. Sometimes the manifestations of the catarrhal form are unusual and not sufficient to distinguish the disease except in family outbreaks. I have seen adults affected by characteristic grippe and some of the children of the family showing the symptoms of spasmodic croup (*laryngeal form of grippe*). Regan and Regan[2] discuss a severe form of influenzal croup, often with marked obstruction to breathing. In cultures from the patients the influenza-bacillus predominated. Cases with similar symptoms have been repeatedly described by others (Coray;[3] Widowitz;[4] Korsak[5]). In many of the subjects intubation is necessary.

Nervous Form.—In this variety the nervous manifestations predominate and the respiratory and digestive symptoms are less in evidence (Fig. 149). To this class belong a large number of cases in quite early life. There is marked apathy, prostration and loss of appetite. In some cases hyperpyrexia, delirium, stupor or convulsions may occur, so that the case closely resembles meningitis; in others the symptoms of the typhoid state may develop. Still other cases exhibit marked prostration and continued fever, which in the absence of special localizing symptoms at first strongly suggests typhoid fever (*febrile form of grippe*) (Fig. 150). Occasionally there is marked dyspnea of purely toxic origin. Severe cases may sometimes end fatally, although generally in the course of a few days all the alarming symptoms ameliorate, showing that they were toxic and not inflammatory in nature. In other instances actual lesions of the cerebrospinal system develop as complications or sequels. (See Influenzal Meningitis, Vol. II, p. 614.) A very common variety of the nervous type of grippe, especially frequent in younger subjects, is that showing continued fever with moderate prostration, and few distinct local manifestations. Filatow[6] has reported cases of this sort in which irregular fever lasted during 5 months.

Epidemic Pleurodynia.—A peculiar form of acute febrile disturbance, first described by Dabney,[7] and supposed by him to resemble dengue, has since been discussed by Reilly,[8] Payne and Armstrong,[9] Hangar, McCoy and Frantz,[10] Greene[11] and others. It was first called "Devil's grip" and later has received the titles epidemic pleurodynia, diaphragmatic pleurodynia and epidemic transient diaphragmatic spasm.

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[1] *Loc. cit.*

[2] Amer. Jour. Dis. Child., 1919, XVII, 377.

[3] Correspbl. f. Schweiz. Aerzte, 1919, XLIX, 474.

[4] Münch. med. Wochenschr., 1920, LXVII, 1522.

[5] Przeglad Lekarski, 1920, LIX, No. 4. Ref. Monatsschr. f. Kinderheilk., 1921, XXI, 229.

[6] Arch. f. Kinderheilk., 1899, XXVII, 433.

[7] Amer. Jour. Med. Sci., 1888, XCVI, 488.

[8] Jour. Amer. Med. Assoc., 1921, LXXVI, 1493.

[9] Jour. Amer. Med. Assoc., 1923, LXXXI, 746.

[10] Jour. Amer. Med. Assoc., 1923, LXXXI, 826.

[11] Arch. of Pediat., 1924, XLI, 322.

The cause is entirely unknown, but the disease seems to be associated with influenza, the nervous form of which it in some respects resembles; and it may tentatively be classed as one of the variants of this. It appears to be of the nature of an intercostal neuralgia or a diaphragmatic spasm. It is distinctly epidemic in character and occurs chiefly in the late summer months, and cases develop by contact. Sex is without influence, and children are attacked as readily as are adults, or even more so. It begins suddenly with severe pain in the lower part of the chest or upper part of the abdomen, which is made worse by deep breathing or movements of the body. With this there are prostration, headache and fever. The number of leucocytes in the blood is normal, or there may be a leucopenia, but there is a relative increase of the polymorphonuclear cells. Physical examination of the chest is entirely negative. At times there is herpes of the face or trunk. Pains elsewhere in the body are usually absent, and there is little, if any, involvement of the nose and throat. The attack may last but a few hours, but is liable to recur, the whole course being from 2 to 3 days or sometimes a week.

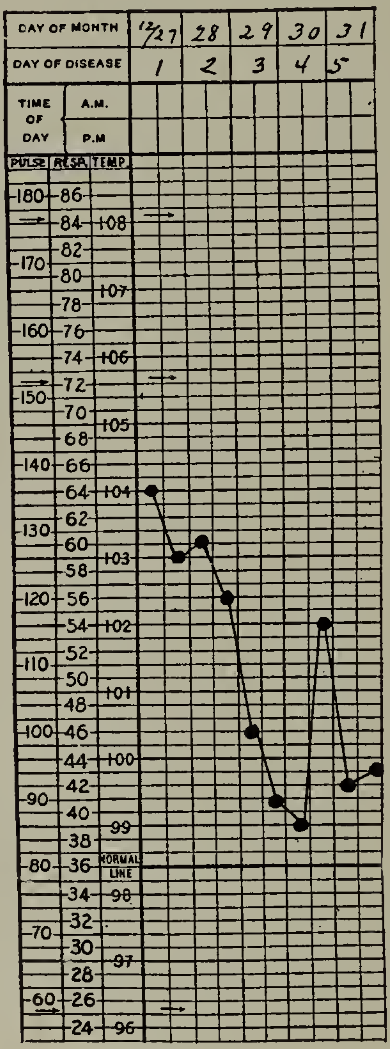

FIG. 149.

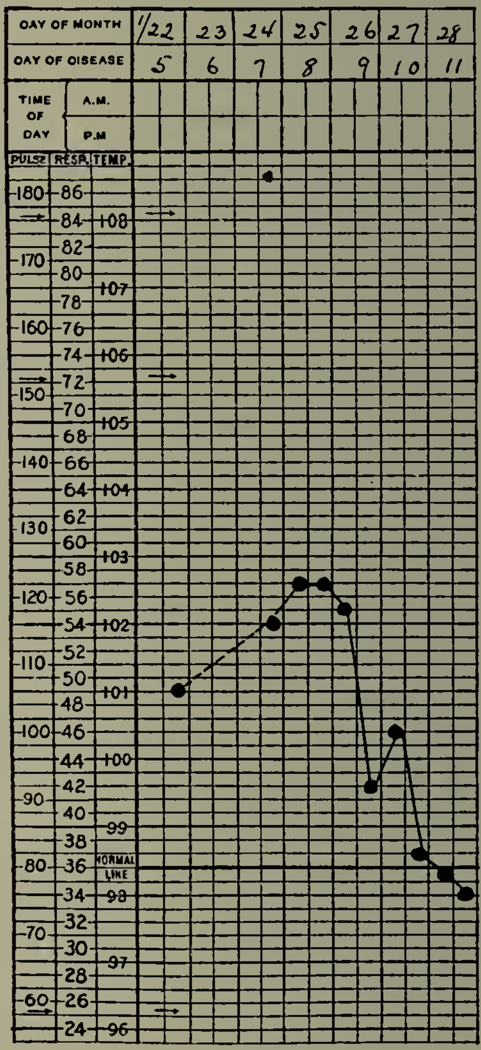

FIG. 150.

FIG. 149.—GRIPPE, NERVOUS FORM.

Helen C., aged 19 months. Pale, prostrated, irregular respiration, very drowsy. No pulmonary symptoms, no diarrhea, vomited only once. Epidemic prevailing.

FIG. 150.—GRIPPE, NERVOUS FORM. PROLONGED FEBRILE TYPE.

Louise F., aged 16 months. Slight cough, apathy, prostration, without any localized symptoms. Fever continued a week or more. Twin sister with same symptoms. Case suggested typhoid fever. Epidemic of grippe prevailing.

GASTROINTESTINAL FORM.—The prominent symptoms of this variety, a common one in infancy, are anorexia nausea, vomiting, abdominal pain, prostration and diarrhea, the stools often containing mucus and blood. The symptoms are those of a severe acute gastroenteritis. The duration of the attack may be only 3 or 4 days, but is not infrequently protracted. In other cases the condition is one resembling intestinal toxemia, and there are present the ordinary symptoms of this, with loss of weight, but without diarrhea or vomiting. This is a common variety in infancy. The intestinal form was much less frequently observed in the 1918 epidemic than in earlier ones. It was of common occurrence in the limited epidemic in the neighborhood of Philadelphia in 1924.

Complications and Sequels.—One of the most dangerous and common complications is *pneumonia.* It was especially frequent in the epidemic of 1918, there being an incidence of about 10 per cent. The form is oftenest a bronchopneumonia, the areas being small and often giving no positive physical signs. Less often it is croupous in nature. The pneumonia of grippe develops either during the attack or as a sequel. The course is irregular, the symptoms being often masked and much more severe than accounted for by the physical signs, and the disease frequently of the wandering type and much prolonged, in which case an incorrect diagnosis of tuberculosis may readily be made. In many other cases it is abortive and lasts only 2 or 3 days. One of the characteristics of the pneumonia in the last epidemic was the tendency often seen for the respiration to exhibit little acceleration; and especially the persistence of leucopenia in spite of the presence of the pneumonic inflammation. (Fig. 151.) Pleurisy not infrequently accompanies the pneumonia and tends to result in empyema. *Bronchitis,* constantly present in the respiratory type of influenza, may be severe enough to constitute an important complication, especially in young children. It then involves the smaller tubules and produces dyspnea and cyanosis. Influenzal croup has already been mentioned as one of the occasional manifestations of the disease (p. 537).

Otitis, purulent, catarrhal, or often hemorrhagic, is a frequent complication especially of the respiratory form of grippe. Either resolution or perforation may occur. Inflammation of the mastoid cells may follow. Affections of the eye are rarely observed except the moderate conjunctivitis which may properly be called a symptom. *Cervical adenitis* is a very striking and common complication. Its frequency varies with the epidemic. It is often out of proportion to the severity of the attack and independent of the presence of affections of the mouth. Resolution is the rule. Parotitis is sometimes observed. Neuritis of various forms may develop as a sequel, although infrequently in children. *Meningitis* depending upon the influenza-bacillus or other germs, may accompany the attack, or occur without other symptoms. Adams[1] reported a case, and collected 20 others from literature, in which the bacillus of influenza was found in the cerebrospinal fluid or in the meninges. I have seen several such instances, in none of which, however, could the condition properly be called a complication of grippe. Numerous other instances of meningitis of influenzal origin have since been reported. (See Influenzal Meningitis, Vol. II, p. 614.) Mental disturbances, spastic paralysis, chorea, encephalitis and other nervous affections exceptionally follow grippe. Nephritis, cystitis and pyelitis are rare. Miller[2] collected 40 recorded cases of acute hemorrhagic *nephritis* occurring as a complication. Only a few of these were in children. Peritonitis has been reported as a complication or sequel. Ileocolitis may develop in the gastrointestinal form of the disease and icterus is sometimes witnessed. Anemia, at times severe, is a frequent sequel. Cardiac complications are few in children, except the tendency to dangerous collapse occasionally observed, dependent at times on acute cardiac dilatation. *Cutaneous complications* are interesting and not rare, especially herpes, urticaria, petechiæ and an erythema which may be either morbilliform or oftener scarlatiniform.

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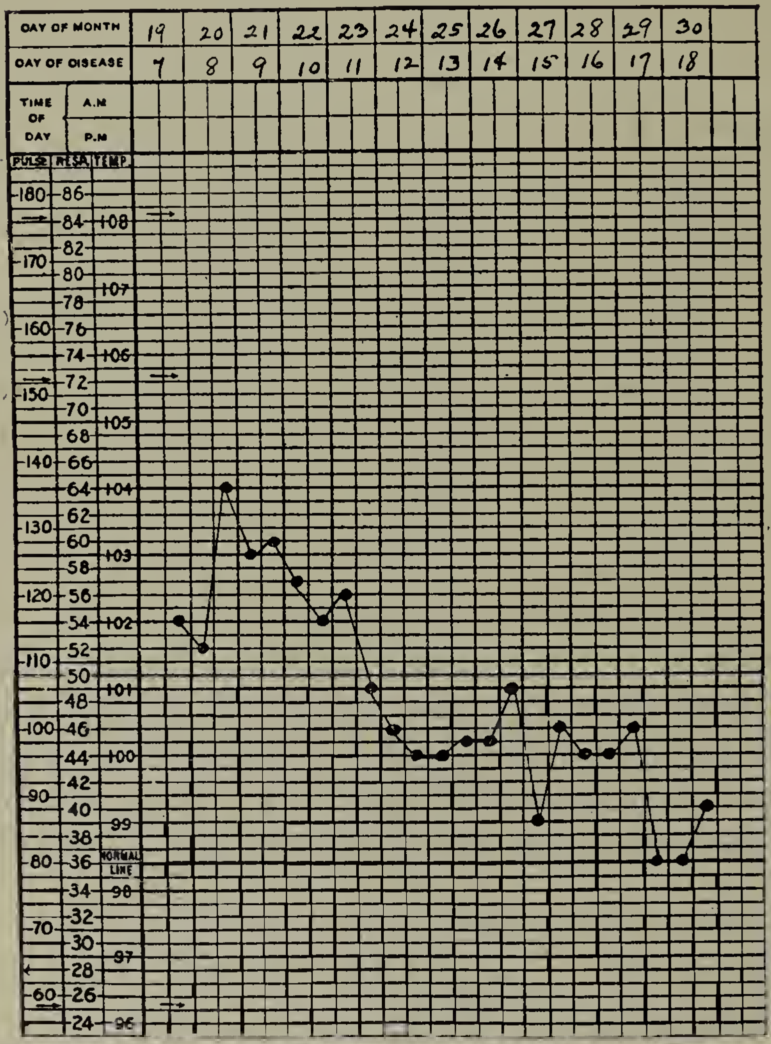
[1] Arch. of Pediat., 1907, XXIV, 721.
[2] Arch. of Pediat., 1902, XIX, 1.



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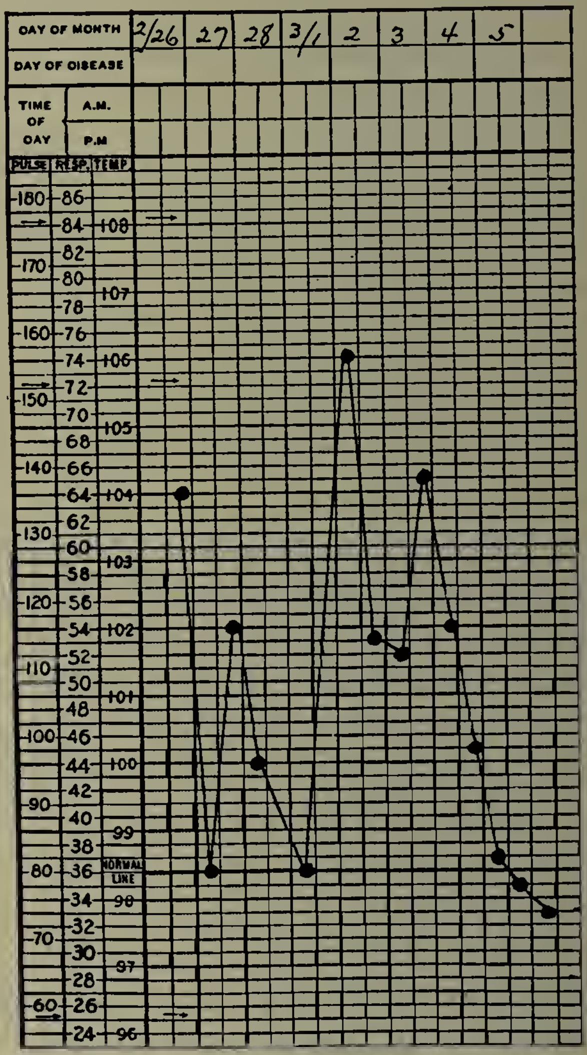

FIG. 152.

FIG. 151.—GRIPPE FOLLOWED BY PNEUMONIA WITH LEUCOPENIA.

Theodore N., aged 3 years. Admitted Oct. 19th, 1918, to the Children's Ward of the Hospital of the University of Pennsylvania. Father and mother and one of the other children of the family ill with grippe. Child developed fever and cough about a week before admission. Examination showed fully developed signs of consolidation, lower part of both lungs; child very toxic and debilitated. Blood on Nov. 4th, gave 7,600 leucocytes. Gradual recovery.

FIG. 152.—GRIPPE, RESPIRATORY FORM WITH PROMPT RELAPSE.

Josephine V., aged 6 years. Feb. 26, slight sore throat, high fever; Feb. 28, better, considerable cough; Mar. 1, apparently convalescent; Mar. 2, throat painful, respiration accelerated, cough, high fever, severe coryza; Mar. 4, numerous râles, in other respects better. Mother and brother ill with grippe.

Other infectious diseases may be associated with grippe, among those reported being erysipelas, mumps, scarlet fever, measles, pertussis and diphtheria. The development of tuberculosis as a sequel is not infrequent.

Relapse and Recurrence.—*Relapse*, in the sense of relighting of symptoms before the disease is thoroughly over, is exceedingly common. It develops a few days after symptoms have subsided, possibly after the patient has been taken into the open air, and may equal or exceed in severity the primary attack (Fig. 152).

Recurrence is common. Protection given by one attack of grippe lasts only a few weeks or months, sometimes longer. The large number of individuals developing a second attack a year or less after the first one shows the uncertainty of immunity. In fact, those who have once suffered appear often especially predisposed later.

Prognosis.—The mortality of uncomplicated grippe has been low, statistics varying from 0.5 per cent. to somewhat over 1 per cent. Nevertheless, when we consider the enormous frequency of the disease, the actual number of deaths from grippe or from its complications has reached a surprising figure, and this is particularly true of infancy. Thus, in London alone from January, 1890 to March, 1892, 4740 persons died from it (Leichtenstern).[1] It has been estimated that 6,000,000 persons throughout the world died of it in the epidemic of 1918–19. As a rule there is little danger in uncomplicated cases in previously healthy subjects. The chief danger of the disease lies in the complications, especially gastrointestinal disorders and pneumonia, the latter being the cause of the majority of deaths. In the 1918 epidemic the mortality was very high, the cause of the fatality apparently being a pneumonia depending upon a hemolytic streptococcus. In 46 of the largest cities of the United States[2] with a combined population of 23,000,000, the number of deaths from grippe from September 8th to November 9th equalled 82,306, chiefly with pneumonia. The normal number of deaths from these causes for the same period would have been about 4000. In Philadelphia[3] during 7 weeks of the epidemic 12,687 deaths were reported from influenza and pneumonia; the number of deaths from the latter disease in the previous year having been only 293. These statistics apply to individuals of all ages. The mortality in childhood as a whole is decidedly less than later in life, although in the 1st year there is a greater tendency to complications, and consequently a greater danger from the disease, than in the years immediately following.

Diagnosis.—This is easy in typical cases during an epidemic. Under other circumstances it may be difficult or impossible. Prostration, which is present in practically every case except the very mildest, and which is out of proportion to any discoverable cause, is the most important diagnostic symptom. Fever, too, not satisfactorily accounted for in any other way, is another characteristic, as is the pain in many cases. The fact that the mucous membrane of more than one region is affected is also suggestive.

Mild isolated cases of the respiratory type cannot be distinguished from ordinary catarrh of the respiratory tract; the severer ones exhibit too great prostration and fever, and in general are more ill than a simple catarrh explains. The presence of cervical adenitis, too, is often suggestive. Cases of the nervous type may present cerebrospinal symptoms indicating meningitis. The diagnosis is impossible at the beginning, but rapid improvement in the course of a few days, with the development of the more ordinary evidences of grippe make the matter clear. In this connection must be emphasized the necessity of caution in making the diagnosis even of a complicating meningitis, unless this is done by means of lumbar puncture. The pleurodynic form may readily simulate pleuro-pneumonia or pleurisy, but is distinguished by the absence of any physical signs of these diseases and by the epidemic incidence. The protracted febrile cases of influenza without special localized manifestations may be difficult to distinguish from typhoid fever. The suddenness of the onset, the frequent presence of catarrhal symptoms of the nose and eyes, and the absence of the typhoid serum-reaction and of roseola aid in distinguishing it. Enlargement of the spleen may be present in either disease, but is more constant in typhoid fever.

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[1] Nothnagel, Encyclopedia of Pract. Med., Amer. Edit., Influenza, 569

[2] Bureau of the Census. Ref., Med. Rec., 1918, XCIV, 906.

[3] Monthly Bull., *loc. cit.*, 24.

Scarlet fever, measles, and especially pneumonia all bear resemblance to grippe at the beginning. I have seen the erythema which sometimes complicates grippe add to the difficulty in diagnosing between grippe and scarlet fever. The course of the case and the absence of typical tongue, typical desquamation and leucocytosis in grippe serve to distinguish them. In cases of real doubt the special tests for scarlet fever may be used (Regan).[1] Malaria exhibits usually a more regular temperature-curve, the spleen is decidedly enlarged, and the malarial organism can be found in the blood.

Gastroenteritis at first resembles exactly the gastroenteric type of grippe. The sudden clearing up of the symptoms will distinguish the latter in the course of a few days. The existence in several members of a family of fever with decided enlargement of the cervical lymphatic glands, an occurrence which has frequently been observed in influenza, sometimes causes the diagnosis of glandular fever to be made. Unquestionably such mistakes are common. The prevalence of grippe and the presence of undoubted symptoms of it in combination with the glandular enlargement, will generally make the diagnosis evident.

Treatment. **Prophylaxis.**—The great contagiousness of the disease renders necessary the protection of those not affected, particularly infants and delicate children. This is, however, difficult of accomplishment during epidemics. Certainly the ill child should be isolated as far as possible. It is still better to remove the other children from the house. The wearing of masks by those in attendance should in theory restrict the spread of the infection to them; but the experiments of Lauterburg[2] showed that these are readily penetrated by bacteria (See also p. 346). Quarantine should continue while evidence of respiratory catarrh persists, since living microörganism are probably still present in the secretion. The value of prophylactic vaccines is still undetermined in spite of the careful study which has been given to the subject, and the wide trial of vaccines which was made in the 1918 epidemic.

Treatment of the Attack.—The brief course of the uncomplicated disease and the low mortality usually renders little treatment necessary. There is no specific known and therapy must be symptomatic. What has been said of vaccines for prophylaxis applies equally well here. Rest in bed is imperative. The diet should be light, nourishing and abundant. Fever may be treated by ordinary febrifuges and a laxative, or if high, by sponging or a warm bath. Pain may be relieved by small repeated doses of the coal-tar derivatives, preferably phenacetin and antipyrine, given cautiously. (See pp. 257, 258 for dosage.) Vomiting may be checked by free opening of the bowels and by the administration of bismuth, lime-water, bromides or, possibly, opiates. Antiseptic alkaline sprays to the nasal mucous membrane are of advantage. Alcoholic stimulants or strychnine are needed if there is much prostration. Harass-

[1] Arch. of Pediat., 1924, XLI, 47.

[2] Correspnbl. f. Schweiz. Aerzte, 1919, XLIX, 1786.

ing cough may require sedative treatment. Cerebral symptoms may need bromides, coal-tar preparations or opiates. Warm baths combined with cold to the head may prove useful under these conditions.

Complications must be guarded against as far as possible. Exposure to chilling by too early leaving the bed or the room may precipitate a relapse or predispose to pneumonia. Keeping the ears carefully covered during the attack may tend to prevent an otitis. Complications developing should receive the treatment appropriate to them. In the severe fulminating cases with broncho-pneumonia, pulmonary edema and cyanosis, in which there is marked blood-concentration, Underhill and Ringer[1] have had some success with carefully controlled venesection and fluid injections in keeping the blood at the normal concentration-level. Very good results are reported by Willmore and Gardner-Medwin[2] in severe cases with pulmonary complications by stimulating leucocyte-production with hypodermic or intramuscular injections of sodium nucleinate, combined with alkalis by the mouth, subcutaneously, or even intravenously.

In the cases where the convalescence is slow and the remaining debility persistent, tonic treatment, careful and abundant diet, and often change of climate may be indicated.

CHAPTER XIV

PERTUSSIS

(Whooping-cough)

History.—The first published recognition of pertussis was by Baillou[3] in Paris in 1578. Willis[4] observed it in England in 1658 and described it clearly. After the middle of the 18th century the disease spread widely and is now one of the commonest of the acute infectious disorders over the whole civilized world.

Etiology. **Predisposing Causes.**—Climate, race and geographical position exert no influence, and that of sex is immaterial, females appearing slightly more predisposed. The statistics regarding season are at variance, but on the whole pertussis would appear more frequent in the colder months, possibly through the more intimate association of children in schools at this time. The statistics of Luttinger,[5] however, upon 6868 cases and of Herrman and Bell[6] in 297 cases showed a greater prevalence during the spring and summer. Poor hygienic conditions favor the dissemination of the disease, but predispose in no other way. The previous state of health is an etiological factor to some extent, weakly and sickly children apparently contracting pertussis more readily than others. The existence of other diseases does not prevent its development, and it is certain that outbreaks of pertussis are especially liable to accompany or to prevail after epidemics of measles or influenza, although uncertain whether there is an actual etiological relationship.

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[1] Jour. Amer. Med. Assoc., 1920, LXXV, 1531.

[2] Lancet, 1922, I, 116.

[3] Geneva Edition, 1762, I, 173.

[4] Path. Cerebri, etc., Cap. XII, 1667.

[5] Amer. Jour. Dis. Child., 1916, XII, 290.

[6] Arch. of Pediat., 1924, XLI, 13.

Age exercises a powerful predisposing influence. The majority of cases occur under 6 and comparatively few after 10 years. Szabó[1] reports 1028 cases (24.58 per cent.) in the 1st year out of a total of 4181. Vladimirov[2] gives the incidence of the disease in 4623 cases as follows:

TABLE 80.—INCIDENCE OF PERTUSSIS AS REGARDS AGE

Under 6 months	5.5 per cent.
6–12 months	11.1 per cent.
1– 2 years	18.1 per cent.
2– 3 years	14.2 per cent.
3– 4 years	11.6 per cent.
4– 5 years	9.1 per cent.
5– 6 years	7.9 per cent.
6– 7 years	6.1 per cent.
7– 8 years	4.7 per cent.
8– 9 years	4.1 per cent.
9–10 years	3.3 per cent.
10–11 years	1.7 per cent.
11–12 years	1.3 per cent.
12 years and over	1.3 per cent.

Baginsky[3] found in 2651 cases in children 830 (31.3 per cent.) from birth to 1 year, 1308 (49.4 per cent.) from 1 to 4 years, 502 (18.2 per cent.) from 4 to 10 years, and 11 (0.4 per cent.) from 10 to 14 years. Finally in an analysis by Luttinger[4] of 10,000 cases the age-incidence was: Under 1 year 19.4 per cent.; 1 to 2 years 20.1 per cent.; 2 to 5 years 40.1 per cent.; 5 to 15 years 17.9 per cent.; 15 years and over 2.3 per cent.

It is evident that the greater immunity of the 1st year seen in some other infectious diseases does not obtain to any extent in pertussis. Even cases of congenital pertussis have been reported (Rilliet and Barthez;[5] Gatti;[6] Cockayne;[7] Milio[8]), or it may be contracted within a few days after birth (Phillips).[9]

Apert and Cambessédès[10] after reviewing various statistics maintain that the disease is more prevalent in the female *sex*.

The *individual susceptibility* is very great and the majority of children exposed contract the disease. That it is comparatively uncommon in adults depends largely on the fact that so many have already suffered earlier in life. Some children, however, possess a natural immunity. Epidemic influence is marked, many more cases occurring in some years than in others. The disease is practically endemic in larger cities.

Exciting Cause.—Pertussis has been variously described as a pure neurosis of the medulla or of the nerves which control cough, a simple bronchial catarrh, a pneumogastric irritation from the pressure of enlarged bronchial lymphatic glands, and as the result of sensitization to pollen (Ward).[11] Its evident infectiousness, however, indicates that it is certainly dependent upon the action of some microbe contained in the respiratory secretions. Very minute bacilli found in the respiratory mucus were reported by Afanassjew,[1] Czaplewski and Hensel,[2] Arnheim,[3] Koplik,[4] Jochmann and Krause[5] and others. In 1906, Bordet and Gengou[6] described a very small, ovoid, Gram-negative bacillus present in large numbers in the sputum in the early part of the attack. It resembles, but is quite distinct from, the influenza-bacillus. The discovery has been confirmed by many later investigators, and the etiological relationship of the germ to pertussis is rendered still more positive by agglutination and complement-fixation tests; and by the claims (Klimenko;[7] Inaba[8]) that with it the disease could be transmitted to apes and to puppies (Mallory, Horner and Henderson).[9] During the first 2 weeks it is present in the secretions from the nose and throat in from 75 to 90 per cent. of the cases according to Herrman and Bell.[10] While it is very probable that pertussis is due to this germ, further studies are needed, since the agglutination and complement-fixation tests do not always respond positively; there are probably several different strains of the bacillus; the germ may be found in other conditions certainly not pertussis (Fränkel);[11] and cases at least clinically pertussis are repeatedly encountered in which no bacterial cause can be discovered, or other bacteria are present, especially those belonging to the group of the influenza-bacillus. The *duration of life* of the term outside of the body is probably brief. Rooms occupied by the patient soon lose their infectiousness.

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[1] Pest. med.-chir. Presse, 1881, XVII, 657.

[2] Bolnitch, Gaz. Botk., 1893, No. 12. Ref. O'Dwyer and Norton, 20th Century Practice of Med., XIV, 217.

[3] Kinderkrankheiten, 1905, 265.

[4] *Loc. cit.*

[5] Sanné, Mal. des enf., 1891, III, 747.

[6] La Pediat., 1914, XXII, 687.

[7] Brit. Jour. Child. Dis., 1913, X, 534.

[8] La Pediat., 1922, XXX, 297.

[9] Amer. Jour. Med. Sci., 1921, CXVI, 163.

[10] Bull. et mem. de la soc. méd. des hôp., 1920, XLIV, 324.

[11] Arch. of Pediat., 1922, XXXIX, 730.

Nature of the Disease.—The mode of action of the infectious agent is uncertain. The studies of Meyer-Huni[12] and of von Herff[13] indicate that the seat of irritation is oftenest the nose, larynx and trachea, but especially the inter-arytenoid fossa on the posterior wall of the larynx—the so-called "cough region." It is probable, however, that the situation of the local irritation upon the respiratory mucous membrane may vary to some extent with the case. As a result of this local irritation a paroxysm of cough is produced by the accumulated mucus upon the sensitive region. In this way is set going a series of reflex clonic spasms of the respiratory muscles, the whoop being due to an inspiratory spasm of the glottis. The process is repeated until the offending mucus is expelled. From this point of view the disease is chiefly a local disturbance produced by an infectious catarrhal process. It seems, however, an unavoidable conclusion that, in addition to the local irritation which precipitates the paroxysms, there exists a general constitutional disorder which determines the peculiar character of these, and that this character may depend upon a disturbance of the superior laryngeal nerves and the respiratory centres in the medulla, brought about by a toxin produced by the germs and circulating in the blood. This view is supported by analogy to other infectious diseases; the production of attacks of cough by excitement and other disturbances acting reflexly; the greater frequency of the cough at night, indicating lesser resisting power of the respiratory centres in the medulla; the occasional occurrence of congenital pertussis, which must necessarily be a blood-disorder; the much increased excitability of the general nervous system which is always present; the occurrence of an agglutinative reaction; etc.

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[1] Petersburg med. Wochenschr., 1887, IV, 323.
[2] Deutsch. med. Wochenschr., 1897, XXII, 586.
[3] Berl. klin. Wochenschr., 1900, XXXVII, 702.
[4] Brit. Med. Jour., 1897, II, 1051.
[5] Zeitschr. f. Hyg., 1901, XXXVI, 193.
[6] Annal. de l'instit. Pasteur, 1906, XX, 731.
[7] Deutsch. med. Wochenschr., 1908, XXXIV, 2030.
[8] Zeitsch. f. Kinderheilk., Orig., 1912, IV, 252.
[9] Jour. Med. Res., 1913, XXVII, 391.
[10] *Loc. cit.*
[11] Münch. med. Wochenschr., 1908, LV, 1683.
[12] Zeitschr. f. klin. Med., 1880, I, 461.
[13] Deutsch. Arch. f. klin. Med., 1886, XXXIX, 392.

Period of Greatest Infestiousness.—This is not certainly determined. There is no question but that the disease is transmitted early in the catarrhal stage, and perhaps most decidedly at this time. There is also no doubt, on the other hand, that it sometimes can be transmitted in the paroxysmal stage and even during the decline. Yet in most cases it is probable that the danger of infection is less in the paroxysmal stage, and disappears entirely before this is over. Weill[1] considers infectiousness as past within at most 1 month after the first cough begins. Based upon numerous studies on the persistence of the bacilli, Meyer[2] believes that infectiousness persists during the first 3 or 4 weeks of the whoop, and Lereboullet[3] views 4 to 5 weeks as the most infectious in the entire period of the cough.

Mode of Transmission.—This is direct by the secretions of the respiratory tract and to a certain extent by the breath, the germs being contained in minute droplets of mucus expelled by coughing. Conveyance to any distance by the air does not appear to occur. Only exceptionally, if ever, is the infection carried by a third person; and it is very probable that in most of such supposed cases the disease has in reality been communicated by someone suffering from an abortive and unrecognized attack. The germs are received through the respiratory tract, the congenital cases of pertussis being the exception.

Pathological Anatomy.—There are no characteristic lesions. During life there is found an intense redness and swelling of the respiratory mucous membrane, especially of the larynx and trachea, and to a less extent of the nose and pharynx, with the secretion of viscid mucus. At autopsy involvement of the bronchial mucous membrane may also be discovered, and very constantly some degree of pulmonary emphysema. Mallory and Horner[4] found the Bordet-Gengou bacillus between the cilia of many of the cells of the mucous membrane of the trachea and bronchi. Acute bronchiectasis, enlargement of the bronchial and tracheal glands, and congestion of the brain, lungs and other internal organs are often found. Some of the lesions of complicating conditions are generally present in fatal cases, among these being bronchopneumonia, atelectasis, cerebral hemorrhage, hypertrophy and dilatation of the heart, especially the right ventricle, and hemorrhages in various organs, especially the brain.

Symptoms.—The attack is usually divided into 4 periods: (1) the incubation; (2) the invasion or the catarrhal stage; (3) the paroxysmal, spasmodic, or convulsive stage; (4) the stage of decline. These are not very sharply differentiated and vary greatly in length in different cases.

Incubation.—Owing to the insidiousness of the invasion the exact *duration* of incubation is difficult to determine, but it probably varies in most cases from 7 to 14 days; sometimes shorter, rarely longer. Funck-Hellet[5] after careful study, places it at 9 to 11 days. No symptoms are present.

[1] La Médecine, 1921, II, 841.
[2] Acta pædiat., 1921, I, 99.
[3] Paris méd., 1919, XXXI, 44.
[4] Jour. of Med. Res., 1912, XXVII, 115.
[5] Arch. de. méd. des enf., 1925, XXVIII, 94.

The Catarrhal Stage.—The attack begins with the symptoms of a tracheo-bronchitis, often with some degree of coryza, sneezing, hoarseness and pharyngeal irritation. Slight elevation of temperature with malaise and irritability may be present at the beginning and last a few days. The cough at first is in no way suspicious, but later becomes hard, dry and annoying. In typical cases the chest reveals few if any râles, but sometimes an attendant bronchitis obscures the symptoms and may be the cause of continued fever. The cough gradually becomes more severe, frequent, and paroxysmal in character, and attacks are especially prone to occur in the night. Finally, distinct whooping develops and the paroxysmal stage may be said to have fairly begun. The condition of the blood will be referred to later.

The *duration* of the catarrhal stage is extremely variable. Some children begin whooping after 2 or 3 days, others only after 3 or 4 weeks, or not at all. In general it averages about 2 weeks, but the younger the child the shorter this period.

The Paroxysmal Stage.—The beginning of this period is usually dated from the commencement of whooping. A typical paroxysm is very characteristic. Often it comes on without warning, but often, too, the child experiences a slight tickling in the throat or beneath the sternum, an inclination to cough, or a sensation of smothering or of intense anxiety. If previously lying down it sits upright with an anxious expression and perhaps grasps the side of the crib. If moving about it drops its toys and runs to its mother or nurse, or takes hold of some of the furniture of the room. There is a brief moment of holding the breath, a deep inspiration follows, and the attack begins. This consists of a series of short explosive coughs, so rapidly repeated that there is no time for respiration between them. These continue for a few moments and number anywhere from 4 or 5 up to 15 or 20. Meanwhile the face becomes swollen, red, cyanotic, and sometimes quite dark and the eyes prominent and congested; the tongue protrudes with each expiratory effort; tears flow from the eyes and saliva from the mouth; the veins of the neck are engorged; perspiration breaks out on the face, and the pulse increases in frequency. Finally the cough ceases and the respiration often apparently also, but in a moment a long-drawn, crowing inspiration is heard which is called the "whoop," and depends upon a spasm of the glottis. Immediately after the whoop a second attack generally occurs and after this perhaps a third or fourth, or more. The whole paroxysm lasts from a few seconds up to several minutes. In the latter case there may be a momentary period of rest between some of the attacks. Toward the end of the paroxysm very tenacious, ropy mucus is often driven from the mouth by the force of the cough and this seems to bring relief, and retching or vomiting is liable to follow.

The paroxysms vary from 6 or 8 up to 60 or more in 24 hours, not all of them in severe cases being equally marked. They are generally most troublesome at night. They occur without discoverable reason, or are brought on by such slight causes as excitement, crying, swallowing, exercise, sudden change of air, inhalation of the air of close rooms, the use of a tongue-depressor, hearing another child in a paroxysm, and the like. The presence of mucus on some part of the irritated mucous membrane is the commonest immediate cause. Auscultation of the chest during the expiratory efforts reveals no respiratory sound and only the impulse of the cough against the ear. During the whoop only a feeble inspiration is heard or none at all. In severe cases the urine and feces may be involuntarily expelled and hemorrhage take place from the nose or mouth or beneath the conjunctiva.

More or less fatigue, usually of brief duration, may follow the paroxysm. In severe cases the child may be covered with perspiration after an attack, confused, and quite exhausted. Between the paroxysms a characteristic appearance of the face is often seen, consisting of some degree of swelling and cyanosis, congestion of the eyes, and blueness of the tongue.

The urine in pertussis may contain albumin in severe cases. A slight blowing apical murmur may be heard, and on percussion the cardiac dullness may be increased (Koplik).[1]

The *blood* as studied by Fröhlich,[2] and later by Crombie,[3] Kolmer,[4] McGay,[5] Hess[6] and others, shows a remarkably high leucocytosis, averaging 20,000, and often reaching higher figures, which begins early in the catarrhal stage and reaches its maximum at the height of the paroxysmal stage. We have several times seen the total leucocyte count rise to over 50,000, occasionally to 80,000 or 90,000, and in one instance to 233,600, this being in an infant of 4 months of age in the 3rd week of the disease. Bourne and Scott[7] report an instance in an infant of 21 months with a leucocyte-count of 176,000 on the 16th day of the disease; Seitz[8] the case of a boy of 4 years, in which, however, there had never been actual whooping, with a leucocyte-count of 192,000, and Crombie[9] a count of 233,000 in an infant of 20 months, who, however, was suffering also from pneumonia. The increase is seen in the lymphocytes, which may reach 80 to 90 per cent. or more; the neutrophiles being relatively diminished. The condition of the blood returns to normal in 2 or 3 months, the neutrophiles gradually increasing and the lymphocytes diminishing. The eosinophiles are diminished (Benstz).[10] In the severest cases the leucocytosis is greater, while the lymphocytes are fewer and the neutrophiles more numerous than in those of average severity (McGay; Crombie). Complications also increase the neutrophilic percentage. Regan and Tolstouhov[11] find that there occurs, beginning in the catarrhal stage, a diminution of the inorganic phosphorus of the blood and an uncompensated acidosis (lowered hydrogen-ion concentration) which is due to the accumulation of free carbon dioxide in the blood.

The severity of the paroxysmal stage increases for about 2 weeks, and then continues for a time unabated. Meanwhile the general nutrition and strength are but little affected in average cases in previously healthy children; the appetite and digestion are good, and there is no fever unless complications are present. In severe cases, however, the child may suffer greatly from lack of sleep and loss of food by vomiting, and emaciation and debility may become extreme. As the disease advances a few moist râles become audible between the paroxysms.

[1] Transac. Amer Pediat. Soc., 1893, V, 90.
[2] Jahrb. f. Kinderheilk., 1897, XLIV, 53.
[3] Edinb. Med. Jour., 1908, I, 222.
[4] Amer. Jour. Dis. Child., 1911, I, 431.
[5] Cleveland Med. Jour., 1911, X, 571.
[6] Zeitschr. f. Kinderheilk., Orig., 1920, XXVII, 117.
[7] Brit. Med. Jour., 1922, I, 387.
[8] Amer. Jour. Dis. Child., 1925, XXX, 670.
[9] *Loc. cit.*
[0] Nederl. Tijdschr. v. Geneesk., 1916, LX, 153.
[11] Jour. Amer. Med. Assoc., 1926, LXXXVI, 1116.

The *duration* of the paroxysmal stage is very variable. It averages 3 to 6 weeks, but may be much longer, while in the mildest cases this stage may continue without change for not more than a week.

This description of the typical spasmodic stage does not apply to all cases. Sometimes the paroxysms are so mild or so infrequent that the child is little disturbed or perhaps needs relief only during the night, if at all. In some cases no whooping occurs at any time and the other symptoms are but little marked. It is likely that these very mild cases are often unrecognized and are one means of the dissemination of the disease. In others, especially in the 1st year of life, the whooping may not occur in the milder cases, or in the severer ones may be replaced by dangerous apnea, with unconsciousness. In some instances the attack may be accompanied by violent repeated sneezing, and exceptionally this may entirely replace the cough (Szegö).[1] The paroxysmal character of the second stage may also be influenced by complications. It may, for instance, disappear completely for a time if pneumonia develops.

Stage of Decline.—This stage is naturally not sharply separated from the preceding one. Its beginning is marked by a lessening in the number and frequency of the paroxysms. This steadily continues, the cough growing looser and losing more and more its peculiar character, while the whooping follows only some of the attacks. Moist râles are heard in greater numbers in the chest and the sputum is more purulent. Finally whooping ceases entirely and the disease is over, although more or less cough may continue for an indefinite time. The *duration* of this stage is even more variable than that of the others. In general it may be said to average from 2 to 3 weeks. It may, however, be indefinitely prolonged by the development of a slight bronchitis, which may cause a return of the severity and an increase in the number of paroxysms. Consequently during the winter-season the paroxysmal stage is liable to be of long duration.

It not infrequently happens that the whooping returns after it has ceased entirely for a short time. This cannot properly be called a continuation of the disease, but is rather now a pure neurosis without any infectious element. The actual termination of the attack of pertussis in average cases may generally be placed at the time when the whooping and the paroxysmal character of the cough have entirely disappeared for a number of days. The total duration of the disease thus equals from 6 weeks to several months.

Complications and Sequels.—*Respiratory* complications and sequels are the most frequent. A moderate bronchitis is a symptom of the stage of decline. If it is unusually severe or develops early in the attack, it is to be regarded as a complication. It is serious if involving the small tubes, or when occurring in the 1st year of life. The most common and one of the most dangerous complications is bronchopneumonia. The younger the child the greater the predisposition to it. It is also especially frequent in feeble or rachitic subjects. It makes its appearance oftenest during the height of the convulsive stage or later. During its presence the whooping character of the cough is liable to lessen or disappear. The proportion of cases of pertussis attacked by bronchopneumonia varies. Of 1731 cases in the Metropolitan Asylums Board's Hospitals in 1912, 10.43 per cent. developed bronchopneumonia (Rolleston).[2] It has reached even as high as 33.3 per cent. (Sée).[3] Atelectasis is of common occurrence in severe cases in infants. A moderate degree of pulmonary emphysema is probably always present, and occasionally remains as a sequel. Pneumothorax and pulmonary abscess have been reported, and croupous pneumonia and pleurisy with serous or purulent effusion are occasionally seen.

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[1] Arch. f. Kinderheilk., 1900, XXIX, 186.

[2] Brit. Jour. Child. Dis., 1914, XI, 38.

[3] Arch. gén. de méd., 1854, II, 279.

Of *digestive disorders* the most troublesome complication is vomiting, which may be so severe and frequent after the paroxysms that great emaciation and loss of strength develop rapidly. Loss of appetite, indigestion, and diarrhea are very often observed in infants, especially in summer, when ileocolitis also is liable to occur. Prolapse of the rectum or hernia may be produced by the violence of the cough, or ulceration of the frænulum linguæ from the repeated impinging of the tongue against the lower incisor teeth. Stomatitis is common in severe cases.

Nervous complications and sequels are frequent, among the most important being general convulsions, which not rarely end fatally. They are oftenest seen in rachitic infants, but occur also in the course of pneumonia or as an indication of intracranial hemorrhage. Some of them probably depend upon a serous meningitis (Reiche)[1] but others, according to our own experience, are certainly spasmophilic in nature; and this is also the opinion of Powers.[2] Convulsions are most liable to develop in severe cases, but it frequently happens that fatal convulsions attack without warning an infant who has been apparently little ill. They may occur in any period of the disease. Spasm of the glottis is another convulsive complication sometimes fatal. Aphasia, blindness, deafness and various psychoses are occasional sequels. They are generally of temporary duration, but sometimes permanent. Coma or a soporose condition may occur as a result of intracranial disturbance. Different forms of paralysis, temporary or lasting, are not infrequent sequels. They have been studied with especial care by Valentin[3] and by Hockenjos.[4] In the majority of instances the paralysis is central in origin and of a hemiplegic type, or occasionally monoplegic or paraplegic, and depends upon cerebral hemorrhage following the violent congestion; or, in cases which recover, sometimes upon temporary passive congestion and edema. Myelitis and multiple neuritis are uncommon. Disseminated sclerosis has been reported as a sequel. Strümpell[5] records a case of optic neuritis combined with cerebellar ataxia.

Of *cardiovascular complications* one frequently seen is dilatation of the heart, especially the right ventricle, which occurs in severe cases (Koplik).[6] Degenerative changes in the cardiac muscle aid in producing it, according to some authors, although such changes have been found at necropsy usually only in complicated cases. Sudden death may result. Ledbetter and White,[7] however, after an analysis of the literature and a study of 232 cases, maintain that there is no evidence of damage to the heart except a temporary mechanical strain, particularly of the right side. Hemorrhages are of frequent occurrence, dependent upon the intense passive congestion which the violent coughing occasions. Epistaxis is the most common form and may occasionally be severe enough to require treatment. Discharge of blood from the mouth is oftenest dependent upon epistaxis, the blood having sometimes been swallowed and then vomited. Hematemesis, the result of gastric hemorrhage, is unusual, as is hemorrhage from the lungs or ears or into the skin. Subconjunctival hemorrhage is frequent and sometimes so extensive that the entire white of the eye is replaced by a blood-red color. The most dangerous, although not common, form of hemorrhage is that within the cranium, oftenest meningeal in nature. The effusion of blood may be small and disappear without permanent injury, or large enough to produce death in convulsions, or to leave lasting paralysis of some sort.

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[1] Zeitschr. f. Kinderheilk., Orig., 1920, XXV, 28.
[2] Amer. Jour. Dis. Child., 1925, XXX, 632.
[3] Thèse de Paris, 1901.
[4] Jahrb. f. Kinderheilk., 1900, LI, 426.
[5] Deutsch. Zeitschr. f. Nervenheilk., 1915, LIII, 321.
[6] *Loc. cit.*
[7] Jour. Amer. Med. Assoc., 1925, LXXXIV, 1022.

Otitis media is a not infrequent complication. Albuminuria is often seen during the attack and nephritis is an occasional complication or sequel. Glycosuria may develop. Cutaneous emphysema is a rare occurrence.

Other infectious diseases, may follow, precede or accompany pertussis, among them being varicella, diphtheria, typhoid fever, rubella, scarlet fever, grippe, and especially measles. Tuberculosis in some form, especially as bronchial or mesenteric adenitis or as tuberculous bronchopneumonia, is a common and dreaded sequel. Nobécourt and Forgeron[1] confirm some earlier observations of others that a previously positive cutaneous tuberculin-reaction is liable to disappear during pertussis.

Relapse.—As already pointed out, the cough of pertussis has a very great tendency to return after a brief interval through the action of slight bronchitis. In the strict sense this is rather a neurosis than a true relapse. It sometimes happens, too, that the symptoms of the disease reappear after an interval of weeks or months. Whether this is to be classified as a true relapse is uncertain.

Recurrence.—This is very rare, one attack almost always giving permanent immunity. Errors in diagnosis account for many supposed second attacks. Le Gendre[2] writing in 1891 could find but 9 recorded cases of recurrence, including 1 observed by himself. Widowitz[3] in 558 cases of whooping cough in children found no instance of a second attack. It is probable that this does not apply so strictly to adult life, and that the immunity may occasionally become exhausted at this period. I have seen a few instances in which mothers of children with pertussis developed a modified second attack, and Widowitz records 7 instances of second attacks of the disease in individuals over 30 years of age.

Prognosis.—Contrary to the wide-spread popular opinion pertussis is a serious disease. Approximately 100,000 children died of it in the United States during 10 years (Johnston)[4] and 65,381 died in England and Wales during 8 years (Sticker).[5] Many additional deaths which have been assigned to complications or sequels might properly be added to these figures. Crum[6] estimated that 1 per cent. of the total deaths from all causes in 24 countries depended upon pertussis.

Not only is the actual number of deaths from pertussis large, but the case-mortality is very considerable, varying according to different statistics from 3 to 15 per cent. or over. The general mortality from it in Philadelphia during 5 years equalled 6.9 per cent. (Graham).[7] Age is a powerful factor in effecting this, the danger being the greater the younger the child. In infants pertussis is a dangerous affection, the mortality reaching probably 25 per cent. According to Neurath[1] the mortality in 6469 cases in Vienna was divided as follows:

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[1] Arch. de méd. des enf., 1922, XXV, 394.
[2] Rev. mens. de mal. de l'enf., 1891, IX, 496.
[3] Wien. klin. Wochenschr., 1909, XXII, 1596.
[4] Arch. of Pediat., 1895, XII, 241.
[5] Nothnagel's Encyclop. of Pract. Med. Amer. Ed. Pertussis, 548.
[6] Amer. Jour. Public Health, 1915, V, 994.
[7] Jour. Amer. Med. Assoc., 1916, LXVII, 1272.

Table 81.—Age and Mortality in Pertussis

Age	No. of Cases	Mortality
1st year	1242	25.3 per cent.
2 to 5 years	3139	6.8 per cent.
6 to 10 years	1926	3.9 per cent.
11 to 15 years	135	7.4 per cent.
After 15 years	27	0 per cent.

Saget and van Gelderen[2] give the mortality for children under 1 year in Amsterdam as 42.3 per cent.; from 1 to 5 years 55.9 per cent. and after the 5th year 1.8 per cent. Luttinger[3] places 97 per cent. of the deaths as under 5 years, and Veeder[4] 80 per cent. under 2 years. After the age of 6 years death is uncommon. More girls die than boys; possibly because more are attacked by the disease. (See p. 543.)

The danger depends much more on the *complications and sequels* than on the disease itself. Weakly and marantic or rachitic infants are especially liable to succumb. The combination of scarlet fever or diphtheria with pertussis, or the immediate precedence of measles, increases the danger decidedly. The same holds true for influenza, Reiche[5] reporting 6 deaths in 9 instances of this combination of diseases in children. Obstinate vomiting is a cause of death through the marantic state which results, and tuberculosis is a not infrequent fatal sequel. Often asphyxia following a severe paroxysm is the immediate cause of a fatal issue in infancy. By far the greatest number of deaths, however, depend upon the development of diarrheal diseases, convulsions and, especially, bronchopneumonia, the last mentioned being particularly serious when complicating whooping-cough. Probably over ½ the fatal cases of pertussis are due to pneumonia. Convulsions or pneumonia is especially liable to develop if rickets and pertussis are combined.

Diagnosis.—The diagnostic symptoms in typical cases consist in the evidence of infection; the gradual development of cough which becomes more and more paroxysmal and is finally followed by a whoop; the congestion of the eyes, vomiting, and cyanosis; the tendency for the cough to be worse during the night, and the absence of fever and of physical signs of bronchitis commensurate with the severity of the symptoms. This group of symptoms usually makes the diagnosis simple. In order to hear the cough himself the physician can sometimes produce a paroxysm by examining the throat of the patient with a tongue-depressor or by tickling the nasal mucous membrane.

Yet so many exceptions occur that the recognition of pertussis often becomes difficult or impossible. Early in the attack the nature of the disease cannot be recognized with certainty. Except for the history of exposure, it is only through the cough becoming more paroxysmal as time passes, without evidence of increasing bronchitis, that the case becomes suspicious. Vomiting may occur after severe coughing from other causes, and occasional whooping, too, is not uncommon in children and especially in infants suffering from severe bronchitis. Yet the combination of fever, shortness of breath and numerous râles in the chest constitute sufficient reason to exclude pertussis. The failure of the whoop to develop renders the diagnosis uncertain, unless the other characteristics are well-marked and there is distinct evidence of infection. In mild cases not only the whoop, but vomiting and the violent paroxysmal nature of the coughing, is absent or but little marked. Still even in such cases the prolonged course and its unyielding character to the ordinary treatment of tracheo-bronchitis renders the condition suspicious especially if an epidemic is prevailing; and the diagnosis is practically certain if typical attacks develop in other members of the family. In severe cases in infancy the presence of cough without whoop but followed by attacks of apnea is strongly suggestive of pertussis. After the occurrence of measles the diagnosis may at first be especially difficult, since a persistence of the bronchitis with fever may mask the ordinary character of the early catarrhal stage of pertussis. A prolonged terminal stage of pertussis may arouse the suspicion of tuberculosis of the lungs occurring as a sequel. Continued observation of the case, and particularly the absence of fever, will settle the diagnosis. Tuberculous or simple inflammation of the tracheal or bronchial lymphatic glands may produce a very paroxysmal cough strongly suggesting pertussis. The long-continuance of the glandular affection, and the absence of distinct stages, of vomiting, and of well-marked whooping, tend to exclude the latter. An examination of the blood may be an important diagnostic aid, inasmuch as the leucocytosis, especially of the lymphocytes, is higher than in other afebrile affections which could be confounded with whooping-cough. The danger of mistaking the condition for that of acute leukemia is not to be forgotten. The agglutinative test may be of diagnostic value early in the spasmodic stage (Povitzky and Worth);[1] but later the complement-fixation is likely to be of more service. Olmstead and Luttinger[2] concluded that the latter is positive in 40 per cent. of the cases at the height of the disease or during its decline. Modigliani and de Villa,[3] Garzia[4] and others recommend a diagnostic test for pertussis consisting in the intradermal injection of 0.1 c.c. of a suspension of the killed Bordet-Gengou bacilli in distilled water. If positive this causes a local inflammatory reaction which appears in 48 hours and lasts 48 hours longer. They believe the test specific and diagnostic. Other investigators, however, among them Rissenfeld[5] and Hull and Nauss,[6] have found it of no value, since positive reactions occur in almost any condition, and negative reactions may be present in pertussis.

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[1] Pfaundler und Schlossmann, Handb. d. Kinderheilk., 1906, I, 2, 871.

[2] Nederl. Tijdschr. v. Geneesk., 1919, II, 1525.

[3] *Loc. cit.*

[4] Arch. of Pediat., 1917, XXXIV, 321.

[5] Münch. med. Wochenschr., 1920, LXVII, 1352.

Treatment. PROPHYLAXIS.—In view of the great infectiousness of pertussis and the danger especially in infancy and early childhood, every care should be taken in the separation of those with the disease from others. This should continue at least for 6 weeks in all, or longer if the whooping persists. Although the infectiousness may generally be considered over when once the whooping has stopped for a short time, or even well before this, it is better that the isolation should continue for 2 or 3 weeks after this event. Since the patient is not confined to bed, and cannot properly be kept in one room, the nonimmune children of the family should, if possible, be sent away from the house, or, still better, from the locality where pertussis is prevailing. This is especially important in the case of those less than 2 years old and in delicate children of any age. Fumigation and disinfection of the house and its contents are not essential, but are nevertheless a wise precaution. (See also Vaccine Treatment, p. 557.)

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[1] Arch. Int. Med., 1916, XVII, 279.

[2] Arch. Int. Med., 1915, XVI, 67.

[3] La Pediat., 1921, XXIX, 337.

[4] La Pediat., 1923, XXXI, 890.

[5] Jour. Amer. Med. Assoc., 1923, LXXX, 158.

[6] Jour. Amer. Med. Assoc., 1923, LXXX, 1840.

Treatment of the Attack.—The number of remedial measures which have been tried is enormous. Some are useless; others good in one case; others in another. Many children require practically no treatment; others tax all the resourcefulness of the physician. A review of some of the methods recommended may be given.

(*A*) **Hygienic Treatment.**—Unless some complication interferes, confinement to bed is not necessary and the children should be kept in the fresh air as much as possible. This does not mean, however, that they should be sent out of doors regardless of the state of the weather. Damp and windy weather and all chilling of the surface of the body are to be avoided on account of the danger of producing bronchitis or pneumonia. This is especially true in infancy and early childhood, and at this period of life during the winter-season the airing is best done in the room with the windows open. Should any bronchitis exist, young patients must be kept in airy rooms frequently and carefully ventilated, and exposure to the air outside must be given cautiously if at all during the cold season. It is an excellent plan to have two rooms, one for the day and one for the night, each being constantly open to the air when unoccupied. The sleeping in a freshly aired room undoubtedly diminishes greatly the number of paroxysms. Clothing should be sufficiently warm and food should be nutritious and easily digestible, and administered in small quantities and frequently when vomiting is troublesome. In such cases it is a good plan to employ a liquid diet, given immediately after the attacks of vomiting. Nutrient enemata are occasionally required in older subjects. Change of air, especially to the seashore, is often wonderfully efficacious, particularly in cases at all prolonged.

(*B*) **Local Medication.**—This has been largely employed, chiefly on the ground that the disease was a local infection. It may be divided into (*a*) Insufflation of powders into the nose and larynx; (*b*) Application of solutions by the spray or brush or by irrigation; (*c*) Inhalation of volatile substances or of gases.

(*a*) Quinine has been mixed with boric acid, acacia, or bicarbonate of soda and insufflated in powdered form into the larynx or nose 2 or 3 times a day. Resorcin, salicylic acid, benzoin, tannic acid, and iodoform are prominent among the other numerous drugs which have been used in this way. In my own experience this method of treatment is inferior to internal medication. It is certain, too, that in the hands of physicians not specially trained in making laryngeal insufflation the medicament employed usually does not reach the larynx. When it does, a threatening spasm of the glottis may readily be produced.

(*b*) A 1 per cent. solution of resorcin applied to the larynx or to the pharynx with the spray or the brush has been much recommended. A solution of cocaine, 2 per cent. or stronger, applied by the brush certainly relieves, but is distinctly dangerous. Prominent among the other numerous drugs which have been used in this manner may be mentioned bromide of potash, nitrate of silver, chloride of ammonium, tannic acid, peroxide of hydrogen, and salicylic acid. Irrigation of the nares has often been advocated, among the remedies employed being peroxide of hydrogen, boric acid, sulphate of iron (1 grain: 1 ounce) (0.065:30) bichloride of mercury (1:6000) and salicylic acid (1:1000). The treatment is unpleasant and offers no advantages over spraying.

(*c*) Inhalations of gaseous or volatile substances have certainly often been helpful. One of the most efficacious is carbolic acid or creosote, which may be vaporized from a strong solution in a croup-kettle, inhaled from cotton in a respirator placed over the mouth and nose, or volatilized on metal by direct heat from a small lamp. The possibility of absorption and consequent poisoning must be borne in mind. Turpentine, benzine, thymol, oil of cypress, camphor, naphthalene, and eucalyptus are some of the other drugs recommended for inhalation. Anæsthetization with chloroform or ether has been advised in exceptionally severe attacks. The value of the fumes of burning sulphur has also been urged. The room is fumigated after the child has left it in the morning, aired after several hours, and slept in at night. Formaldehyde preparations have been highly recommended for inhalation, and good results have been reported with ozone. Chlorine gas also has its adherents. It is administered as in the treatment of acute rhinitis (Vol. II, p. 251).

(*C*) **Systemic Medication.**—This method is, in my experience, more reliable and convenient than that just described. What drugs will be of benefit depends partly on the stage of the disease and partly on the individual reaction of the child. In the initial stage such sedative treatment as is useful in acute tracheo-bronchitis will often answer, while in the stage of decline expectorants may be indicated, or drugs exhibited to check excessive secretion. The age of the patient is a factor also, general and cardiac stimulation being especially needed in infancy.

Of the very numerous drugs employed only a few have proven useful in the hands of many physicians. First to be mentioned as one of the best in antipyrine. Shortly after its high recommendation by Sonnenberger[1] I[2] began its administration with excellent results. Children tolerate it in relatively large amount. An initial dose at 3 months may be ¼ grain (0.016) which in severe cases may be rapidly increased to ¾ grain (0.049) or even 1 grain (0.065) every 3 hours. At 2 years 2 to 3 grains (0.13 to 0.194) every 3 hours may be given. It is practically only when fever is present, due to complications, that antipyrine may exert a depressing effect, and in such conditions it should not be used. The favorable results in some cases seem little short of miraculous. In others it is of no avail whatever. Tussol, a combination of antipyrine with hydrocyanic acid, has been highly praised in doses of ½ to 5 grains (0.032 to 0.324) according to the age. Bromoform, recommended by Stepp,[3] is often very useful. To a child of from 3 to 6 years it may be given 3 or 4 times a day in doses of from 2 to 10 minims (0.123 to 0.616) on moistened sugar. The initial dose should be increased cautiously, and if the drug causes drowsiness it should be abandoned.

Belladonna is an old-time favorite often very effective. A child of 2 years may begin with 2 minims (0.123) of the tincture or 1/500 grain (0.00004) of atropine 3 times a day. The amount may usually be rapidly but carefully increased, since full doses are generally required.

Quinine has been proven of value in many cases, but often deranges the digestion. The amount required is generally large, 1 grain (0.065) or more every 2 to 4 hours at 2 years of age. The modern comparatively tasteless derivatives of quinine, such as the ethylcarbamate (aristochin), have been employed successfully to replace it. The bromides are often serviceable in combination with antipyrine and belladonna. Chloral, too, is useful, especially in producing sleep at night. It must, however, be given cautiously to young children on account of its depressing influence. Opium, or its derivatives, is often of great value, particularly if given in a single full dose at night, or sometimes in small doses during the day. Phenacetin, acetanilid and other drugs of this class are sometimes used in place of antipyrine, and are occasionally effective when this fails.

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[1] Deutsch. med. Wochenschr., 1887, XIII, 280.

[2] Therap. Gaz., 1888, Feb., 84.

[3] Deutsch. med. Wochenschr., 1889, XV, 639.

Among the numerous other drugs recommended, for the value of which there is distinctly trustworthy testimony, may be mentioned turpentine, castanea, drosera or droserin, cannabis Indica, asafetida, fluoroform, quebracho, camphor, hyoscine, grindelia, eulatin, adrenaline, veronal, and thyme or its derivative pertussin. Antitussin, an ointment containing difluorphenyl, although used locally by inunction on the thorax, is a systemic remedy in its action. It seems to be undoubtedly of benefit in some instances. Macht[1] and others have reported favorable effects from the use of a 20 per cent. solution of benzyl-benzoate, given in doses of from 5 to 40 drops 3 or 4 times a day. We have not found it produce any noticeable effect. Many reports have appeared in European medical literature concerning the beneficial action of intramuscular injections of ether. Audrain[2] was among the first to use this treatment, although Bedö[3] claims to have antedated him. One to 2 c.c. of ether is given every other day for 3 or more injections. The treatment is very painful, unless great care is taken that none of the ether enters the layers of the skin; so much so that the cure, if any, may be worse than the disease. Tow[4] claimed good results, but had 8 instances of local necrosis.

(*D*) **Mechanical and Miscellaneous Treatment.**—Here may be placed a number of methods of treatment not already discussed. Prominent among these is the employment of vaccination with anti-smallpox vaccine, the usefulness of which Italian physicians have often maintained. As the occurrence of other diseases sometimes modifies the symptoms of pertussis, vaccination may possibly act in this way. I have personally never been convinced of its value. A modification proposed by Violi[5] and found to be very useful by Stern[6] consists in the subcutaneous injection of serum from vaccinated heifers. Fitting the child with an elastic abdominal belt has been highly recommended by Kilmer,[7] a modification of which is suggested by Luttinger,[8] for the control especially of the vomiting attending the paroxysms, and its good effect has been maintained by others. The employment of the constant galvanic current to the neck and spine and the use of the pneumatic cabinet have each had their advocates. Intubation was tried by O'Dwyer[9] in very severe cases with remarkable relief. Nägeli[10] maintained that the pulling of the lower jaw downward and forward would abbreviate or mitigate the paroxysm, and this statement, has been corroborated by Sobel.[11] Treatment with the

[1] Bull. Johns Hopk. Hosp., 1920, XXXI, 236.

[2] Bull. et mém. de soc. méd. d. hôp. de Paris, 1920, XLIV, 795.

[3] Zeitschr. f. Kinderheilk., 1923, XXXV, 325.

[4] Amer. Jour. Dis. Child., 1925, XXIX, 477.

[5] Gaz. hebdom., 1897, XLIV, 904.

[6] Deutsch. med. Wochenschr., 1921, XLVII, 557.

[7] New York Med. Jour., 1903, LXXVII, 1101.

[8] Jour. Amer. Med. Assoc., 1922, LXXVIII, 1636.

[9] 20th Cent. Prac. of Med., XIV, 213.

[10] Correspondbl. f. Schweiz. Aerzte, 1889, XIX, 417.

[11] Arch. of Pediat., 1903, XX, 418.

x-ray applied to the chest has been maintained as very effective by Bowditch and Leonard,[1] Kingston and Faber[2] and Strothers.[3] Remarkable and prompt improvement is said to occur in some instances. From 1 to 3 treatments are required.

A few general remarks may be made on the forms of treatment described. Beginning any of them too early in the attack is to be avoided, since at the most they are symptomatic, and intended only to relieve the severe attacks of coughing. We cannot expect, as a rule, to curtail by them the duration of the disease. Mild cases, in which the paroxysms are few and the general health excellent, may require no therapy at all other than hygienic, or at most, a sedative, such as opium or antipyrine, given at night to lessen the cough and insure sleep. The child should be spared medication when there is no real demand for it. Certainly remedies should not be given which disturb the digestion, since the general nutrition is so liable to suffer in many instances. Further, to determine the value of any treatment for the relief of the paroxysms it is necessary that it be given *before or at the height of the disease.* Remedies employed toward the end of the paroxysmal stage may appear to do good, only because the severity is naturally lessening by this time. Again, before assuming that a remedy is useless it should be given in sufficiently large dose, of course with careful watching. If it proves of no avail in a few days, we should not abandon our efforts but try something else, since no one method can be equally good in all cases. Complications require measures appropriate to them.

(*E*) **Vaccine and Serum Therapy.**—Various efforts have been made to control or to prevent the disease by specific sera and vaccines. Sylvestri[4] employed the blood-serum from convalescent cases of pertussis, and Leuriaux[5] and Klimenko[6] that obtained from inoculated horses. Funck-Hellet[7] believes convalescent serum to be effective as a preventive measure. The greatest interest centers around the employment of vaccines of the Bordet-Gengou bacillus. While the reports of many investigators are most encouraging, the experience of others is opposed. Luttinger,[8] Shaw,[9] Bloom,[10] Caronia[11] and others claimed excellent results; while Von Sholly, Blum and Smith,[12] Davison,[13] Spolverini,[14] Paterson and Smellie[15] and others attributed uncertain or no value to the treatment. Huenekens[16] and Freeman[17] insist that stock vaccines some months old are valueless, but that freshly prepared vaccines may be of service.

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[1] Bost. Med. and Surg. Jour., 1923, CLXXXVIII, 312; Amer. Jour. of Roentgenol., 1924, XI, 264; Jour. Amer. Med. Assoc., 1924, LXXXII, 1422.

[2] Calif. State Jour. of Med., 1923, XXI, 429.

[3] Canad. Med. Assoc. Jour., 1924, XIV, 141.

[4] Gaz. degl. osp., 1901, No. 114. Ref. Münch. med. Wochenschr., 1901, XLVIII, 2020.

[5] La sem. méd., 1902, XXII, 233.

[6] Arch. des sciences biol. de St. Petersb., 1912, XVII, 103.

[7] *Loc. cit.*

[8] Jour. Amer. Med. Assoc., 1917, XLVIII, 1461; New York Med. Jour., 1919, CIX, 322.

[9] Pediatrics, 1917, XXIX, 205.

[10] Arch. of Pediat., 1919, XXXVI, 1.

[11] La Pediat., 1917, XXV, 358.

[12] Jour. Amer. Med. Assoc., 1917, LXVIII, 1451.

[13] Jour. Amer. Med. Assoc., 1921, LXXVI, 242; 466

[14] Il Policlinico, 1920, XXVII, 1043.

[15] Brit. Med. Jour., 1922, I, 713.

[16] Amer. Jour. Dis. Child., 1918, XVI, 30.

[17] Arch. of Pediat., 1920, XXXVII, 410; Med. Rec., 1920, XCVIII, 762.

Hess[1] and Barenburg[2] believe that the treatment has no curative value but is of service as a prophylactic. As already pointed out, the Bordet-Gengou bacillus is not found in all cases of pertussis, and, moreover, exists in several different strains. Further, the severity of pertussis varies greatly, and the disease is liable in the individual case to exhibit improvement or increase in symptoms regardless of treatment; and it is consequently extremely difficult to draw logical conclusions, especially regarding a treatment which extends over a considerable period, or which may have been commenced at a time when the symptoms of themselves were about to undergo improvement. On the whole it must be concluded that the evidences for any curative value of vaccines is still most conflicting, without unquestionable proof of their value or the lack of it; but that there is decidedly more in favor of their employment as a prophylactic measure, if given reasonably soon after exposure.

The treatment appears to be harmless, except for a febrile reaction sometimes seen, and may well be tried especially in infancy and in the first portion of early childhood, since it is at this time that the disease is most to be feared. The dosage is still under discussion, varying from 25,000,000 to 8,000,000,000 or more of the Bordet-Gengou bacilli for the initial dose, with the amount increased in later ones. For prevention 3 injections may be given at intervals of 2 or 3 days. For treatment of the attack 5 or 6 injections may be administered at similar intervals. A mixed vaccine is sometimes recommended containing, in addition to the Bordet-Gengou bacillus, other organisms such as the influenza-bacillus, pneumococcus, micrococcus catarrhalis, and the like. Similar in nature to the employment of the bacillary vaccines is that of the subcutaneous injection of the sterilized sputum from patients with whooping-cough, as recommended by Kraus[3]. Ritter[4] obtained no benefit from it in a trial in 100 cases.

CHAPTER XV

MUMPS

(Epidemic Parotitis)

The title Epidemic Parotitis is only a partially satisfactory one, for, although the parotid glands are the usual seat of the disease, the other salivary glands may be secondarily or occasionally primarily or even solely involved, or the disorder may be represented merely by an orchitis. The affection was well described by Hippocrates. Although confounded later with other disorders it was again clearly differentiated toward the end of the 18th century.

Etiology. Predisposing Causes.—*Age* has a powerful predisposing influence, attacks being most frequent at from 5 to 15 years, and not uncommon in young adults. It is infrequent under 2 years. Ringberg's[1] statistics based on 58,331 cases in Denmark gave:

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[1] Jour. Amer. Med. Assoc., 1914, LXIII, 1007.

[2] Amer. Jour. Dis. Child., 1918, XVI, 23.

[3] Wien. klin. Wochenschr., 1915, XVIII, 1405; Deutsch. med. Wochenschr., 1916, XLII, 281.

[4] Jahrb. f. Kinderheilk., 1916, LXXXIV, 205.

TABLE 82.—INCIDENCE OF MUMPS

Under 1 year of age	205 cases,	0.35 per cent.
1 to 5 years of age	4,512 cases,	7.74 per cent.
5 to 15 years of age	12,103 cases,	20.85 per cent.

Even cases in the new-born have been reported (Gautier,[2] 13 days; Demme,[3] 2 weeks; White,[4] 7 days), and it is even possible for the infection to be acquired during fetal life (Homans).[5] The youngest case coming under my observation was an infant of 10½ months.

Sex has been claimed to be a factor, males being oftener attacked than females, but its action is very questionable. Race and climate exert no influence, and the disease is widely spread over the earth. It is most prevalent in the colder season. *Epidemic influence* is very decided. Localities may be unvisited for several years and then exhibit a number of cases. The epidemic is seldom, however, wide-spread and is usually of short duration. It may be limited to a school or other public institution, or to a small portion of a city, and extend thence only slowly, lasting a few weeks or months. The varying influence of the epidemic is very marked, too, in the severity, the infectiousness, and the tendency to complications. The *individual susceptibility* is not great except where persons are closely associated, as in boarding-schools or in barracks, and the decided majority of those exposed escape. As a result the affection is much less frequent than most of the other acute infectious diseases.

Exciting Cause.—The disease is clearly an infectious one, being a blood-infection with localization usually in the salivary glands. That it is a general infection and not merely a local inflammation is proven by the cases in which the testicle is first or solely involved. The nature of the germ is still not positively known. Various studies have been made by Capitan and Charrin;[6] Ollivier;[7] Bordas[8] and others, and different organisms described. Among the most important contributions is that of Laveran and Catrin[9] who found a diplococcus chiefly in the blood or the parotid secretion in 67 out of 92 cases. These observations were confirmed by Mecray and Walsh[10] and by Bein and Michaelis.[11] Pick[12] discovered the same organism in fluid obtained from the parotid by puncture but failed to find it in the blood. Haden[13] observed a gram-positive diplococcus in the spinal fluid, blood and lymph-glands, which, when injected into a rabbit's testicle, produced orchitis after an incubation-period of 10 days. Teissier and Esmein[14] found a microörganism in the blood and saliva, with which they obtained a positive agglutinative reaction. Kermorgant[1] believes that a spirochete may be the causative organism. Clearly, the germ is not yet certainly known. The experiments which have apparently succeeded in transmitting the disease to monkeys (Gordon)[2] and to cats (Wollstein)[3] would indicate that the virus is a filterable one.

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[1] Ugeskr. f. Läger 5 R. III. 5, 1896. Ref., Jahrb. f. Kinderheilk., 1898, XLVII, 313.

[2] Revue méd. Suisse Rom., 1883, III, 81

[3] Wien med. Blätt, 1888, XI, 1613.

[4] Brit. Med. Jour., 1902, I, 1537.

[5] Am. Jour. Med. Sciences, 1855, XXIX, 56.

[6] Compt. rend. soc. biol., 1881, III, 192; 358.

[7] Rev. mens. des mal. d l'enf., 1885, III, 297.

[8] Compt. rend. soc. biol., 1889, XLI, 644.

[9] Compt. rend. soc. biol., 1893, XLV, 95, 528.

[10] Med. Rec., 1896, L, 440.

[11] Verhandl. 15 Cong. f. inn. Med., 1897, 441.

[12] Wien. klin. Rundsch., 1902, XVI, 309.

[13] Amer. Jour. Med. Sci., 1919, CLVIII, 698.

[14] Compt. rend. soc. biol., 1906, LX, 803; 853; 897.

Transmission and Period of Infectiousness.—Transmission is almost invariably direct, close proximity being required; the germ probably being contained in the breath or the saliva. It may be possible that it can occasionally be carried by the clothing of a third person, or by letters and the like, but this seems certainly uncommon. It is not transmitted by the air to any distance. The tenacity of life of the germ is probably not great. The period of greatest infectiousness is during the presence of symptoms, but numbers of instances prove that mumps can be transmitted some weeks after complete recovery; as also even before swelling appears. The mode of entrance of the germs into the system is usually from the mouth through the duct of Steno into the parotid gland. That there may be other routes is indicated by the occurrence of primary involvement of the testicle or of other salivary glands than the parotid.

Pathological Anatomy.—The benign character of the disease makes the nature of the pathological process a matter little understood, Virchow's[4] opinion, based upon investigations upon secondary parotitis, was that the primary lesion is a catarrhal inflammation of the ducts with consequent obstruction, followed by inflammation of the glandular tissue and a secondary periparotitis. This latter, together with involvement of the cervical lymph-glands, accounts for the diffuse character of the swelling of the neck as the disease advances. Suppuration does not occur except through a secondary infection by pyogenic germs. This theory was supported by certain later investigators. Other views make the primary lesion an inflammation of the interacinous and periglandular connective tissue, the epithelium remaining normal.

The swelling is limited to the parotid glands in the majority of cases. Sometimes the submaxillary salivary glands are also or only affected, and the sublingual gland is less often involved. The testicles are rarely attacked in childhood.

Symptoms. Incubation.—The stage of incubation is a variable one, a general average being 2 to 3 weeks. Periods as short as 3 (Demme)[5] and as long as 30 days (Antony)[6] have been reported.

Invasion.—Prodromal symptoms are absent or overlooked in the majority of cases. When present they consist of irritability, malaise, chilliness, headache, general neuralgic pain, disturbed sleep or somnolence, loss of appetite, and moderate fever. These continue from a few hours to 2 days. Sometimes earache, moderate sore throat, vomiting and diarrhea are seen. Convulsions are exceptional. Barthez and Sanné[7] observed prodromes in 1 out of every 3 cases in their hospital practice.

Stage of Swelling.—On the 2d or 3d day of the invasion, if prodromal symptoms have been noted, evidences of local involvement appear. There is a dull, aching *pain* in the region of the ear and cheek, usually on but one side. It is made worse by pressure and often by movement of the jaws, or by the presence in the mouth of acid or cold substances. It increases gradually during several days, keeping pace with the swelling, and movement of the head may become painful. Pain in the ear is not uncommon. The degree of pain in general varies greatly with the individual. In mild cases the mouth can be opened readily and widely, chewing offers little difficulty, and there is but little pain in the face. In severe cases the teeth can scarcely be separated, chewing is impossible, speaking and swallowing difficult, and there is much aching in the parotid gland, increased by the congestion which eating occasions.

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[1] Ann. instit. Pasteur, 1925, XXXIX, 565.

[2] Rep. Local Gov. Board, London, 1914. Ref., Jour. Amer. Med. Assoc., 1914, LXIII, 414.

[3] Jour. Amer. Med. Assoc., 1918, LXXI, 639.

[4] Annalen des Charité Krankenhaus, 1858, VIII, 3, 1.

[5] *Loc. cit.*

[6] La sem. méd., 1893, XIII, 99.

[7] Mal. des enf., 1891, III, 696.

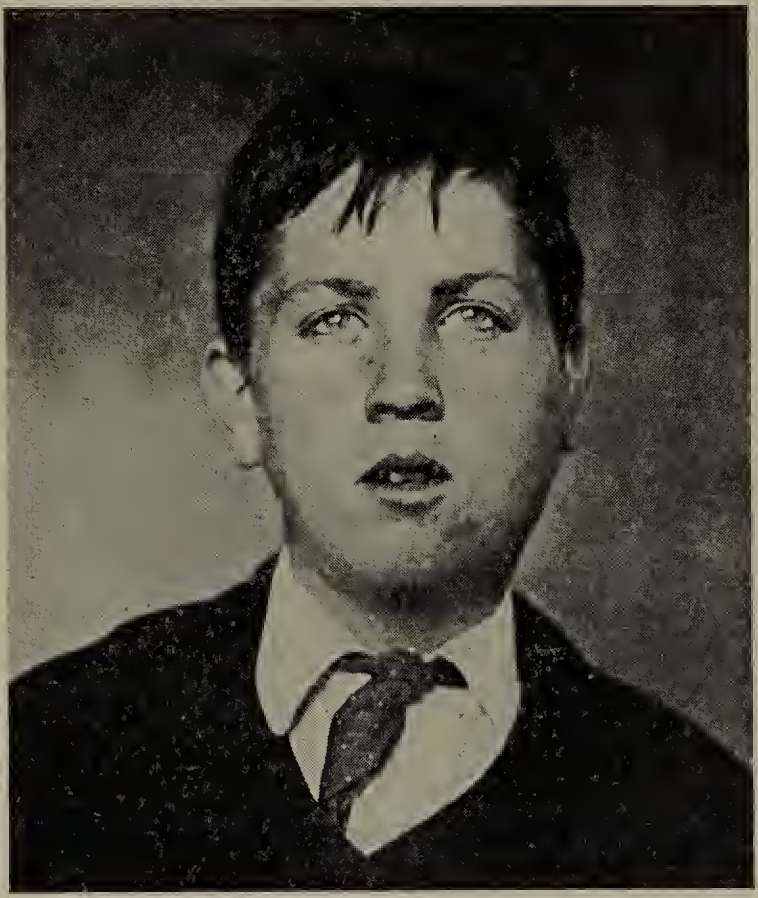

Fig. 153.—Mumps.

Swelling of left parotid gland. From a patient in the Children's Hospital of Philadelphia.

A few hours after the first development of pain, *swelling* begins and increases rapidly. It appears first in the region of the parotid gland, the outline of which can be more or less distinctly felt below, in front of, and behind the ear (Fig. 153). Sometimes only a portion of the gland is attacked at first. The swelling spreads in every direction to an extent dependent upon the degree of involvement of the other salivary glands, the cervical lymphatic glands, and the subcutaneous tissue. In well-marked cases it reaches from the angle of the jaw well up toward the eye, and from the mastoid process to the anterior portion of the neck. The lobe of the ear seems lifted up and pushed outward (Fig. 154). In severe cases the process may extend about the eye, causing edema of the lid, chemosis, or even exophthalmos; or it may reach to the clavicle, or across the neck connecting the two sides. In some instances the whole side of the face is so swollen that the outline of the jaw is obliterated, and the patient may be almost unrecognizable. The swelling is hard and moderately tender on pressure. This is especially so over the central portion, where it is chiefly parotid; while in the periphery the infiltrated

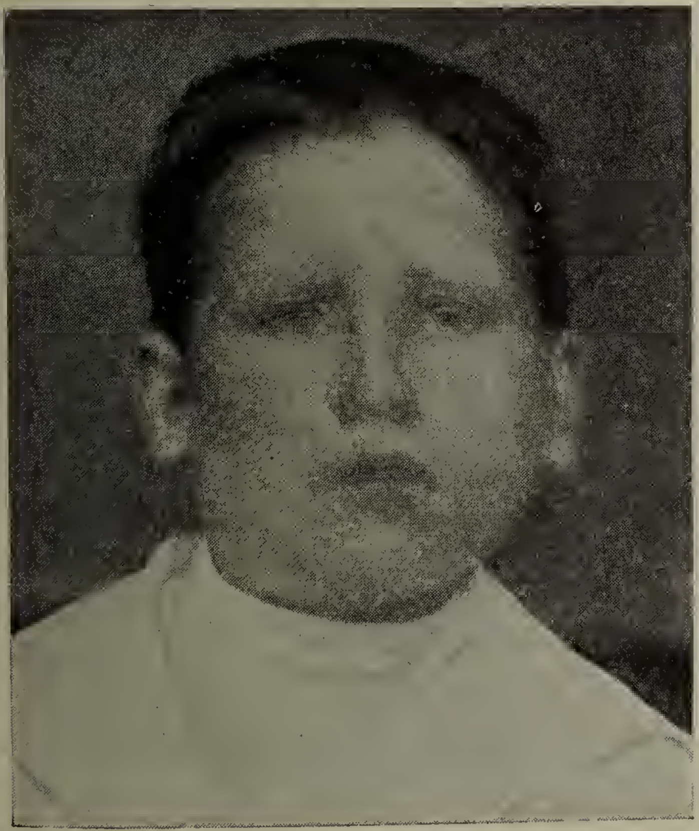

Fig. 154.—Mumps.

Patient in 5th day of the disease, showing eversion of the lobes of the ears and swelling of the parotid region on both sides.

connective tissue offers a lesser degree of resistance. The skin covering it is tense and shining, but not reddened. After the maximum is reached, in from 2 to 4 days or sometimes later, diminution in the size of the swelling begins and progresses with variable rapidity.

In the great majority of cases the second parotid is attacked 1 to 2 days after the onset of the disease in the first. Sometimes the interval is decidedly longer, and the involvement of the first parotid may have entirely disappeared before that of the second begins. The degree of swelling in the two glands is frequently different. In some epidemics involvement of both parotids is much less common than in others.

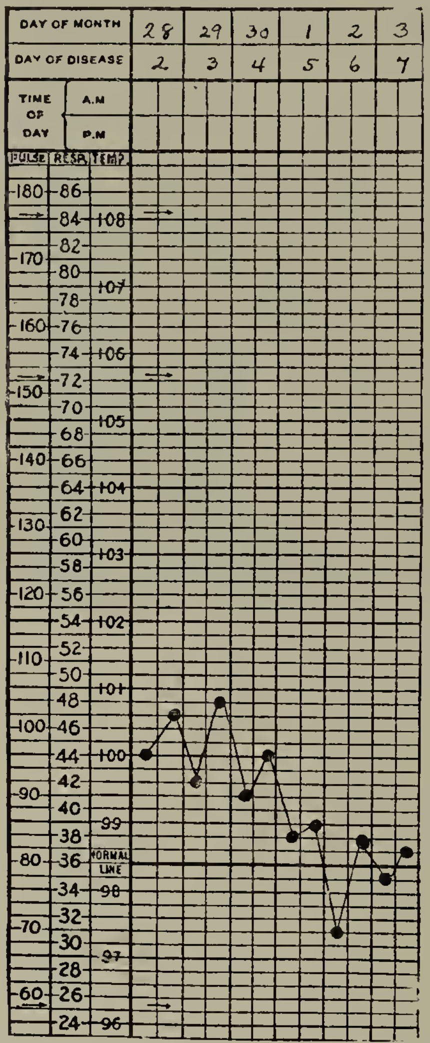

Fig. 155.—Mumps of Moderate Severity.

William K. F., aged 9 years. Apr. 27, swelling began in right parotid without pain or tenderness. On left side slight tenderness; Apr. 29, both sides decidedly swollen, ear-lobes everted, anything cold in mouth gives pain; May 2, swelling of right side nearly gone, that of left diminishing rapidly; May 4, right side normal, some puffiness on left side and under chin.

Quite frequently the disease attacks the submaxillary salivary glands as well as the parotids. The incidence of this occurrence varies with the epidemic. Fabre[1] found it 29 times in 58 cases, the swelling being nearly always secondary to the parotid involvement; but this frequency is rather uncommon. Nevertheless, in an exceptional epidemic reported by Spengler[2] the inflammation was predominantly submaxillary. Quite unusually the submaxillary glands are attacked without involvement of the parotid. This was true, for instance, in 7 of Fabre's cases, while in 9 others the infection was primary here, and secondary in the parotids. In 6 of Wertheimber's[3] 77 patients the submaxillary was alone affected. In cases of submaxillary involvement, an oval, rather soft swelling is found below the jaw on one or both sides. Involvement of the sublingual gland is uncommon. It may exceptionally occur alone or be followed by parotitis (Fabre; Modigliani[4]). It produces a swelling under the anterior portion of the floor of the mouth.

During the first 3 or 4 days of mumps *fever* is present, the degree depending upon the severity of the attack (Fig. 155). A rise to 102°F. (38.9°C.) is the average, although occasionally 104°F. (40°C.) is attained, while in mild cases it may be absent. The secretion of saliva is often much diminished, leaving the mouth dry; often unaffected or occasionally increased. Malaise and some degree of prostration are evident, the appetite poor and sleep disturbed. Swelling of the tonsils and redness of the mucous membrane of the fauces and of the mouth is not infrequent. There may be deafness and tinnitus, vomiting, diarrhea or epistaxis. In severe cases there may be headache, delirium, apathy, somnolence, and even exceptionally convulsions or the symptoms of the typhoid state with enlargement of the spleen. Very frequently, however, the children do not feel ill enough to desire to stay in bed. The urine may exhibit a febrile albuminuria if the temperature is high. Bradycardia is a common symptom, the pulse not infrequently equalling 40 or 50 in the minute (Teissier;[1] Roux[2]).

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[1] Gaz. méd. de Paris., 1887, 7s, IV, 510.
[2] Med. Zeitung., 1852, XXI, 183.
[3] Münch. med. Wochenschr., 1893, XL, 656.
[4] La Pediat., 1916, XXIV, 733.

The *blood* in mumps, according to the investigations of Sacquépée,[3] Krestnikoff,[4] F Pick,[5] Wile,[6] Feiling[7] and others, shows slight or no absolute leucocytosis, or even a leucopenia; but always a relative increase in the unmber of lymphocytes and decrease of the polymorphonuclear cells.

The **duration** of the disease in average cases is 5 to 8 days, but in severer attacks it is sometimes 2 to 3 weeks before the swelling is entirely gone. With the beginning of the diminution of the swelling there is a rapid decline of the constitutional symptoms. The involvement of the second parotid some days after the appearance of the disease in the first naturally prolongs the course.

Complications and Sequels.—The most important of these involve the *genitourinary apparatus.* Orchitis could with equal propriety be considered one of the forms of the disease. This is only rarely observed in subjects under 12 years of age, although in adults it is common. Comby[8] estimated that it is seen in 1 out of every 3 cases of mumps in soldiers, and Wesselhoeft[9] found in 8153 cases of mumps of all ages, and collected from many sources, orchitis reported in 18 per cent. Steiner[10] recorded it in a child of 9 months, in this instance being the primary affection. Barthez and Sanné[11] saw it 3 times in children of 12 years in 230 cases of mumps in early life, and Grognot[12] reports an instance in a boy of 2 years. It develops oftenest in the 2d or 3d week of the disease, and generally only one testicle is attacked. Sometimes inflammation of the testicle is primary and occurs alone, or may be secondary to inflammation of the submaxillary glands. The pathological changes are those of a simple orchitis. The attending symptoms are in part local and in part general. The latter may be severe, consisting of high fever, prostration, feeble pulse, and sometimes vomiting, diarrhea, delirium or unconsciousness. Sometimes adynamic symptoms with low temperature are observed. After the 3d or 4th day the constitutional symptoms rapidly disappear, the resolution of the swelling taking a somewhat longer time. Recovery is generally complete, but atrophy of the gland has resulted not infrequently. Very rarely an analogous inflammation may develop in the ovaries, uterus, female external genitals or the breasts. It is possible that ovarian involvement occurs oftener than supposed, since pain and tenderness are present in a considerable number of cases, as has been pointed out by Troitzky[1] and MacNaughton.[2] Prostatitis and urethritis are rare complications. Infectious nephritis is uncommon. J. A. Miller[3] collected 30 cases from medical literature including 1 reported by himself. It is oftener a sequel than a complication; is nearly always of a hemorrhagic character, analogous to the nephritis of scarlatina, and usually terminates in recovery.

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[1] Bull. acad. de méd., 1912, Jan. 16. Ref. Arch. f. Kinderheilk., 1913, Suppl. Bd. 41.

[2] Thèse de Paris, 1913.

[3] Arch. de méd. expér. et d'anat. path., 1902, XIV, 114.

[4] Dissert. St. Petersb., 1902. Ref. Archiv. f. Kinderheilk., 1905, XLI, 139.

[5] Wien. klin. Rundsch., 1902, XVI, 309.

[6] Arch. of Pediat., 1906, XXIII, 669.

[7] Lancet, 1913, II, 71.

[8] Grancher and Comby, Traité des mal. de l'enf., 1904, I, 449.

[9] Bost. Med. and Surg. Jour., 1920, CLXXXIII, 425.

[10] Wien. med. Blätt., 1896, XIX, 387.

[11] Mal. des enf., 1891, III, 701.

[12] Gaz. méd. de Nantes, 1907. Ref. Arch. de méd. des enf., 1908, XI, 279.

Swelling of the *lachrymal glands* or of the *thyroid* or *thymus* is exceptionally seen. Leriche[4] quoted from literature 9 cases of involvement of the lachrymal glands, and Joly[5] reports 7 instances in 37 cases of mumps. So great a frequency is certainly unusual. Occasionally symptoms of *pancreatic* involvement are exhibited. Simonin[6] observed it 10 times in 652 cases. The subject of pancreatitis in mumps has been reviewed by Farman,[7] with an analysis of 119 collected cases of all ages. Stevens[8] reports 4 cases in children in which he believes there was pancreatic involvement. Swelling of the *cervical lymph-glands* is a common complication, and occasionally persists as a sequel. *Stomatitis* is sometimes seen. Severe *nervous disturbances* occasionally develop as complications or sequels. Prominent here is meningitis which has been repeatedly reported. Feliciano[9] collected 14 cases, and the subject has been reviewed by Acker[10] who reported 2 cases and abstracted 29 in young persons collected from medical literature, and by Casparis[11] who reports also 2 cases. The spinal fluid may be turbid and with an excess of cells (Chauffard and Bordin)[12] or sero-fibrinous (Maximowitsch).[13] Meningeal symptoms may occur, too, without an actual meningitis being discoverable, as has been pointed out by Bonaba,[14] and as we have ourselves observed. Polyneuritis as a sequel has been studied by Rompe.[15] Paralysis of various forms may occur as a sequel. That of the face may be the result of pressure by the inflamed tissue upon the facial nerve. It may develop, too, in other parts of the body, depending upon neuritis or, rarely, meningoencephalitis. Such cerebral conditions as aphasia, choreiform states, and severe psychoses have occasionally been reported. *Involvement of the ear* has been repeatedly recorded. Deafness may be temporary or lasting, usually unilateral, depending sometimes upon an otitis media, but much oftener upon labyrinthine disease or neuritis. Gallavardin[16] and Boot[17] each collected 51 cases of the latter. The *eye* may exhibit conjunctivitis not infrequently, and keratitis, optic neuritis, iritis, and ocular paralysis have occasionally occurred. Woodward[18] collected 23 cases of optic neuritis and neuroretinitis. In a number of instances permanent blindness resulted. *Polyarthritis* associated upon mumps has repeatedly been described. The condition has been studied especially by Lannois and Lemoine[1] and by Sarda[2] who saw this 7 times in about 250 cases. It appears analogous to scarlatinal arthritis. *Laryngeal stenosis* is an occasional and very dangerous complication, depending upon edema or upon pressure by the swollen tissues of the neck.

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[1] Roussky Wratsch., 1902, I, No. 16. Ref. Nothnagel, Spec. Pathol. und Therap., 1904, Parotitis, 55.

[2] Brooklyn Med. Jour., 1903, XVII, 115.

[3] Med. News, 1905, LXXXVI, 585.

[4] Thèse de Paris, 1893.

[5] Arch. de méd. et de pharm. milit., 1903, XLI, 481.

[6] Gaz. mal. inf., 1903, V, 307.

[7] Amer. Jour. Med. Sci., 1922, CLXIII, 859.

[8] Arch. of Pediat., 1925, XLII, 333.

[9] Thèse de Paris, 1907.

[10] Amer. Jour. Dis. Child., 1913, VI, 399.

[11] Amer. Jour. Dis. Child., 1919, XVIII, 187.

[12] Lancet, 1904, I, 1297.

[13] St. Petersb. med. Wochenschr., 1880, V, 185.

[14] Arch. Lat.-Amer. de pediat., 1919, XIII, 130. Ref. Monatsschr. f. Kinderheilk., 1921, XXI, 231.

[15] Monatsschr. f. Kinderheilk., Orig. 1918, XV, 147.

[16] Gaz. des hôp., 1898, LXXI, 1329.

[17] Jour. Amer. Med. Assoc., 1908, LI, 1961.

[18] Phys. and Surgeon, XXXIX, No. 5. Ref. Centralbl. f. inn. Med., 1908, XXIX, 67.

In a small percentage of cases the parotid gland suppurates as a result of a secondary pyogenic infection. This is, however, infrequent in children. Gangrene of the gland is a rare sequel (Demme).[3]

Mumps may occur in combination with *other infectious diseases*, such as varicella, rubeola, pertussis, grippe and scarlet fever. Among other very exceptional complications and sequels recorded may be mentioned erythema, purpura hæmorrhagica, endocarditis, pericarditis, polyneuritis, peritonitis and pneumonia.

Recurrence and Relapse.—Second attacks of mumps are uncommon, but occasionally seen. Some writers state that recurrence is not infrequent; but it is safe to say that the large majority of pediatrists have never observed it. Friedjung,[4] however, saw 1 patient with 4 separate attacks, the last perhaps a relapse; and Rommel[5] reports an instance of 5 attacks. Relapse occurs occasionally in the sense of a reawakening of the process in a gland just recovering, or its development in the second parotid some days after the disease is completely over in the first. Barthez and Sanné[6] observed it 20 times in 230 cases after an interval of from 10 days to 3 weeks. The appearance of inflammation in the second parotid before the disease has run its course in the first cannot properly be considered a relapse.

Prognosis.—Mumps is one of the mildest and least dangerous of the acute infectious diseases; yet complications may occur and exceptionally render the attack very severe and even fatal. In Ringberg's[7] 58,331 cases there were but 7 deaths. Demme[8] saw 2 fatal in 117 cases, both of them from gangrene of the parotid gland. Death has also occurred from a complicating meningitis or nephritis. The danger of atrophy of the testicle or of permanent deafness is to be borne in mind. In childhood, however, all severe complications are very uncommon.

Diagnosis.—This rests upon the rapid development, the characteristic situation and form of the swelling, and the course of the disease. A useful diagnostic sign, to which Cowie[9] has called renewed attention, is the redness and increased prominence of the opening of Steno's duct in the mucous membrane of the cheek. *Acute cervical adenitis* is a frequent source of error in diagnosis. In it, however, the centre of the swollen area appears to be *below the jaw*, while in mumps it is just below the lobe of the ear. The course of adenitis is much more prolonged and the swelling more tender, with redness of the skin covering it. Mumps primary in the submaxillary gland cannot at first be distinguished from lymphadenitis. The more rapid course and sudden onset of the salivary inflammation, the lesser degree of induration, and the development of cases of parotid disease in the household aid in making a diagnosis. Sometimes, however, the distinction cannot be made immediately, especially if the case is not seen at the beginning of the attack. The diagnosis of sublingual involvement should be made only with great reserve, and after exclusion of other possible inflammations in the locality. Association with mumps in other salivary glands or attacking other inmates of the house would generally be necessary to make the diagnosis certain. A *secondary parotitis* occurring in the course of other diseases, such as typhoid fever, sepsis, and the like, is slower in development, unilateral, and tends to suppuration. I have more than once seen *diphtheria* supposed to be mumps, the swelling in the lymphatic glands and subcutaneous tissue of the neck being referred by the observer to the parotid gland. Only a careless failure to examine the fauces can account for the error. In *Mikulicz's disease* or in disease due to tumors or to syphilis the enlargement of the glands is chronic and non-inflammatory in nature, and the disorder can scarcely be mistaken for mumps.

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[1] Revue de méd., 1885, V, 192.

[2] Montpellier méd., 1888, X, 509; XI, 15.

[3] *Loc. cit.*

[4] Wien. med. Wochenschr., 1921, XVII, 637.

[5] Pfaundler and Schlossmann's Handb. d. Kinderheilk., 1923, II, 336.

[6] *Loc. cit.*, 705.

[7] *Loc. cit.*

[8] *Loc. cit.*

[9] Amer. Jour. Dis. Child., 1920, XX, 75.

Treatment. Prophylaxis.—The prevention of the spread of the disease is difficult, owing to the possibility of transmission both after the symptoms have disappeared and probably during incubation. Mild and unrecognized cases also readily spread it in schools. Quarantine to be of any service should continue 3 to 4 weeks or longer from the appearance of symptoms. Fumigation of the room is a precaution hardly required. Hess[1] claims that protection against the development of the disease in exposed children may be obtained by the intramuscular injection of the blood of convalescents, or of those who have had mumps some years earlier. The dosage is 2 to 4 c.c. (.068 to .136 fl.oz.) given before the 7th day after exposure (Regan).[2]

Treatment of the Attack.—This is purely symptomatic. The child should be confined to bed while there is fever, and given a light diet, especially one which does not require chewing. Acid substances, too, should be avoided, as they sometimes increase the pain A mild laxative may well be administered at the beginning of the attack, and a febrifuge should be ordered if required. Careful attention should be paid to the mouth and throat to prevent stomatitis or to relieve pharyngitis. Pain may be alleviated by hot fomentations or by rubbing with warm olive oil, or the gland may be covered with raw cotton and the face rested against a hot water bag. A 5 per cent. guaiacol ointment has been recommended to relieve pain. Mallié[3] believes that the intramuscular injection of diphtheria-antitoxin tends to prevent or to relieve orchitis. Severe nervous symptoms with high fever require warm baths and other sedative and antipyretic treatment. It is claimed that the incidence of orchitis can be lowered by injections of convalescent serum if given early in the disease. This procedure is hardly necessary in childhood.

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[1] Amer. Jour. Obstet., 1915, LXXII, 183.

[2] Jour. Amer. Med. Assoc., 1925, LXXXIV, 279.

[3] Jour. de méd. de Bordeaux, 1922, XCIV, 12.

CHAPTER XVI

MALARIA

History.—The disease has existed since early times, and different forms of it were well described by Hippocrates. It was, however, confounded with many other affections. A clearer understanding arose after the discovery of the specific action of cinchona.

Etiology. Predisposing Causes.—*Climate* is of the greatest importance, tropical and sub-tropical countries being especially the home of the disease; where it prevails, in many localities, to an extent and with a severity unknown in cooler regions. It is, however, very widely distributed, although in temperate climates it has been steadily growing less frequent. Formerly common in the Middle and North Atlantic States, it is now generally seen but seldom, or in few regions; and in those parts of the tropics where precautions against it are taken its occurrence has been greatly lessened. *Season* is also a factor, more cases being observed in summer and autumn. All *ages* are attacked by it, but children seem especially predisposed. Even the new-born not infrequently exhibit it, and cases of fetal malaria are on record in which the parasite has been found in the blood shortly after birth. The possibility of the occurrence has repeatedly been denied, and without doubt it is very exceptional; but cases which appear to be beyond question have been reported by Crandall,[1] Pies,[2] Lemaire, Dumolard and Laffont,[3] Bass,[4] Simms and Warwick,[5] Cuadra,[6] Forbes,[7] Trimble[8] and others; and Laffont[9] has collected 47 observations up to the date of his writing, some of these belonging, however, to the older literature.

It should be stated that the degree of susceptibility of children is disputed. In Concetti's[10] 360 cases occurring in the first 7 years of life, there were but 9 in the 1st year, and 24 in the 2d year. It is now, however, generally believed that the disease is much more frequent at an early age than these figures would indicate, and Koch's[11] studies in Java appear to prove that infection by the parasite is very common even in infancy, especially in malarial districts. It is probable that in a large number of subjects at this period of life the symptoms are uncharacteristic and unrecognized; that the organism may even be present without producing any clinical manifestations, and that the children later possess a comparative immunity against the development of the disease. Epidemic influence exists in a sense, in that some years show very few cases, and other numerous ones in the same locality. The affection is endemic in some regions; rarely seen in others.

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[1] New York Polyclinic, 1893, I, 38.
[2] Monatsschr. f. Kinderheilk., Orig., 1910, IX, 51.
[3] Bull. et mém. de la soc. méd. des hôpitaux, 1910, XX, 866.
[4] Arch. of Pediat., 1914, XXXI, 251.
[5] Jour. Amer. Med. Assoc., 1908, LI, 916.
[6] Arch. de méd. des enf., 1920, XXIII, 606.
[7] Amer. Jour. Dis. Child., 1923, XXV, 130.
[8] China Med. Jour., 1924, XXXVIII, 121.
[9] Thèse de Paris, 1910.
[10] Traité des mal. de l'enf., Grancher and Comby, 1904, I, 554.
[11] Deutsch. med. Wochenschr., 1900, XXVI, 88.

Exciting Cause.—Many later observations have confirmed the discovery by Laveran[1] in 1880 that the cause of the disease is a parasite of the sporozoa class called the plasmodium malariæ, hæmatozoon malariæ, hæmamœba, and by other names. Unlike bacteria, these germs pass through a definite life-cycle in which they exhibit forms seemingly diverse. In the process they destroy the red blood-corpuscles, producing pigment which appears in the leucocytes and in some of the tissues of the body. It was later discovered by Golgi[2] that there is more than one variety of the parasite, and that each produced a different form of the disease.

But a very brief description of the microörganism can be given here:

1. *The Parasite of Tertian Malarial Fever* (Plasmodium vivax).—The life-cycle of this variety in human blood is about 48 hours. The parasite consists at first of a small, hyaline, unpigmented body with lively ameboid movements, contained in the red blood-corpuscle. In 24 hours it has become about the size of the swollen, decolorized corpuscle, and exhibits a large amount of black, granular pigment. Segmentation now begins, producing a radial arrangement of the body, and in 48 hours from the beginning 15 to 20 small round spores are set free at the time of the chill, which enter other red blood-corpuscles and begin again the *asexual* cycle. Some of the mature bodies do not segment but pass through the *sexual* cycle when absorbed by the mosquito.

2. *The Parasite of Quartan Malarial Fever* (Plasmodium malariæ).—The life-cycle of this form is 72 hours. It appears at first as a small, hyaline, unpigmented body with very little and very slow ameboid movement. In 48 hours it grows to about 1/3 or 2/3 the size of the red blood-corpuscle containing it, becomes more pigmented, and loses all movement. In 60 hours it fills the red blood-cell, which is not enlarged or decolorized. Segmentation now begins, producing a star-shaped or "daisy"-shaped body. This is followed by sporulation at 72 hours at the time of the chill, 5 to 10 spores being discharged to enter other red blood-cells. Certain mature bodies do not segment, as in the case of the tertian parasite.

3. *The Parasite of Æstivo-autumnal* (*Tropical*) *Fever* (Plasmodium precox).—The life-cycle of this parasite is probably variable, ranging from 24 to 48 hours. It consists at first of a very small body, hyaline or with little pigment, actively ameboid, or, when at rest, having a ring-form. It develops pigment which is distributed about the periphery of the organism. It is much smaller than the preceding forms, and the red blood-cell containing it is much shrunken. The later stages of development take place in the internal organs, especially the spleen and the bone-marrow, where the concentration of pigment, segmentation, and sporulation occur. After the attack has lasted about a week crescentic pigmented bodies appear free in the blood-serum. These are the forms of the parasite which later pass through the sexual cycle in the body of the mosquito. They are characteristic of estivo-autumnal malaria. In the course of development some of them form flagellæ.

Transmission.—Malaria, although an infectious disease, is not contagious in the ordinary sense; *i.e.*, it is impossible for one individual to contract it directly from another, except by experimental inoculation with the blood of the patient. That it may be given in this way was first demonstrated by Gerhardt[3] and since then repeatedly by others.

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[1] Bull. de l'acad. de méd., 1880, XLV, 1346.

[2] Gaz. degli osp., 1886, VII, 419.

[3] Zeitschr. f. klin. Med., 1884, VII, 372.

Manson[1] was the first to indicate clearly that the disease was communicated to man by the mosquito, which acted as host; Ross[2] showed that the parasite developed in the body of mosquitoes which had bitten malarial patients, and Grassi and Bignami[3] succeeded in transmitting the disease directly from the insect to man. Various species of the genus Anopheles are the only ones harmful. The insects acquire the parasite, as far as known, only by sucking the blood of a patient with malaria. In their bodies the organism then passes through another, viz. a sexual, cycle of development different from that seen in man. The *duration of life* of the plasmodium is uncertain, since whereas the asexual, segmented form is present in human blood only during the attack of malaria, the sexual, non-segmented form may persist there an indefinite time without producing symptoms. This accounts for the occurrence of relapses after intervals of months. It explains, too the multiplication of cases at the season of the year when anopheles begin to be abundant, since the insects then acquire the parasites from such individuals.

Pathological Anatomy.—Many of the changes found depend upon the extensive destruction of the blood which the parasites produce. The spleen is always more or less enlarged; very soft, especially in children; and exhibits pigment and broken-down corpuscles. The enlargement in chronic cases is great. The liver may be hypertrophied and contain pigment, which gives it a slate-brown color; the kidneys enlarged and gray-red, with pigmentation of the glomeruli. The lesions of acute nephritis are sometimes seen, or chronic nephritis after repeated attacks of the disease. The gastrointestinal mucous membrane and even the skin may exhibit pigment.

Symptoms. Period of Incubation.—The duration of this period is uncertain. Even in the disease produced experimentally it varies from 3 to 21 days (Mannaberg).[4]

TYPICAL FORM.—The ordinary form of the disease occurs in adults and in later childhood much oftener than at earlier periods. Prodromal symptoms are often present, lasting 1 or more days and consisting of malaise, loss of appetite, vertigo, chilliness, yawning, and irritability. A distinct paroxysm then develops, marked by headache, lassitude or prostration, coated tongue, general pains, and often vomiting. There promptly follows an evident chill of varying intensity lasting 10 minutes to an hour or more, with shaking of the body, chattering of the teeth, sensations of cold, and a pinched and blue appearance of the face. In this, the *cold stage*, although the surface of the body feels cold to the touch, the thermometer shows a decided rise of axillary or rectal temperature. The pulse is small. The second, or *hot stage*, is marked by the beginning of the sensation of fever. The temperature reaches 104° to 106°F. (40° to 41.1°C.), the skin is flushed, the pulse full and accelerated, and headache and thirst are complained of. The spleen can often be felt. The maximum temperature as shown by the thermometer is reached during or shortly after the chill. The duration of this stage is from 3 or 4 up to 12 hours.

The second stage of the paroxysm is gradually replaced by the third, or *sweating stage*, in which all the uncomfortable symptoms of fever disappear and the temperature rapidly returns to normal. The amount of sweating is very variable. Comfortable sleep usually follows. The total duration of the paroxysm averages 6 to 12 hours. After the paroxysm the patient usually feels entirely well until the next one occurs, the length of the interval depending upon the type of malaria present.

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[1] Brit. Med. Jour., 1894, II, 1306.

[2] Brit. Med. Jour., 1897, II, 1786.

[3] Ref. Mannaberg, Nothnagel's Encycl. Pract. Med. Amer. Edit., Malaria, 117.

[4] Nothnagel's Encyclop. Pract. Med., American Edit., Malaria, 103.

The urine in malaria frequently contains albumin in moderate amount. It may often be scanty just before the chill and increase during and after the paroxysm. The blood exhibits, besides the parasites, a very decided and rapidly developing anemia in cases which have had several paroxysms. Leucocytosis is rare in malaria, but a relative increase of the large mononuclear cells is considered characteristic by some investigators. The leucocytes often contain pigment-granules. Netter[1] found the fragility of the red corpuscles decreased during the attack. Genoese[2] states that, in severe cases in infants, the cerebrospinal fluid exhibits during the attack an increase of albumin and sometimes a lymphocytosis, but not to the degree seen in actual meningitis.

Instead of beginning at almost the same hour on the days of the attacks, which is the rule in malaria, the paroxysms sometimes "anticipate" slightly, occurring from 1 to several hours before the full period has elapsed (Fig. 156). Under treatment with quinine "postponement" of the paroxysms for some hours is sometimes observed.

The description of the symptoms as given applies to the intermittent form of the disease and as occurring in older children. In many instances, however, especially in tropical climates or in young children anywhere, malaria assumes an irregular, continuous, or remittent type, with the various stages of the paroxysm less marked, or not separated at all.

Varieties of the Typical Form.—1. *Tertian Malarial fever*, the most frequent form in temperate zones, is that produced by the tertian parasite. In *Simple Tertian* but one set of germs is present, and paroxysms occur every other day, usually at about the same hour (Fig. 156). Should, however, two sets of the tertian organism be present, reaching the stage of sporulation on alternate days, *Double Tertian*, or *Quotidian* fever occurs (Fig. 157), the patient having a paroxysm every day.

2. *Quartan fever*, depending upon the quartan parasite, exhibits a paroxysm every 4th day; *i.e.*, with free intervals of 2 days (Fig. 158). Should two sets of the organism be present, paroxysms occur on two consecutive days, with only one day free. If three sets are present, quotidian (*i.e.*, triple quartan) fever is produced.

3. *Æstivo-autumnal Fever*, or *Tropical Fever*.—Both the other forms described are often denominated "*Intermittent fever*," since the temperature is normal for at least a part of every day. In the form due to the estivo-autumnal parasite the attack is usually of a *remittent* type; *i.e.* the temperature, although lesssening at intervals, does not reach normal, and the paroxysms, if present, are of longer duration (Fig. 159). Jaundice is common and gastrointestinal symptoms may be marked. In other cases the individual paroxysms may be little or not at all observed, and great irregularities in the course are liable to be exhibited, and in still others the temperature is continuous, with little variation, and the case may resemble typhoid fever closely. Sometimes, although dependent upon the æstivo-autumnal parasite, the attack is distinctly intermittent and either quotidian or tertian in character. The cold stage is, however, absent or less marked, the rise and fall of temperature are

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[1] Rev. méd. de Bogotá, 1919, XXXVII, 52. Ref. Jour. Amer. Med. Assoc., 1919, LXXIII, 72.

[2] Il Policlinico, 1919, XXVI, 737.

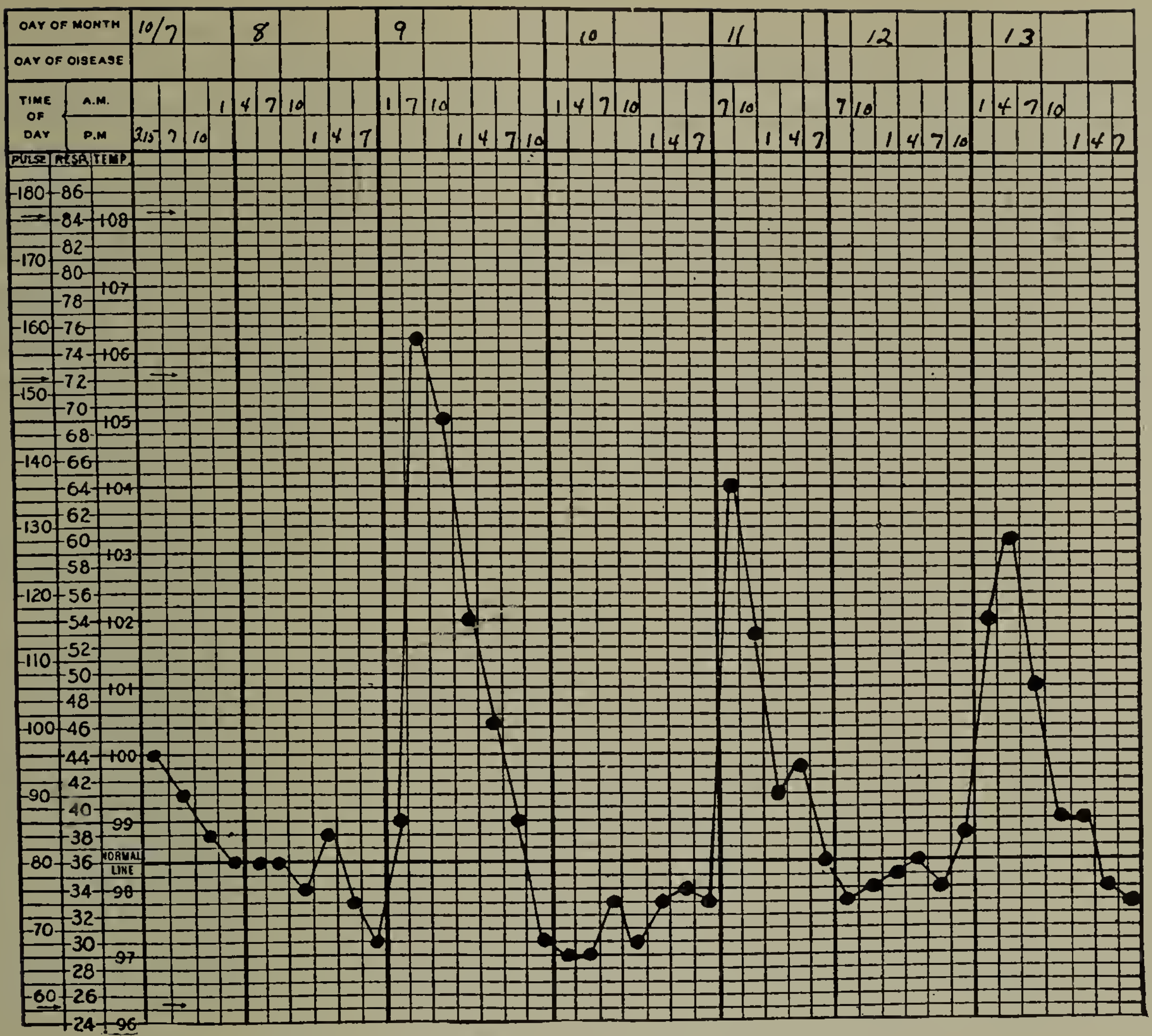

FIG. 156.—MALARIA, SIMPLE TERTIAN.

John T., aged 2 years. Oct. 6, fever, thirst, diarrhea; Oct. 7, spleen large, free urination; Oct. 9, plasmodium found in blood; Oct. 13, paroxysm anticipated several hours.

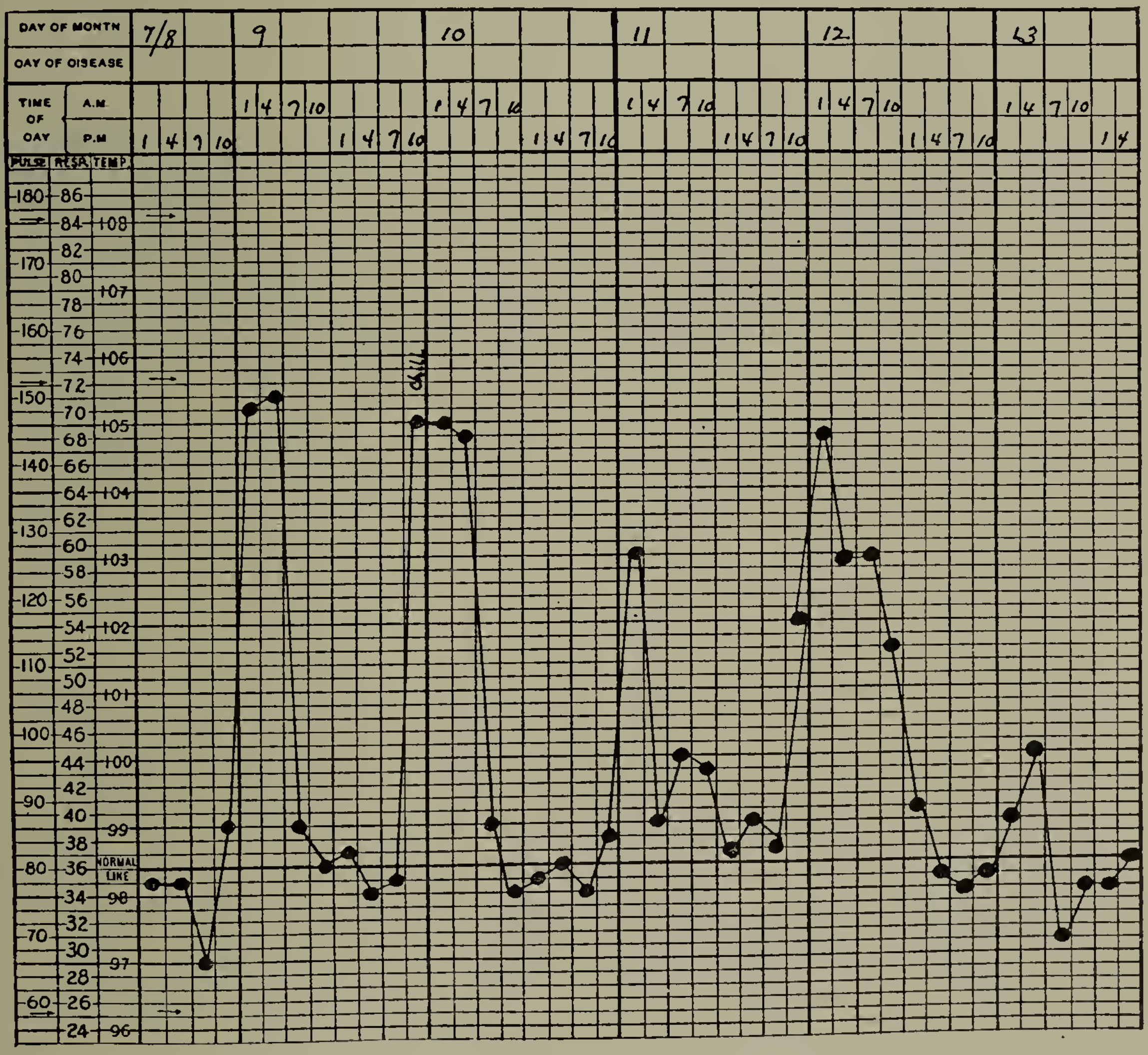

FIG. 157.—MALARIA, DOUBLE TERTIAN.

Lizzie Q., aged 8 years. Been having chill every night followed by fever. Vomited once. Examination showed enlarged spleen and parasites in blood. Attacks controlled by 20 grains (1.296) of quinine by mouth and 10 grains (.648) by suppository given on 12th. Previous small doses without effect.

usually more prolonged, and the patient does not feel so well in the apyretic period as when the disease is due to the tertian parasite. The cases show, also, an obstinate tendency to relapse.

Cases of æstivo-autumnal fever occur in both temperate and tropical regions, but those of the severer form are encountered principally in tropical and sub-tropical countries, including the Southern United States. They may pass into the *pernicious type.* The *algid form* belongs here. It is marked especially by an extreme sensation of coldness, scanty urine, excessive prostration, vomiting, and sometimes profuse diarrhea. The temperature is, at most, only slightly elevated; often sub-normal. Respiration is accelerated, the pulse weak, and the mind clear. Another variety of pernicious malaria is the *comatose form,* with high fever and rapidly developing unconsciousness and delirium. The *hemorrhagic form,* or "blackwater fever," is that variety of pernicious malaria characterized especially by hemoglobinuria, jaundice and uncontrollable vomiting. It is very uncommon in childhood. It is to be noted that occasionally cases of the pernicious type may be produced by the tertian or quartan parasite.

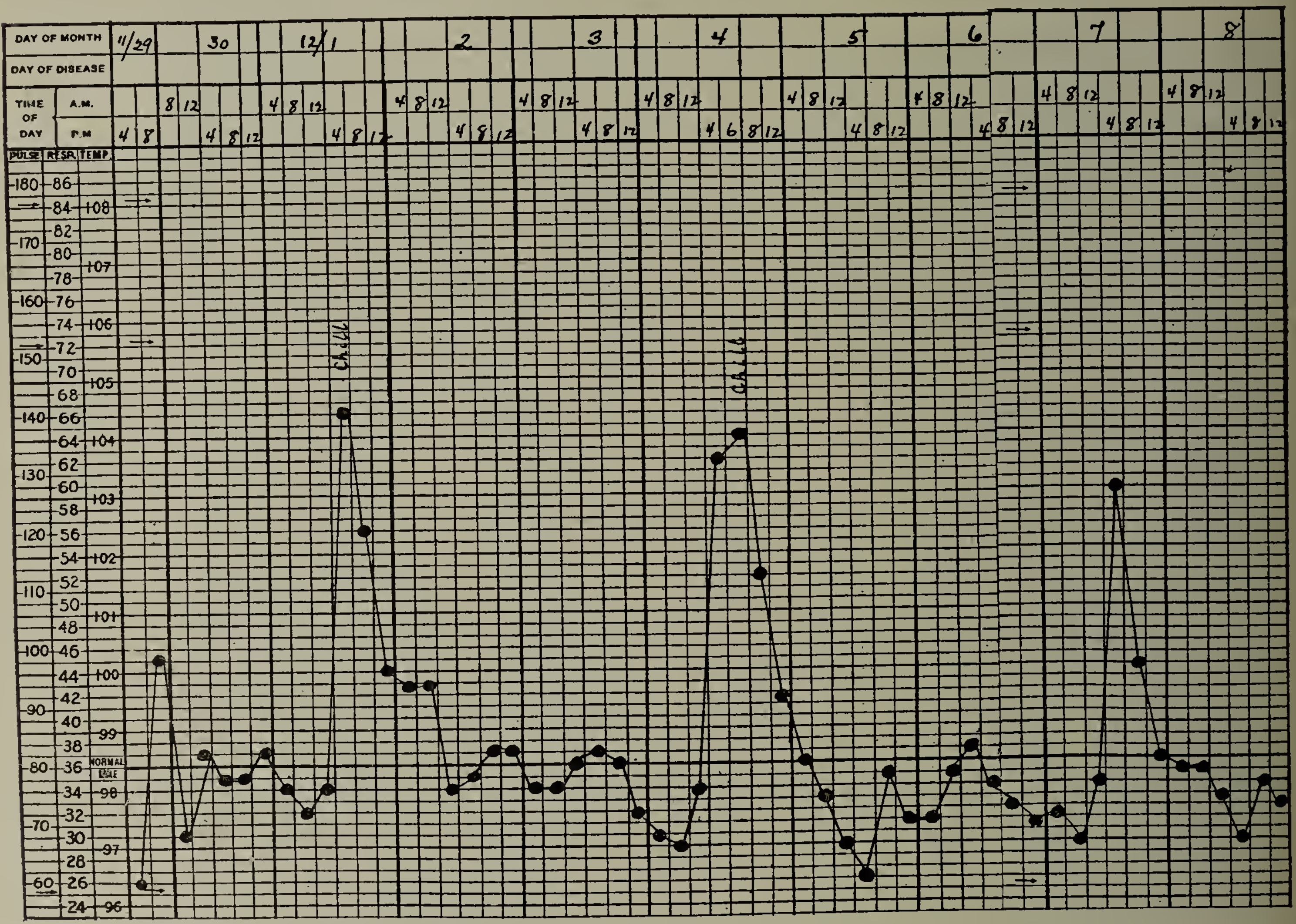

FIG. 158.—MALARIA, QUARTAN.

Lawrence G., aged 16 years. Tertian fever a year before. Chills at intervals for over a year. Blood showed typical quartan bodies. Spleen much enlarged. (*Thayer & Hewetsons' "Malarial Fevers of Baltimore."*)

Tertian malarial fever is by far the most frequent form of the disease in children in temperate zones, the quotidian type being especially common. Quartan fever is rare in the northern portions of the United States, and in the southern States is less frequent than other forms. The æstivo-autumnal fever is the prevailing form in tropical regions. Occasionally there is a combination of the æstivo-autumnal type with the other varieties.

Irregular Forms.—It is particularly in infancy and early childhood, although by no means solely then, that variations from the definite types of the disease are seen, or that certain symptoms are especially prominent. The typical course is more often absent than present at this time of life. The onset is more abrupt, the whole paroxysm is often shorter, and the division of the attack into stages is frequently absent or little marked. In infants especially the symptoms may be so irregular and so devoid of periodicity that the disease is frequently overlooked. The chill in early life is usually replaced by mere coldness, pallor, and blueness of the face and extremities; or by yawning and drowsiness, or not infrequently convulsions. Vomiting is a common initial symptom. In the hot stage vomiting may continue and there is coating of the tongue and loss of appetite. Constipation may be present, but diarrhea is very common and sometimes is profuse. The younger the child the more liable is diarrhea to occur. The infant exhibits either drowsiness, or restlessness and crying. The temperature is usually higher in early life and is prone to run a much more irregular course, approaching sometimes, even in tertian fever, a remittent or continuous type (Figs. 160 and 161), due, probably, to the presence of a number of sets of the tertian parasite; or fever may be absent in young infants (Mulherin and Mulherin).[1] Nervous symptoms are prominent. Headache and pain in the epigastrium, limbs, or splenic or hepatic region are very frequent and delirium

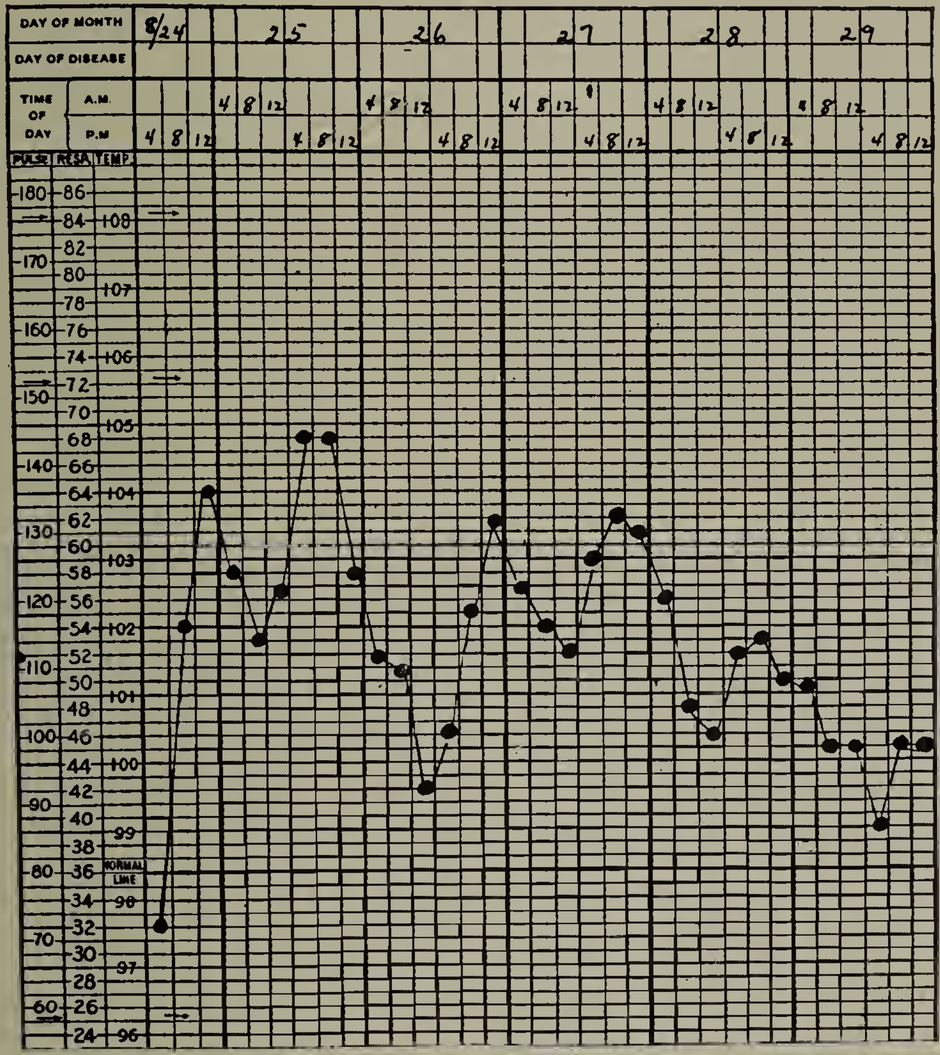

Fig. 159.—Malaria, Aestivo-autumnal. Showing Irregularly Remittent Temperature.

Emma B., aged 13 years. Illness began 10 days before, pain in abdomen and side, cough, enlarged spleen, crescentic bodies in blood. No chills or distinct paroxysms. Quinine treatment commenced on 28th. (*Thayer & Hewetson's "Malarial Fevers of Baltimore."*)

[1] Jour. Amer. Med. Assoc., 1922, LXXVIII, 1873.

is not uncommon. Bronchitis is a very common symptom and not infrequently there may be a degree of pulmonary congestion, especially in infancy, which suggests pneumonia. The sweating stage is absent or little marked in children under 2 years of age, and when seen is liable to be attended by considerable prostration. According to Mulherin and Mulherin[1] enlargement of the spleen is not discoverable in fully 30 per cent. of the cases in young infants.

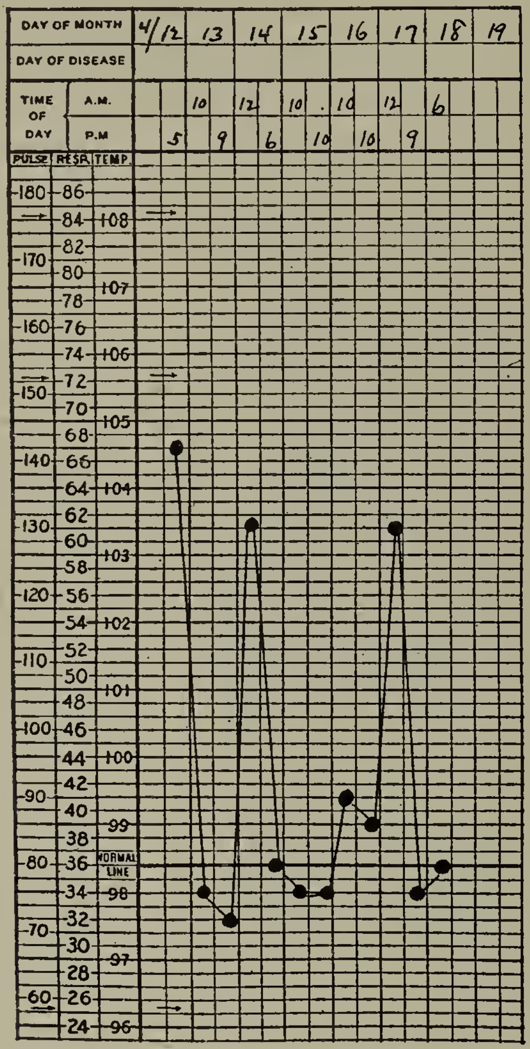

Fig 160.—Malaria, Tertian Type Showing Irregularity. with Absence of Rise of Temperature on One Occasion.

Mamie R., aged 2 years, 8 months. Cough and cold in head 2 weeks previously, lost appetite. A week before seen began to have fever every other day. Headache, pain in abdomen, drowsy. Examination on admission to the Hospital of the University of Pennsylvania showed spleen slightly enlarged to palpation. Nothing else of note. Malarial organisms found. Child removed. Quinine not administered.

Latent Malaria.—In some cases malaria may be "latent," the usual symptoms, including fever, being absent, and the disease suggested only by such obscure manifestations as a periodically recurring cephalalgia or cardialgia, periodic diarrhea, periodic torticollis, or an obstinate cough which yields only to quinine. It is to be noted, too, that the malarial parasite may remain dormant in the system for months, and probably indefinitely, without producing symptoms, the patient having at times recurrent attacks.

Chronic Malaria. Malarial Cachexia. In patients who have had repeated attacks of malaria a cachexia develops, characterized especially by large spleen and great anemia, with consequent debility, pallor, and dropsy of the skin and sometimes of the serous cavities. There is usually little or no fever, or slight rises occur only for a short period in the 24 hours. The general symptoms are indefinite, consisting of anorexia, coating of the tongue, malaise, debility, cough and vague pains. There is, in fact, nothing especially suggestive, unless the patient is a resident of a malarial district, and only the examination of the blood can determine the diagnosis.

Complications and Sequels.—One of the most prominent complications is bronchitis. As already pointed out, it may sometimes be the most suggestive evidence of the disease. Congestion of the lungs often occurs and even exceptionally a fully developed pneumonia. Epistaxis is an occasional complication. Gastrointestinal affections of moderate severity are sufficiently frequent to constitute a symptom of the disease. Vomiting, however, may become very obstinate and diarrhea may at times take on a dysenteric or choleraic character. Jaundice is not uncommon. Herpes is seen very frequently and urticaria and erythema occasionally, and symmetrical gangrene has been reported. Endocarditis and torticollis have been observed and neuritis is an occasional sequel. Aphasia and hemiplegia or other paralyses have been noted. Acute glomerular nephritis occurs not infrequently in severe

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[1] *Loc. cit*

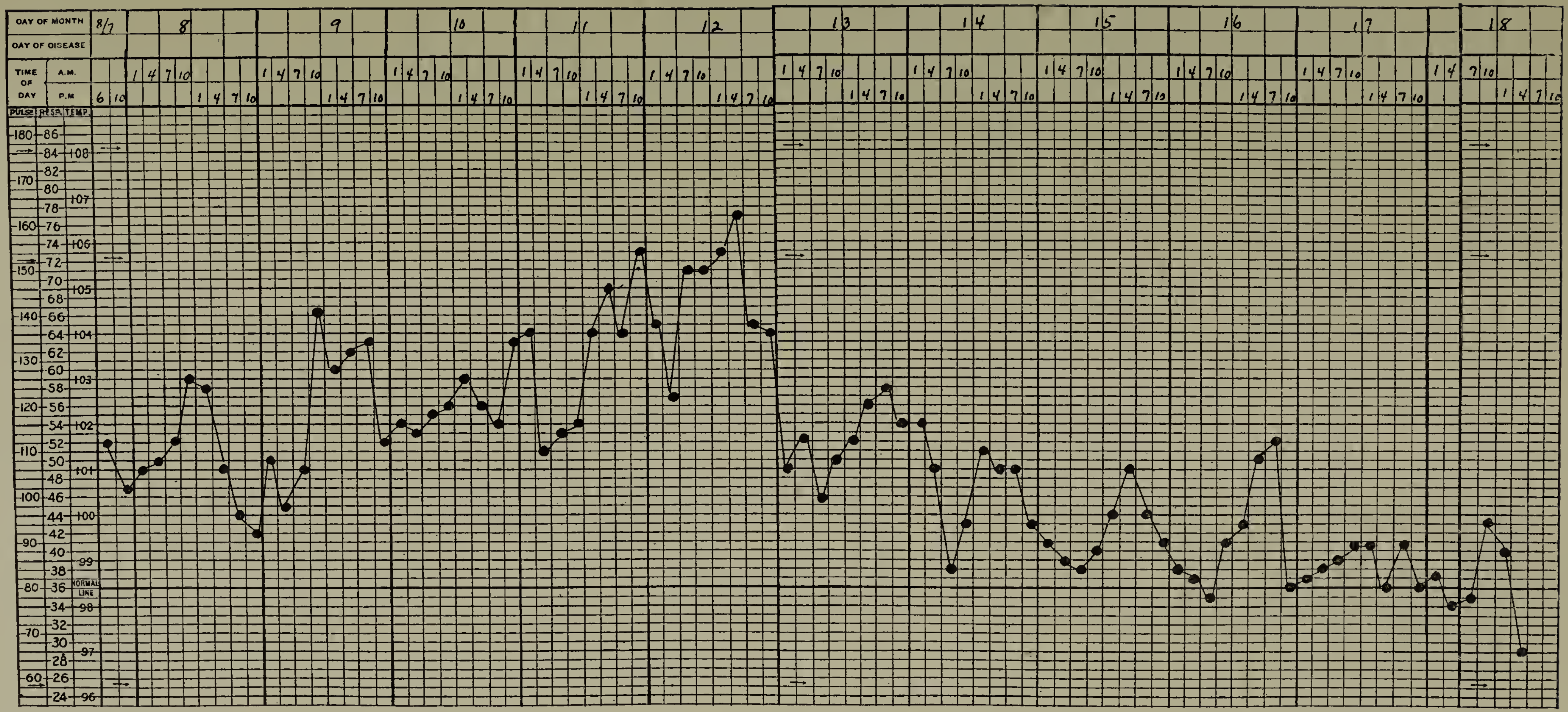

FIG. 161.—MALARIA, IRREGULAR INFANTILE TYPE.

Harriet L., aged 19 months. Been ill for 5 days with loss of appetite, malaise, diarrhea and repeated vomiting. Examination showed enlarged spleen and liver and numerous plasmodia in the blood. Given 10 grains (.648) of quinine daily, by suppository, beginning on Aug. 9, without effect; and 10 grains (.648) quinine, taken by mouth, daily, beginning on the 13th.

cases and chronic nephritis may develop in malarial cachexia. Glycosuria has been frequently observed and vesical irritability with enuresis is sometimes seen. Malaria may occur in combination with or as a sequel to other infectious diseases as, for instance, pertussis, scarlet fever, smallpox, and syphilis. In some regions it is frequently combined with typhoid fever.

Recurrence and Relapse.—The tendency to relapse is one of the great characteristics of malaria. The germs may lie dormant in the system for an indefinite period and the disease reappear at any time. How often the new attack is a true *relapse* due to an infection already present, and how often a *recurrence* through a reinfection from without, it is usually impossible to say, unless the patient has removed to a locality certainly free from anopheles. The occurrence of one attack usually seems to render the subject particularly liable to later ones. On the other hand, the studies of Koch[1] have confirmed the opinion of many physicians in the tropics that infected individuals may finally become immune, and that the natives of some malarial districts do not readily acquire the disease because of having developed this immunity through repeated atypical attacks in infancy.

Course and Prognosis.—The duration of the disease is uncertain. Unless treated with quinine it may continue indefinitely and pass into the chronic stage. In other cases, particularly of the tertian type, it stops of itself after a week or more, although very prone to relapse frequently. The pernicious cases end fatally in a day or two. Malarial cachexia may last months or years before complete recovery is obtained. The prognosis varies with the locality. In malarial regions, especially in the tropics, the disease reaches the proportions of a plague, and is the cause of many deaths. In temperate climates the prognosis is usually good in districts where the malady is sporadic or occurs only exceptionally in epidemic form. This is most true of the intermittent type, which tends to recover spontaneously, particularly if the subject is removed from malarial districts and repeated reinfection prevented. The irregular forms run an indefinite course, and when they are dependent upon the æstivo-autumnal parasite they are liable to be severe. In the Southern States of this country and in tropical regions the æstivo-autumnal form is attended by a higher mortality in infancy, and death may take place in a few hours during the first attack. This applies, however, only to the pernicious variety of the disease. Under treatment and outside of malarial districts the prognosis of malaria is favorable, and death rarely occurs except from complications. The great tendency to relapse has already been referred to.

Diagnosis.—The diagnosis is easy in typical cases, but often presents great difficulties in the anomalous and irregular forms, which are especially liable to develop in early childhood and infancy. A certain tendency to periodicity may sometimes be discovered in these cases, which is very suggestive, as is also enlargement of the spleen; but the most satisfactory diagnostic characteristics are the cure by quinine, and the discovery of the parasite in the blood. This latter can usually be accomplished when quinine has not been administered for any length of time, but considerable practice and experience are required. Consequently the failure to find the germs is no proof that the disease is not malaria. A single negative result of examination should not suffice, inasmuch as the organisms may at the time be absent from the peripheral circulation.

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[1] *Loc. cit.*, 88.

The study is best made a few hours before the paroxysm is expected. At its height they may not be discoverable. The finding of much blood-pigment free in the blood is suspicious. On the other hand, a fever of intermittent type which is not cured by quinine is almost certainly not malaria, and even the more irregular and remittent types will generally respond to this therapeutic test, if sufficiently large doses are administered. It must be remembered, however, that the cure, although usually prompt, is not invariably so.

There are a number of disorders which simulate malaria closely, prominent among them being *suppurative conditions*, such as empyema, pyelitis, and septic processes of any sort. These may exhibit an intermittent fever with chills and sweats. Careful local examination may be required to show their true nature. The failure of quinine to relieve them permanently, and the presence of leucocytosis, show that they are not malaria even without an examination of the blood for the parasites being made. Malarial fever may also be closely simulated at times by *tuberculosis* and by *typhoid fever*. The latter, especially, may be very like the remittent form due to the æstivo-autumnal parasite, and sometimes, too, strongly suggests the intermittent type of malaria. (See Typhoid Fever, p. 450, Fig. 99.)

Rickets, *syphilis* and forms of *splenomegaly* may exhibit both anemia and enlargement of the spleen, but have nothing else to suggest the malaria cachexia.

It was a habit very common in former years of attributing to malaria numerous obscure conditions for which no satisfactory explanation could be found. This has been the cause of many a fatal error in diagnosis. No doubtful case should be called malaria unless an examination of the blood or successful treatment with quinine shows that it really belongs in this category.

Treatment. **Prophylaxis.**—This consists in the destruction of the anopheles, the protection from the bites of the insects, and the prevention of relapse in those who have had the disease. The first of these has been successfully undertaken on a large scale by governments in some regions. All accumulations of water or other breeding places should be sprayed with kerosene. The avoidance of relapse is to be accomplished by the frequent administration of small doses of quinine, especially at the time of the year when malaria is most liable to occur. Individuals who are obliged to visit a malarial region should take quinine in small doses constantly. Mosquitoes should be kept as far as possible from biting by the use of mosquito-nettings at night; by seeing that the children are in the house before sunset, since the anopheles do their biting at night; and by applications to the skin of an ointment containing pennyroyal, menthol, tar, citronella or other substance disagreeable to the insects.

Treatment of the Attack.—Quinine is a specific, and the only one, although good results have been reported from arsenic in large doses. To subjects old enough it can best be given in capsules; for those younger it must be in solution. Either the sulphate (74.31 per cent. quinine), the bisulphate (59.12 per cent.) the hydrochloride (81.71 per cent.) or the dihydrochloride (81.61 per cent.) may be employed. For solutions the last is the best, as it is very soluble and is fairly rich in the quinine base. The sulphate is comparatively insoluble. The taste of these salts may be disguised to a certain extent by the admixture of syrup of yerba santa, syrup of licorice, syrup of chocolate, or other pleasant menstruum. The tannate (30 to 35 per cent. quinine) is much less effective, but in the form of "quinine chocolates," has very little unpleasant taste and is often taken readily by children. Quinine ethylcarbamate (aristochin) (96 per cent.) is a comparatively tasteless quinine derivative which may be used instead of those mentioned.

Quinine should be given, when possible, in a single large dose 2 or 3 hours before the time of sporulation of the parasites, which coincides with the occurrence of the paroxysm, since at this time the germs are outside of the red blood-cells and are more easily killed. A great objection to this method is the tendency to cause vomiting. When this occurs it is better to administer the drug in smaller doses every 2 or 3 hours beginning 3 or 4 hours after the paroxysm is over and giving, if possible, a slightly larger amount shortly before the time of the expected recurrence.

In the irregular or remittent forms, especially in serious cases, the patient should receive a large dose as quickly as possible, and then be kept fully under the influence of the drug by smaller doses repeated at regular intervals. A somewhat larger amount is generally required in such cases, particularly if the organism is of the æstivo-autumnal type. Sometimes quinine is borne better immediately after food, sometimes on an empty stomach. When vomiting cannot be overcome, and in all pernicious cases, the remedy must be given by the bowel, either by enema or suppository, or parenterally. For hypodermic use the dihydrochloride of quinine, or the hydrochloride of quinine and urea, may be employed. The hypodermic employment of quinine is, however, liable to be very irritating, and may be followed by abscess. The irritation is less when the drug is given intramuscularly. Intravenous administration of the two preparations last mentioned can be employed when rapid action is needed or when other methods fail. After the attack of malaria has been controlled quinine should be continued in somewhat smaller doses for 2 or 3 weeks longer, or relapse will be very liable to occur.

Dose.—Quinine is borne by children in relatively large doses, and, except for the tendency to produce vomiting, appears to be a safe remedy. Taking the sulphate as the type, and selecting that one of the salts to be used with due regard to its comparative basic strength, an infant of 1 year, with a mild attack of malaria, should receive 6 to 8 grains (0.39 to 0.52) in the course of 24 hours, and sometimes much larger amounts are needed. In later childhood the dose may be as large as for adults. The dose for parenteral use for 24 hours should be about the same as for administration by the mouth. For rectal employment it should be 2 or 3 times as large, since absorption is not perfect. Arsphenamin or its modifications and methylene blue have been recommended as substitutes for quinine, but offer no special advantages.

Treatment of the Paroxysm.—Little is needed as a rule, and that is purely symptomatic. Warm covers and dry heat may be employed in the cold stage, and sponging, if necessary, in the hot stage.

Treatment of Chronic Malaria.—Not only is quinine needed here, but often such tonics as iron, arsenic, and the like, to overcome the anemia. Removal from a malarial region is all-important. The spleen has been removed in some cases of chronic malaria, but this does not offer a certain promise of cure[1] (Cignozzi). Roentgen-ray treatment of the spleen in chronic malaria causes diminution of its size and in some cases a cure of recurrent, more acute attacks.[2] It should not be employed in acute malaria.

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[1] Il Policlinico, Sezion. Chirurg., 1923, XXX, 57.

[2] Italian Official Commission. Il Policlinico, Sezion. prat., 1923, XXX, 53.

CHAPTER XVII

RABIES. RAT-BITE FEVER. TULAREMIA

RABIES

(Hydrophobia, Lyssa)

Etiology.—Rabies is an acute infectious disease communicated to man by the bite of other infected mammals. Dogs are especially prominent in transmission, both on account of their opportunity for attack on numbers of individuals, particularly children, and because of their great susceptibility to the disorder. It may occur, however, in other domestic animals, such as the cat, horse and cow, and in many wild animals, as the wolf, skunk and fox. In fact no species of mammal is immune.

No microörganism has as yet been demonstrated as the cause of the disease, although the specific virus may be found in the saliva and the central nervous system. It is transmitted from the wound of entrance to the central nervous system along the nerve-sheaths, a fact which explains the shorter incubation-period occurring after bites of the head and arms as compared with those of the lower extremities. So, too, children, on account of their smaller stature and the shorter distance that the virus has to travel to reach the central nervous system, have a more rapid onset of symptoms than do adults. The possibility of more numerous bites in the former, and thus the introduction of a larger amount of virus, helps to shorten the incubation period. Babes[1] gives an average incidence of 24 per cent. in those bitten by rabid dogs, according to the estimations from his tables made by Williams.[2] The bite of the cat and especially of the wolf, however, causes relatively more infections. At least part of the low morbidity is explained by the action of clothing in wiping off the virus, and it is noticeable that wounds on uncovered parts are particularly fatal. Punctured wounds with the possibility of sealing in of the virus are more dangerous than lacerations, unless the latter are extensive and offer a large surface for absorption. For the reasons given, the period of incubation is quite variable and may be as short as 2 weeks, although the average is about 6 weeks. It is said that several months, or even a year or two, may intervene between infection and onset of symptoms, but the probability of the development of the disease after 3 months is slight. Rabies is not more frequent in animals in summer, the incidence in humans being greater at that time only because of the greater opportunity for transmission resulting from the lack of protection by heavier clothing.

Pathological Anatomy.—The important feature here in both man and animals rests in the lesions of the central nervous system. Minute hemorrhages and areas of softening may be seen. The so-called "Rabic tubercles" of Babes,[3] consisting of groups of small round-cells surrounding the blood-vessels and nerve-cells of the spinal cord, have been considered characteristic by some authors, but they are not always present. The bodies first described by Negri[1] can be found in almost all cases and are pathognomonic. They consist of small oval or round bodies 10 to 15μ in diameter or larger and with one or more nuclei, are found especially in the large ganglion-cells of the hippocampus major, but are often wide-spread throughout the cellular nervous tissue. By some they have been thought to be protozoa and the cause of the disease, and by others as areas of focal necrosis in the cells. There is often observed the disappearance of the large cells in the cerebrospinal ganglia and their replacement by small round-cells. The other findings are those of an encephalitis and a myelitis.

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[1] Traité de la rage, 1st ed., 1912.

[2] Therapeutics of Internal Diseases; Blumer, Billings, Forchheimer, 1924, III, 102.

[3] *Loc. cit.*

Symptoms.—In the *early* stage, lasting about 24 hours, nervous excitability is manifested by irritability and apprehension. General evidences of infection are present, such as slight fever and anorexia, rapid pulse and the like, and the site of the bite may become inflamed and painful or numb. Hoarseness and the characteristic difficulty in swallowing soon appear, and shortly any attempt at drinking, or even external stimuli such as noises or the sight or suggestion of food and water, produce violent laryngeal and pharyngeal spasms. It is from this characteristic that the popular name Hydrophobia is derived. Drooling occurs because of the inability to swallow the saliva. The *second* stage, that of excitement, has now developed, and the nervous symptoms increase in severity and mania may occur, although usually the patient is rational between the spasmodic attacks. Humans seldom injure or attempt to bite attendants. The hoarse sounds made with the contracted larynx have given rise to the false idea that the patient barks. The temperature in this stage may reach 102° or 103°F. (38.9° or 39.4°C.). After 2 or 3 days the patient passes into the *third,* or paralytic, stage in which unconsciousness soon appears and death occurs within 24 hours from syncope. The mortality is practically 100 per cent.

Diagnosis.—A differential diagnosis should be made from a hysterical condition (*lyssophobia*), in which a neurotic individual who has been bitten by a dog and dreads the possibility of the development of rabies presents some of its symptoms. This would be unlikely to occur in a young child. In older children it could be recognized by the lack of severity of the symptoms, as well as the continuance of them beyond a few days, and the final recovery of the patient. Tetanus might give rise to confusion if it is of the type in which only the muscles of the head are involved and laryngeal spasm is associated. Diagnosis of the condition of the attacking animal is of great importance. This should be made by the history of its behavior and by observation of its symptoms, death occurring in the animal in less than 5 or, at most, 10 days. For these reasons it should not be killed, as well as because the Negri bodies in its central nervous system are more likely to be found in the later stages than at the onset of symptoms. Inoculation of rabbits with infected tissue of the central nervous system will produce the disease in them, but only after 2 or 3 weeks. Many cities and practically all States make provision for the examination of animals for rabies. The head and neck should be sent packed in ice, or the brain and medulla placed in equal parts of glycerine and water, the mixture having been previously sterilized by boiling.

Treatment.—The most effective *preventive* treatment is the systematic muzzling of all dogs. It is also possible to vaccinate dogs against rabies. This is done by a single injection or by dividing the dose into

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[1] Zeitschr. f. Hyg., 1903, XLIII, 507.

6 parts. It is not protective against all strains of virus. In the case of children who have been bitten by an animal, the wound should be cauterized as soon as possible with nitric or carbolic acid or formalin, punctured wounds having first been laid open and bleeding encouraged. A diagnosis of the condition of the attacking animal is ascertained in the manner described above, but in suspicious cases the preventive inoculations of Pasteur[1] should be begun without delay. These consist in the daily injections of attenuated virus obtained by drying the spinal cords of infected rabbits, an emulsion of those which have been dried the longest being used in the earlier injections and about 20 treatments given in all. Several other methods of preparing the vaccine are now recommended, such as killing it by phenol or dialyzing it. In cases where the treatment has been started late, or where the wound has been on the face or hands, the first 4 or 6 injections are given at 12 instead of 24 hour intervals. Of 793 patients at the Pasteur Institute of Paris treated in 1923, none died (Viala).[2] The protection afforded by the inoculations is probably not of more than a year's duration. If the Pasteur treatments cannot be obtained from the health-department of the city or state they may be purchased from several of the large drug-manufacturers. A serum obtained from vaccinated animals has also been used in prevention in combination with the attenuated virus. The use of this serum alone has been tried in the treatment of active rabies but without avail.

The treatment of the attack already developed can be only palliative. Quiet in a darkened room with avoidance of stimuli of all kinds, the frequent hypodermic injection of morphine in full doses, and inhalations of chloroform during the spasms constitute all that can be done.

RAT-BITE FEVER

This disease is common in Japan, and has been found at times in Europe following lines of marine traffic. Strauch and Bissell,[3] in a review of the subject, state that about 86 instances have been reported in the United States, to which number they add another case; and Reuben and Steffen[4] record 3 more. Dembo[5] and his colleagues report a case in an infant of 7 months. This article contains a good discussion and bibliography.

Etiology.—The bite of an infected rat transmits the disease. It is probable that the infective agent is a spirochete called the *spirochœta morsus muris* (Futake *et al.*).[6] This organism has been found by different observers in many of the patients, and immune bodies have been demonstrated in the blood of those ill with the disease.

Symptoms.—The period of incubation is about 1 to 3 weeks. The onset is sudden, with chills, fever, and inflammatory reaction in the region of the rat-bite. The latter may become so marked that necrosis follows. The fever is intermittent, with febrile periods of 2 to 4 days and afebrile periods of 3 to 5 days. The temperature varies from 101° to 105°F. (38.3° to 40.5°C.). Usually at the time of fever there is a peculiar rash observed, consisting of bluish-red, slightly raised areas varying in size from ¼ to ¾ inch (.6 to 1.9 cm.) scattered over the neck, trunk and extremities.

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[1] Compt. rend. acad. d. sc., Paris, 1885, CI, 765; 1886, CII; 459, 835; 1886, CIII, 777.

[2] Ann. de l'inst. Pasteur, 1924, XXXVIII, 648.

[3] Arch. of Pediat., 1924, XLI, 315.

[4] Arch. of Pediat., 1924, XLI, 499.

[5] Amer. Jour. Dis. Child., 1925, XXIX, 182.

[6] Jour. Exper. Med., 1916, XXIII, 249.

Prognosis.—The disease is usually self-limited after a course of about 2 months, but some cases are long-continued and result in anæmia and malnutrition in much the same way as malaria. The mortality without treatment is about 10 per cent.

Treatment.—Cauterization of a rat-bite wound is indicated immediately after its occurrence. The treatment is symptomatic unless the infection is severe or long continued, in which event the employment of intravenous injections of arsenical preparations is effective.

TULAREMIA

Tularemia is a disease of squirrels (McCoy and Chapin)[1] and rabbits (Wherry and Lamb),[2] caused by the *Bacterium tularense*, which may be transmitted to man by handling infected rabbits or by the bite of a blood-sucking insect such as the horse-fly. The first human cases in this country appear to have been reported by Pearse[3] in Utah, and the disease has appeared in Ohio, West Virginia, District of Columbia, California, Idaho, Wyoming, Colorado, Indiana and North Carolina. It appears to be identical with Ohara's disease in Japan (Francis and Moore)[4]. Many cases are probably not recognized, being mistaken for other diseases such as typhoid fever, septicemia, or the like. Clear descriptions of tularemia are given by Francis[5] and Verbrycke.[6]

Symptoms.—A characteristic local lesion appears 4 or 5 days after infection. It consists of a dark or black center, which later becomes necrotic and is surrounded by an area of very mild inflammation. The neighboring lymphatic glands are decidedly enlarged and not frequently suppurate. The onset is accompanied by chills and fever, the latter continuing for from 1 to 4 weeks and in severe cases rising to 104°F. or 105°F. (40° or 40.5°C.). The temperature-chart resembles that of typhoid fever, except that there is a rather characteristic fall to normal on the 3rd or 4th day of the disease quickly followed by a rise. There is marked prostration and the usual symptoms attending any febrile state. Local pain in the involved lymphatic glands is often present. The **diagnosis** is suggested by the history of contact with rabbits, and by the characteristic local lesion with glandular enlargement. The white blood-cells number about 12,000 or 14,000 and the blood-serum of the patient will give an agglutinative reaction with the *Bacterium tularense*. The **prognosis** is not grave, as the disease may run a mild course; although in severer cases there is marked prostration and a prolonged convalescence. One fatal instance has been reported (Verbrycke). The **treatment** is symptomatic. Shelton[7] believes that quinine may be useful.

[1] Jour. Infect. Dis., 1912, X, 61.
[2] Jour. Infect. Dis., 1914, XV, 331.
[3] Northwest Med., 1911, III, 81.
[4] Jour. Amer. Med. Assoc., 1926, LXXXVI, 1329.
[5] Jour. Amer. Med. Assoc., 1922, LXXVIII, 1015; 1925, LXXXIV, 1243.
[6] Jour. Amer. Med. Assoc., 1924, LXXXII, 1577.
[7] Jour. Amer. Med. Assoc., 1925, LXXXIV, 1019.

CHAPTER XVIII

TETANUS

(Lock Jaw)

History.—The disease was known to the ancients and was described by Hippocrates. It occasionally attacks older children, the cause and symptoms being the same as in the case of adults; while in the new-born it is sufficiently common and important to receive in many text-books a separate description under the title of Tetanus Neonatorum.

Etiology. Predisposing Causes.—One of the most important is that of *age*, the great majority of cases in infancy occurring in the new-born. The influence of age is, however, greatly modified by locality. In some countries tetanus neonatorum has at times been endemic, and the cause of many deaths. These regions are often widely separated and of entirely different climatic conditions. Thus, it has in certain periods been very frequent in the Hebrides, in parts of the West Indies and of the Southern United States, and in a portion of Long Island. Of 23,398 infants dying in Roumania in the 1st month of life, according to Miron,[1] 10,257 were cases of tetanus. In general it is most frequent in hot climates. The disposition of the disease depends, however, not so much on climatic influences as upon the *absence of cleanliness*. Under proper hygienic conditions it is rare. The fact that the Negro infants in some regions of the Southern States are especially liable to it is not a proof of any special predisposing influence of race, but rather of the greater carelessness regarding hygiene common in these localities among the Negroes.

The *presence of a wound*, although minute and perhaps undiscovered, is apparently necessary to the development of the disease. From this standpoint all cases of tetanus must be considered as traumatic. Punctured, lacerated and contused wounds, especially of the hands and face, are those particularly dangerous; clean, incised wounds much less so. Consequently, cases frequently develop after Fourth of July accidents, or those associated with similar celebrations. I have seen it, for instance, follow a punctured wound by a nail in the ruins of a stable, this illustrating the connection of the disease with the horse. It is very probable that abraded surfaces of the mucous membrane of the intestine offer a portal of entry in some instances. It may follow burns. The wound of ritual circumcision or that of vaccination, if imperfectly cared for, may readily give rise to the disease. (See Vaccination, p. 432.) Much the most frequent portal of entry in the new-born is the umbilical wound.

Exciting Cause.—The direct cause has been proven to be the *bacillus tetani*, discovered by Nicolaier[2] in 1884 and isolated by Kitasato[3] in 1889. This is found in garden-soil and in the dust of the streets in nearly all countries, although particularly abundant in some localities. Derived from this source it is found, too, in the intestinal canal of some herbivora.

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[1] II Cong. d. Rüman, Gessellsch. f. d. Fortsch. u. d. Verbreit. d. Wissensch., 1903, Sept. 22. Ref. Schmidt's Jahrbücher, 1904, B. CCLXXXI, 206.

[2] Deutsch. med. Wochenschr., 1884, X, 842.

[3] Zeitschr. f. Hyg., 1889, VII, 225.

It is chiefly on account of the occurrence of the disease in cattle that the occasional danger from vaccine-virus arises when proper precautions have not been taken in its production. (See Vaccination.)

The bacilli inhabit the superficial portion of the wound of the patient, where an exceedingly virulent poison, the tetanus-toxin, is produced and whence very rapid absorption of this takes place. The poison exerts a special action on the motor cells in the medulla and on the anterior horns of the spinal cord. Nearly all investigators agree that the germs do not enter the blood or the organs. The disease is readily inoculated into animals. The spores of the bacilli are very tenacious of life, are uninjured by exposure to air or light, and offer considerable resistance to the temperature of boiling water. The action of disinfectant solutions also must be prolonged to accomplish their destruction.

Pathological Anatomy.—There are no characteristic post-mortem lesions. Congestion of the brain and spinal cord and of their membranes occurs frequently, but is probably the result of the convulsive condition rather than of the poison. The lungs are usually congested likewise. There is nothing peculiar about the condition of the wound.

Symptoms.—These vary slightly according as the disease affects the new-born or older children.

TETANUS NEONATORUM.—The **incubation** varies from a few hours to 10 or 12 days or, exceptionally, longer. Tetanus may occur several months after an injury has healed, if the wound is reopened. In the majority of cases symptoms appear toward the end of the 1st week of life. The **onset** is marked by restlessness, crying, sleeplessness and difficulty in nursing, the infant grasping the nipple and pressing it between the jaws and then dropping it with a cry of pain. On examination the masseters will be found contracted and hard and the jaws can be forced open only with difficulty (*trismus*). The eyes are closed, the forehead wrinkled and the lips pouting. In a few hours the stiffness extends to the muscles of the trunk and limbs, at times attended by arching of the body. To these symptoms are next added violent increase of the tonic contraction; this coming on in paroxysms which last a few moments, and which are repeated in severe cases perhaps every few minutes, but in milder cases at much longer intervals. They are brought on by such slight causes as movement of the body, a draught of air, and the like, or occur without any discoverable reason whatever. During well-developed paroxysms the surface of the body, especially the face, becomes red, cyanotic and swollen; the eyes injected; and the labial commissure pulled downward and outward, producing the *risus sardonicus*. (See Fig. 113, under Cerebrospinal Fever, p. 474.) The jaws are closed and foam comes from the lips; the head is retracted; the spine often much arched; the arms and thighs extended; the forearms and legs extended or slightly flexed; the thumbs bent into the palms; the fingers clinched; the toes flexed; and the muscles of the chest and abdomen hard. The whole body is as rigid as a rod of iron. Sometimes the infant is supported only on its heels and the back of its head. The respiration is irregular, superficial and difficult; swallowing impossible; the pulse rapid and weak, and the cry feeble. Jaundice is not uncommon. Slight clonic spasmodic movements sometimes accompany the attack.

The persistence of tonic contraction, especially of the trismus, appears to vary greatly with the case. In the severest it is practically unbroken almost from the beginning; in the milder ones complete relaxation occurs for quite long intervals. The temperature of tetanus is variable and often irregular; moderate fever or even normal temperature being the rule in the milder cases; higher fever and occasionally very high final temperature in the severer ones. Sometimes the low temperature of collapse develops.

Tetanus in Older Infants and in Childhood.—The symptoms differ in no way from those seen in adults. After a very variable **incubation** period, usually not over 10 days from the reception of a wound, **symptoms** appear, the patient complaining of chilliness, headache, fever, stiffness in the neck, and especially of difficulty in masticating and in opening the mouth. Gradual increase of stiffness in the muscles of the jaws develops, making it impossible for the patient to separate them; and the risus sardonicus appears. The stiffness then extends to the muscles of the body in general, especially those of the back. Distinct paroxysms now occur, similar to those described under tetanus neonatorum, the skin being bathed in perspiration; the pain very intense, and the body often in the position of opisthotonos, but sometimes in a straight position (orthotonos) or drawn to one side (pleurothotonos); or doubled foward by the contraction of the abdominal muscles (emprosthotonos). There may be partial relaxation between the paroxysms. Sometimes only certain regions are attacked, especially the head and neck. The condition of the mind is normal throughout; the eyes are usually not involved. In the acute, rapid cases the paroxysms are very frequent and the fever high; in the more chronic ones incubation is longer, the periods of relaxation between the paroxysms more prolonged and complete; and the duration of the attack greater.

Course and Prognosis.—The *duration* of fatal cases of tetanus is seldom more than 3 to 4 days; sometimes less than 24 hours or as long as 6 days. In cases which recover the attack may last several weeks, the paroxysms gradually diminishing in number and severity. The more severe the case, the more rapid is the course, and the more frequent the paroxysms. Death is the result of exhaustion, collapse, or of interference with respiration.

The *prognosis* of tetanus neonatorum is extremely bad. Death generally occurs. Probably the most favorable statistics give a mortality of nearly 50 per cent., and the usual death-rate is very much greater than this. Only the cases of long incubation and slow development of symptoms offer much hope from treatment. The mortality appears to be inversely proportional to the length of incubation. When under 5 days it is extremely unfavorable; decidedly more favorable when over 10 days.

In tetanus after the period of the new-born the prognosis is somewhat better, although still very grave. The absence of fever and the slow and late development of symptoms are the most favorable indications, the prognosis being very much better when incubation is over 10 days. The average death-rate varies from 50 to 80 per cent. Involvement of respiration is very unfavorable.

Diagnosis.—In well-marked cases the diagnosis is easy. The disease is closely simulated by the convulsions of *strychnine poisoning*, except that there is in this condition complete relaxation between the paroxysms, and that trismus rarely occurs, and certainly never early. Severe cases of *tetany* sometimes simulate tetanus, and I have more than once seen errors in diagnosis between the two diseases. In tetany, however, the muscles of the limbs are primarily and chiefly affected, and involvement of the jaws and of the neck seldom or never occurs. In cases of intermittent tetany the periods of relaxation are much longer and more complete than in tetanus. The peculiar electrical reactions of tetany are absent in tetanus. The history of the ailment is often serviceable in arriving at a conclusion. I have seen *meningitis* in rare instances strongly suggest tetanus. There is, however, in this the absence of trismus, and the presence of tenderness on moving the neck and of a disordered mental state.

Treatment. Prophylaxis.—Most careful antisepsis of the umbilical wound is the greatest safe-guard against tetanus in the new-born. In regions where the disease prevails, consideration must be given, too, to the character of the water employed for the first washing of the child, as well as the cleanliness of the towels, and the like. When a case of tetanus has occurred in a lying-in institution careful disinfection of the room is to be carried out, the infant isolated, and all other exposed infants given an immunizing dose of tetanus-antitoxin. (See Antitoxin-Treatment.

General Treatment of the Attack.—The first indication is antiseptic treatment of the wound, in order to prevent further production of the poison. The patient should be handled very little and kept as quiet as possible in a darkened room, in order to diminish the tendency to paroxysms. Drugs to quiet the nervous system should be given in frequently repeated doses, large enough to produce some physiological effect. Chloral is one of the best for this purpose. A child of 2 years may begin with 2 or 3 grains (0.13 to 0.19) every hour, the amount to be increased if necessary. Potassium bromide is another valuable remedy, the dose at 2 years being 8 to 10 grains (0.52 to 0.65), or eventually more, every 2 hours. When swallowing is impossible, remedies may be administered by the bowel or through a nasal tube. Physostigma has long been a favorite remedy. Its alkaloid, eserine, may be given hypodermically in doses of 1/500 of a grain (0.00013) at this age, repeated as needed. It is to be borne in mind that both chloral and eserine are depressant remedies. This statement merely indicates the necessity of care in their employment, not avoidance of them. Morphine hypodermically may be useful. The administration of an anæsthetic may be necessary during the paroxysms if they are severe. Feeding is important and may be accomplished by the rectal or nasal tube if the ability to swallow has ceased.

In recent years the value of magnesium sulphate for the control of the spasms appears to have been thoroughly demonstrated. It should be administered subcutaneously, and in severe cases intraspinally or intravenously. For subcutaneous injection the dose should be 0.6 to 0.8 c.c. (9 to 13 m.) of a 25 per cent. solution per kilogram (0.27 to 0.36 c.c. (4.5 to 5.5 m.) per pound) of body weight, 3 or 4 times a day (Meltzer).[1] Much larger doses have been used with advantage by some observers. Hotz[2] gave 10 c.c. (0.34 fl.oz.) of a 20 per cent. solution per rectum to an infant of 10 months with good results. For intraspinal and intravenous administration the solution should be weaker and the dosage much smaller. A survey of the reported cases in which the treatment has been tried is given by Robertson.[3] The treatment is not without danger of producing respiratory paralysis.

Antitoxin-treatment.—In 1890 Behring and Kitasato[4] were able to immunize rabbits against, or to cure them of, tetanus, by injecting a tetanus-antitoxin, and soon afterward antitetanic serum was employed in man and has been used repeatedly since that time. The experience of the World War has shown beyond question that, as a prophylactic, the serum is of undoubted value. To be of service, however, it should be administered as promptly as possible after the receipt of a wound. The practice approved by the Surgeon General's Department of the United States (Stone)[1] was to give 1500 units at once and to repeat this in 10 days. This was found very effective. Proportionately less should be used for children. The treatment is advisable whenever the wound is of a suspicious nature, as when punctured or contaminated by dirt or garden-soil. The prophylactic treatment will have been of value even should an attack of tetanus develop later, since it lengthens the period of incubation, and in that way favorably affects the prognosis. Paralysis has occasionally followed the injections. As a method of treatment for the attack itself the results have been uncertain, due probably to the fact that the union of the toxin with the cells of the nervous system occurs very early, and that it can then no longer be reached by the antitoxin. In order to neutralize any of the latter which is being formed and is still uncombined, the attempt should be made to saturate the patient with full doses, begun as early as possible and repeated every 4 to 6 hours. For adults 20,000 units may be given intravenously, followed by an equal amount intraspinally. A child of 10 years may receive 10,000 units; and a total of 50,000 to 100,000 units should be given in the first 2 or 3 days. These doses are larger than some clinicians think necessary. The antitoxin may be administered subcutaneously, intravenously or intraspinally. Under any condition, and at any time of life, at least half of the amount given should be by the intraspinal route. Nicoll[2] entertains doubt regarding the value of subcutaneous and intravenous administration, but thinks that that of the intraspinal method is beyond question serviceable. In spite of the difference of opinion regarding the value of the serum for treatment of the developed disease, with an affection so serious as this it should certainly be tried; but other methods should not be neglected.

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[1] Jour. Amer. Med. Assoc., 1916, LXVI, 931.

[2] Schweiz. med. Wochenschr., 1921, LI, 651.

[3] Arch. of Int. Med., 1916, XVII, 677.

[4] Deutsch. med. Wochenschr., 1890, XVI, 1113.

CHAPTER XIX

ACUTE POLIOMYELITIS

(Acute Infantile Paralysis; Meningo-encephalo-myelitis)

History.—There have been in the last few years such changes in our knowledge of this disease that we are now forced beyond question to place it among the acute infectious disorders.

We have no account of it until that of Underwood[3] in 1784, who, however, confessed entire ignorance of its cause and considered it not a common disorder. It was not until 1840 that a satisfactory description was given of it by J. Heine.[4] The disease was called the "essential

[1] Jour. Amer. Med. Assoc., 1922, LXXVIII, 1939.

[2] Jour. Amer. Med. Assoc., 1921, LXXVI, 112.

[3] Diseases of Children, 1789, Second edition, II, 53.

[4] Beobachtungen ü. Lähmungszustande der untern Extremitäten. Stuttgart, 1840.

paralysis of children" by Rilliet and Barthez[1] in 1843. The association with lesions of the anterior horns of the spinal cord was shown by Prévost[2] in 1865 and afterward, as a result of careful investigations, by Charcot and Joffroy[3] in 1870. The first study of its epidemic relations was made in Norway and Sweden especially by Medin.[4] As a result some writers follow Wickman[5] in naming it the "Heine-Medin disease." Upon the basis of the newer recognition of its pathology it is properly denominated meningo-encephalo-myelitis, but as this name is cumbersome the older title "poliomyelitis" (πολιος, grey) may be retained as representing the more common seat of the lesion; or that of "acute infantile paralysis," since the great majority of cases of acute paralysis in early life are to be classified here.

Formerly a malady of no great frequency as compared with many others, in more recent years its prevalence has increased enormously and the disease has taken its place among the common and serious disorders of early life.

Etiology. Predisposing Causes.—Of the predisposing causes *age* is of great importance. The great majority of cases occur between the ages of 1 and 5 years and especially in the 2d year of life. The following table by Frost,[6] illustrates this well:

Table 83.—Incidence of Poliomyelitis—United States

	New York Commission 729 cases percentage of total	Massachusetts Commission 615 cases percentage of total	Minnesota Commission 324 cases percentage of tota
Under 1 year	8.50	7.20	6.50
1 to 5 years	82.00	64.50	48.60
6 to 10 years	6.40	15.90	23.70
11 to 15 years	1.90	5.00	7.70
16 to 20 years	0.68	2.4	6.50
Over 20 years	0.40	5.0	7.0

European statistics give similar figures, although the percentage among adults is somewhat increased. This is well shown in the following table condensed from one given by Wickman.[7]

Table 84.—Incidence of Poliomyelitis—Sweden

	1025 Cases percentage of total
0 to 3 years	17.85
3 to 6 years	20.88
6 to 9 years	17.46
9 to 12 years	12.00
12 to 15 years	10.35
Over 15 years	21.46

Although less common in the 1st year, it may exceptionally occur even in very early life. Sinkler[8] observed 1 case developing at 6 weeks, and 2 at 3 months; and still younger cases are recorded (Gunewardene)[1] infant of 12 days). A comparative study of the reports of a number of observers shows a slight preponderance of the disease among males (Wickman).[2]

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[1] Maladies des enfants, 1843, II, 335.

[2] Comptes rend. de la soc. de biol., 1865, II, 215.

[3] Arch. de physiol. norm. et pathol., 1870, III, 134

[4] Nord. Med. Arkiv., 1896, VI, No. 1.

[5] Beiträge zur Kenntnis der Heine-Medinschen Krankheit, 1907

[6] Public Health Bulletin, 1911, No. 44, 16.

[7] Die acute Poliomyelitis, Berlin, 1911, 11.

[8] Keating's Cyclopedia Diseases of Children, 1890, IV, 685.

Season also is an important predisposing factor. Everywhere the majority of cases occur in the summer-time, the height of an epidemic in the northern hemisphere being July, August and September, the number diminishing rapidly after this; in the southern hemisphere February, March and April. *Geographical position* has no real influence, for although some parts of the world have suffered from it much more than others, the disease is now widely spread. *Residence* and *social position* are without much influence, poliomyelitis occuring in both cities and rural districts, although somewhat more frequent in the latter in proportion to the population. Other circumstances were formerly supposed to predispose, such as the occurrence of other infectious disorders, exposure to cold, over-exercise, and the like; but their action seems problematical. The *individual susceptibility* is not great. There seems to be a protective ability of the healthy mucous membrane to neutralize the virus (Flexner and Amoss).[3] The majority of those exposed, as far as this can be determined, do not contract the disorder. Herrman[4] estimates that only about 2 per cent. of children exposed acquire the disease. It is unusual that more than one child in a family is affected. In 7000 cases in the New York epidemic of 1916 there were 6521 which did not lead to a second case in the same family.[5] To this there are exceptions, however, especially during epidemics. Notable here is the record of the Swedish epidemic, in which repeatedly more than one child in a house was attacked by the disease. Of 1031 cases reported by Wickman[6] 627 houses had 1 case each; 95 houses 2 cases each; 39 houses 3 cases; 14 houses 4 cases; 7 houses 5 cases; 1 house 6 cases. Everhart and Cole[7] report 2 families, in 1 of which all of 6 children were affected and in the other 5 out of 7.

The *epidemic influence* has already been alluded to. Early in its history poliomyelitis occurred chiefly sporadically, and no thought of its infectious or epidemic nature was entertained. The first local epidemic recorded appears to have been one of 8 or 10 cases in a rural region of Louisiana reported by Colmer.[8] The first epidemic in Norway is said by Harbitz[9] to have consisted of 14 cases observed by Bull in 1868. After this, at intervals, small epidemics were recorded in different parts of the world, each confined to a small area and limited in number; much the largest being in the Otter Creek Valley of Vermont in 1894, with 123 cases (Caverly).[10,11] There were frequent local outbreaks in Scandinavia. In 1887 an epidemic of 43 cases occurred in Stockholm and was fully described by Medin.[12] In 1905 the disease became rather wide-spread over Norway and Sweden—719 cases being recorded by Harbitz and Scheel[13] in Norway and 1025 cases by Wickman[1] in Sweden. Up to 1907 Holt and Bartlett[2] were able to collect a total of 35 reported epidemics, chiefly small, equalling in all about 2000 cases; the majority of these in Scandinavia. After this year the number of epidemics and the total number of cases increased rapidly. In 1907 about 2500 cases occurred in New York City and vicinity.[3] In 1909 the disease spread rapidly over many parts of the United States, approximately over 9000 cases in 43 different States being reported in 1910 (Lovett and Richardson).[4] Meanwhile other epidemics of less size were observed in Australia, Scandinavia, England, Denmark, Cuba, Canada, Germany, and elsewhere, but nothing to be compared with the frequency of the disease in the United States, with the exception of Scandinavia. Here, according to Wernstedt,[5] a second great epidemic occurred in Sweden, from the beginning of 1911 to September 15, 1912 more than 6000 cases being reported; while in Norway in 1911, Johannessen[6] states there were not less than 1407 cases. In the summer of 1916 another very large epidemic occurred in the United States, chiefly in the northeastern portion, the principal locality being New York City and vicinity, in the city itself nearly 9000 cases being reported. In the same summer about 1000 cases occurred in Philadelphia (Le Boutillier).[7] In all there were about 24,000 cases in the United States in this epidemic (Emerson).[8] In 1917 there were at least 35,000 cases in the United States (Lavinder).[9]

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[1] Lancet, 1918, II, 847.
[2] Die acute Poliomyelitis, 1911, 12.
[3] Jour. Exper. Med., 1920, XXXI, 123.
[4] Jour. Amer. Med. Assoc., 1917, LXIX, 163.
[5] Committee Report. Med. Rec., 1917, XCII, 477.
[6] Beiträge zur Kenntnis der Heine-Medinschen Krankheit, 1907, 267.
[7] Atlantic Med. Jour., 1925, XXVIII, 269.
[8] Amer. Jour. Med. Sciences, 1843, V, 248.
[9] Jour. Amer. Med. Assoc., 1912, LIX, 782.
[10] Yale Med. Jour., 1894, I, 1.
[11] A later report (Jour. Amer. Assoc., 1896, XXVI, 1) gives the number of cases as 132.
[12] Nord. Med. Ark., 1896, VI, 1.
[13] Die acute Poliomyelitis u. verwandte Krankheiten, 1907.

Exciting Cause.—That the disease is an infectious one seems to have been first suggested by Strümpell,[10] and all recent clinical experience together with animal experimentation has proven the truth of this. The nature of the infectious element, however, has not been at all understood until within a few years. Of the many earlier bacteriological investigations, one of the most important was made by Geirsvold[11] who described a diplococcus found in the spinal fluid and the organs. Yet later investigations show that, whatever its significance, this is not the causative factor. In 1909 Landsteiner and Popper[12] injected into the peritoneal cavity of two monkeys the emulsified spinal cord of a fatal case of poliomyelitis, and succeeded in producing the disease in these animals. In the same year Flexner and Lewis[13] also produced the disease in monkeys, and were able in addition to transmit it indefinitely through a series of these animals. Later studies by Flexner and Lewis[14] showed that the virus was filterable, passing through a porcelain filter and consequently containing a germ which necessarily is exceedingly minute. That it is, however, a true germ was proven by the fact that the virus is destroyed by heat and by weak disinfectant solutions, such as peroxide of hydrogen, permanganate of potash, and menthol; and that it required a certain incubative time in animal experimentation for the development of symptoms. It withstands the action of glycerine and of freezing; and Römer and Joseph[1] found that it resisted the effect of drying for as long as 28 days. The virus has not been successfully transmitted to other animals than monkeys, with the possible exceptions of rabbits (Krause and Meinicke).[2] The virus is contained in the brain and spinal cord, salivary and lymphatic glands, tonsils, nasopharngeal and oral mucous membrane, intestines, and, early in the attack and to a very limited extent, in the blood and cerebrospinal fluid. It can be transmitted experimentally by inoculation into various regions of the body, including the brain, spinal cord, peritoneum and subcutaneous tissue, and can be given by introducing it into the stomach and intestines or by rubbing it upon the nasal mucous membrane. The monkeys never acquire it from each other by mere association. It has been found to persist on the nasal mucous membrane of the animal for as long as 5½ months after the attack is over. Osgood and Lucas[3] and Pettersson, Kling and Wernstedt[4] have found it in the washings from the nose of parents and other attendants upon the patient even 7 months after the occurrence of the attack. The question of the actual nature of the virus is brought nearer solution by the discovery in 1913 by Flexner and Noguchi[5] in cultures and in the tissues of the brain and spinal cord of very minute globoid bodies in which the power of infection appears to reside, and which would seem to be the actual germ of the disease. Rosenow, Towne and Wheeler,[6] Nuzum and Herzog[7] and Mathers[8] have found a pleomorphic streptococcus in the central nervous system of patients dying from the disease, and have been able experimentally to produce the typical symptoms and pathological changes in monkeys, and to recover the organism from the central nervous tissues of these animals. Rosenow[9] and Nuzum and Willy[10] have produced a serum from this streptococcus, and believe that the good effect of this in treatment supports the etiological significance of the germ (see p. 609). Whether this streptococcus is only a secondary invader or really a larger form of the globoid bodies described has not yet been definitely determined.

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[1] Beiträge z. Kenntnis der Heine-Medinschen Krankheit., 1907.

[2] Amer. Journ. Med. Sci., 1908, CXXXV, 647.

[3] Report on the New York Epidemic; by the Collective Investigation Committee, 1910, 27.

[4] Infantile Paralysis in Massachusetts during 1910, 55; 57.

[5] Jahrb. f. Kinderheilk., 1912, LXXVI, 605.

[6] Jahrb. f. Kinderheilk., 1912, LXXVI, 603.

[7] Amer. Jour. Med. Sci., 1917, CLIII, 188.

[8] Bull. Johns Hopk. Hosp., 1917, XXVIII, 131.

[9] Bost. Med. and Surg. Jour., 1918, CLXXVIII, 747.

[10] Jahrb. f. Kinderheilk, 1885, XXII, 173.

[11] Norsk. Magazin for Laegevidenskaben, 1905, LXVI, 1280.

[12] Zeitsch. f. Immunitätsforschung, Orig., 1909, II, 377.

[13] Jour. Amer. Med. Assoc., 1909, LIII, 1639.

[14] Jour. Amer. Med. Assoc., 1909, LIII, 2095.

Method of Transmission. Infectivity.—The method of transmission in human beings and the degree of infectiousness are not clearly understood. It is probable that the virus enters the system by way of the nose—or perhaps the intestine—being acquired from the nasal mucous membrane of an affected individual. Flexner and Amoss'[11] experiments support the view that it reaches the nervous system through the lymph-channels and without involvement of the blood, and that the portal of entry appears to be the mucous membrane of the upper respiratory tract.

The conditions obtaining are very like those seen in cerebrospinal fever. In both diseases wide-spread epidemics may occur and the infectivity appear to be much increased; or the disease may develop sporadically and seem to be but little infectious. Both are transmitted in some way from the sick to the well, and probably either directly or indirectly, yet usually not more than one case occurs in a family, and cases of either disease treated in hospitals do not transfer the infection to others in the ward.

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[1] Münch. med. Wochenschr., 1910, LVII, 568, 945.
[2] Deutsch. med. Wochenschr., 1909, XXXV, 1825.
[3] Jour. Amer. Med. Assoc., 1911, LVI, 495.
[4] 15th Intern. Cong. Hyg. and Demog., 1912, I, 597
[5] Jour. Amer. Med. Assoc., 1913, LX, 362.
[6] Jour. Amer. Med. Assoc., 1916, LXVII, 1202.
[7] Jour. Amer. Med. Assoc., 1916, LXVII, 1205; 1437.
[8] Jour. Amer. Med. Assoc., 1916, LXVII, 1019.
[9] Jour. Amer. Med. Assoc., 1917, LXIX, 261.
[10] Jour. Amer. Med. Assoc., 1917, LXIX, 1247.
[11] Jour. Exper. Med., 1914, XX, 249.

That poliomyelitis may be transmitted by unaffected persons acting as carriers has been shown in many instances, notably in the local epidemics reported by Wickman[1] in Trästena in Sweden, and by Shidler[2] in York, Nebraska, respectively. These carriers are of 3 types; those who are suffering from the disease in an unrecognized, mild or abortive form; healthy individuals who have been in close contact with patients with the disease, and chronic carriers who have recovered from an acute attack. It is uncertain how frequently transmission by carriers takes place. Very rarely cases have developed after occupying a house which had recently sheltered a patient. This raises the question of the possibility of the spread of the disease by such other agencies as dust (Neustaedter),[3] bed-bugs, (Howard and Clark),[4] house-flies (Flexner and Clark),[5] domestic animals and the like. That some or all of these are factors of importance is possible. Rosenau[6] was confirmed by Anderson and Frost[7] in his belief that the infection might be carried by the stable-fly. In subsequent experiments such transmission has not been confirmed even by the original investigators, and it is quite improbable that the infection is communicated either by the bite of the fly, or by this insect or any other acting as a carrier.

At what period of the disease the infectiousness is greatest is still unknown. That it may persist for a considerable time after convalescence has already been pointed out (5½ months; Osgood and Lucas). Flexner and Amoss[8] make the statment that the greatest infectiousness is in the early stages.

Pathological Anatomy.—This has been studied with especial care in human beings by Wickman[9] and by Harbitz and Scheel;[10] and in monkeys by Landsteiner and Popper,[11] Flexner[12] and others. In brief, there is a disseminated lymphocytic infiltration of the pia and of all parts of the central nervous system, but chiefly in most instances of the grey matter of the cord, especially in the anterior horns. The changes are most marked in the lumbar and cervical enlargements. The earliest alterations in the spinal cord consist of congestion of the blood-vessels of the pia and nervous tissue, with small-celled infiltration and edema. Hemorrhages, small or more diffuse, sometimes occur into the grey matter. These changes are rapidly followed by degeneration and disappearance of the ganglion-cells of the grey matter, the result of the pressure by the inflammatory exudate and edema with the consequent interference with the circulation. The degree and extent of the affection of the ganglion-cells varies greatly, dependent upon the degree of involvement of the blood-vessels and of the general small-celled infiltration.

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[1] Beiträge z. Kenntnis der Heine-Medicinschen Krankheit., 1907, 150.

[2] Jour. Amer. Med. Assoc., 1910, LIV, 277.

[3] Jour. Amer. Med. Assoc., 1912, LIX, 785.

[4] Jour. Exper. Med., 1912, XVI, 850.

[5] Jour. Amer. Med. Assoc., 1911, LVI, 1717.

[6] Jour. Amer. Med. Assoc., 1912, LIX, 1314.

[7] U. S. Public Health Rep., 1912, XXVII, pt. 2, 1733.

[8] Jour. Exper. Med., 1919, XXIX, 379.

[9] Studien ü Poliomyelitis acuta. Arbeiten aus dem path. Inst. d. Universit. Helsingfors, 1905, I, 109.

[10] Path.-anatom. Untersuch. u. akute Poliomyelitis u. verwandt. Krankh., 1907.

[11] Zeitschr. f. Immunitätsforsch., Orig., 1909, II, 377.

[12] Jour. Amer. Med. Assoc., 1910, LV, 1105.

In some cases but few cells are involved; in others none at all can be found remaining in certain regions.

The white matter of the cord shows similar congestion, edema, and infiltration of the perivascular spaces, although to a much less extent, with small foci of cellular infiltration in the white matter itself. Changes like those in the white matter are seen in the spinal ganglia and sometimes in the anterior nerve-roots. In the medulla and pons there are lesions similar to those in the cord, but usually with less degeneration of the ganglion-cells. The cerebellum and cerebral hemispheres are not so often involved, but the changes resemble those described, consisting of congestion and hemorrhages, with small-celled infiltration of the perivascular spaces and of areas of the brain-tissue itself. As already stated, the anterior horns of the lumbar and cervical enlargements of the cord are the favorite seat of the lesions found; but the process is, in fact, a diffuse one, involving perhaps the entire cord, the meninges of the brain and cord, and many parts of the brain itself as well. Exceptions to the usual distribution are not infrequent, and the medulla, cerebellum. or cerebrum may suffer to a greater extent than the spinal cord.

Other organs show alterations in acute cases, these consisting of degenerative changes such as are seen in any infectious disorder, and situated especially in the liver, lungs, kidneys and myocardium. Hyperplasia of the lymphoid tissue of the intestine, spleen and mesenteric glands is common. The spleen, lymphatic glands and thymus may be much enlarged. Swelling of the tonsils, with purulent secretion, is frequent.

In cases of long-standing, autopsy shows the affected portion of the spinal cord shrunken and the nervous tissue replaced by sclerotic tissue and neuroglia. The anterior nerve-roots and the muscles exhibit degenerative changes and atrophy, fatty and connective tissue replacing the muscular fibres. The degree of these secondary nervous and muscular lesions corresponds to that of the primary involvement of the spinal cord. Consequently in the milder cases entirely healthy nervous and muscular fibres may be found associated with those more or less atrophied.

Symptoms.—A number of distinct types have been described in addition to the ordinary spinal form which has long been well-recognized. Of the various classifications proposed one frequently adopted is that of Wickman,[1] which divides the types into: (1) Ordinary spinal form; (2) progressive form; (3) bulbar form; (4) acute encephalitic form; (5) ataxic form; (6) meningitic form; (7) polyneuritic form; (8) abortive form. These types shade into each other, or may be combined to such an extent that a sharp differentiation may be impossible.

1. SPINAL FORM.—With the exception of the abortive form this is much the most frequent variety, 88.65 per cent. of Zappert's[2] 555 cases belonging to this category. Many of the statements which follow upon such topics as incubation, method of invasion, mortality, and many of the symptoms of the attack, and its duration and prognosis apply equally well to all forms of the disease.

Incubation.—This would appear to average a week or less, with a range of from 5 to 10 days or exceptionally shorter or longer. Aycock and Eaton[3] believe it to be from 10 to 18 days with an average of 14. In experimental work upon monkeys the incubation may vary through a still greater range. There are no symptoms during this period.

[1] Beiträge z. Kenntnis der Heine-Medinschen Krankheit, 1907, 10.

[2] Studien ü. d. Heine-Medinsche Krankheit, 1911, 34.

[3] Amer. Jour. Hyg., 1925, V, 724.

Stage of Invasion.—The onset is usually acute, with fever, headache, prostration, and occasionally convulsions; as may occur in any of the infectious diseases. Vomiting is a frequent early or even initial symptom, but is seldom often repeated. Constipation is the rule, but in some cases there is very severe diarrhea. The combination of the latter with vomiting is suggestive of some gastrointestinal affection. In many cases respiratory symptoms predominate, with coryza, conjunctivitis, or bronchitis, and including sore throat. Respiratory symptoms were most prominent in over ½ of Müller's[1] cases, but in the 400 cases analyzed by Wilson[2] they were not of frequent occurrence. Decided general hyperesthesia, sweating, and great nervous irritability are common and suggestive. The mind may be clear or there may be somnolence or occasionally coma. An early persistent drowsiness was present in 72 per cent. of Wilson's series. It is accompanied often by a remarkable degree of irritability when the child is disturbed. There is very commonly tenderness and stiffness of the neck and back, and pain on moving the body. The tendon-reflexes may in some cases be temporarily increased, but are soon lost. The temperature varies, ranging from 102° to 103°F. (38.9° to 39.4°C.) or occasionally higher; sometimes continuous; sometimes remittent; sometimes falling when the paralysis appears; and

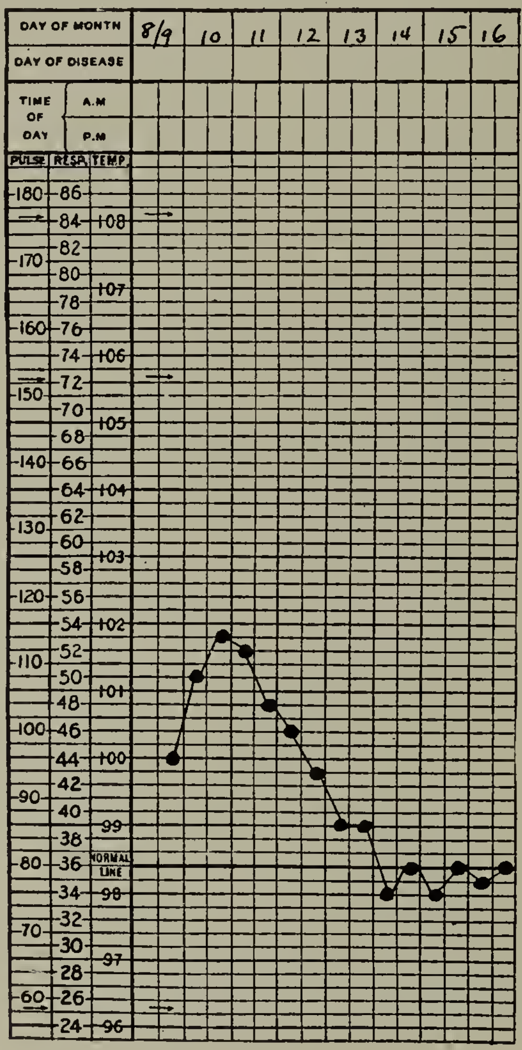

FIG. 162.

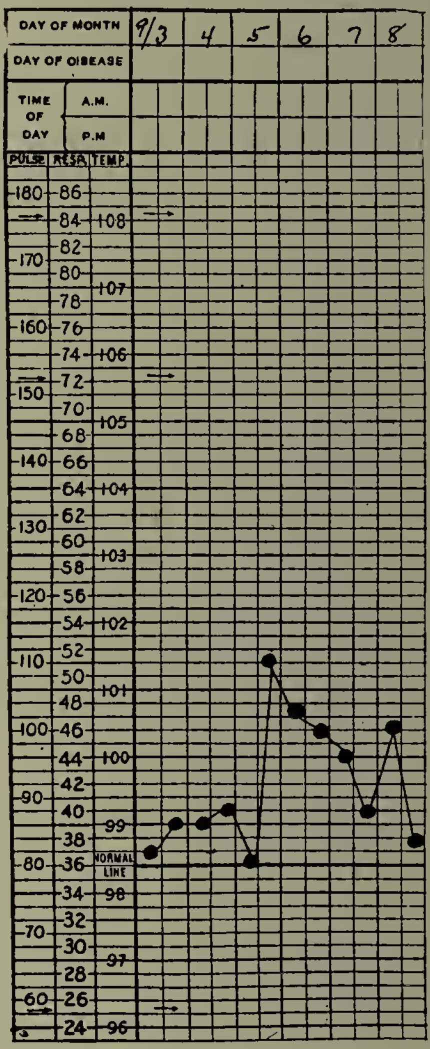

FIG. 163.

FIG. 162.—POLIOMYELITIS, ORDINARY SPINAL FORM.

S. D., girl, aged 6 years. Aug. 9, taken ill with loss of appetite, restlessness, constipation, tenderness and rigidity in neck, sweating of head, twitching and fever; Aug. 10, fever continued, weakness in both legs; Aug. 11, both legs absolutely paralyzed. Cerebrospinal fluid obtained under no pressure showed 36 cells to the c. mm. *Courtesy of Dr. M. Ostheimer.*

FIG. 163.—POLIOMYELITIS WITH LITTLE FEVER. DEATH.

Boy, aged 4 months. Extensive paralysis. Death on the 4th day. *Wickman, Beiträge z. Kentniss d. Heine-Medinsch. Krankh.*, 1907; 15.

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[1] E. Müller, Die Spinale Kinderlähmung, Berlin, 1910.

[2] Arch. of Pediat., 1916, XXXIII, 850.

continuing from 1 to 10 days (Fig. 162) with an average of 4 days (Wilson)[1] and falling by lysis or by crisis. In the mildest cases fever may be slight and of short duration or occasionally absent. Yet, on the other hand, severe, fatal cases may exhibit but little rise of temperature (Figs. 163 and 166) and those of much less severity may run a high febrile course (Fig. 164). The temperature in this disease is, in fact, entirely uncharacteristic. (See temperature-charts.) Yet more or less elevation of temperature is nearly always present. The pulse is accelerated, often out of proportion to the fever. The urinary functions are generally undisturbed, but retention is not infrequent, while incontinence is uncommon. Zingher[2] has pointed out the fact that there is an unusual susceptibility to the Schick test in subjects with poliomyelitis. Examination of the *blood* gives inconstant results. Occasionally there is decided leucopenia, with slight increase of the lympohcytes; but oftener this condition is absent and there is a moderate or even decided leucocytosis. Examination of the *spinal fluid* of the monkey 24 hours after inoculation, as demonstrated by Flexner and Lewis,[3] and of human cases very early in the prodromal stage shows a moderate increase in the number of cells, especially those of the polymorphonuclear type, which equal 80 per cent. or 90 per cent. of the total number. Promptly, however, before paralysis develops, the lymphocytes become predominant and soon number 90 per cent. or more of the cellular element. The total number of cells ranges from 15 or 20 up to 100, or exceptionally much more in the cubic millimeter; in contradistinction to normal fluid which contains only 5 or 10 cells. The fluid is clear or opalescent, forms a fibrin clot in some instances (Kolmer *et al.*)[4] and exhibits a moderate reaction for globulin. There is a prompt reduction of Fehling's solution. The cellular increase rapidly diminishes after paralysis has become evident, and by the end of the 2d week the numbers are normal. The globulin diminishes rather more slowly. The pressure is increased in over 90 per cent. of the cases (Larkin and Cornwall).[5] Jeans and Johnston[6] and Regan and Cheney,[7]

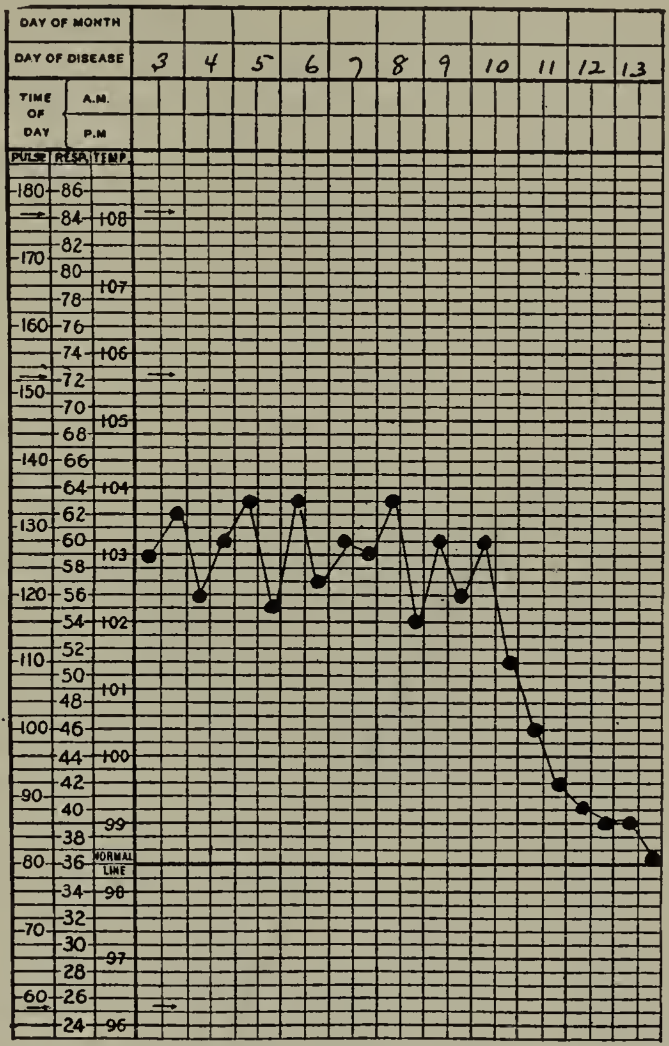

FIG. 164.—POLIOMYELITIS, SPINAL FORM. SEVERE INITIAL SYMPTOMS.

E. D., male, aged 2 years. Taken ill with loss of appetite, fever, great restlessness, profuse sweating, hyperæsthesia and pain on passive movement, slight angina. Paralysis appeared on the 3d day. Recovered with residual paralysis. (*Müller, Die spinale Kinderlähmung,* 1910, 45; 74.)

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[1] *Loc. cit.*
[2] Amer. Jour. Dis. Child., 1917, XIII, 247
[3] Jour. Exper. Med., 1910, XII, 227.
[4] Amer. Jour. Med. Sci., 1917, CLIV, 720.
[5] Arch. of Pediat., 1918, XXXV, 459.
[6] Amer. Jour. Dis. Child., 1917, XIII, 239.
[7] Amer. Jour. Dis. Child., 1922, XXIII, 107.

using the colloidal gold test, found an early transitory reaction in the syphilitic zone. That this is characteristic is not the experience of Larkin and Cornwall.[1]

The *duration* of the period of invasion varies. The average may be placed at 3 or 4 days, but there are many instances in which the prodromal symptoms are wanting, or so slight that they have been overlooked, and the paralysis comes on suddenly while the child is walking or sitting. In other cases the constitutional symptoms last only during the night and the child is found to be paralyzed in the morning. In still others symptoms may continue for a week before the paralysis appears. It has been pointed out by Kling and Levaditi[2] that there is not infrequently a free interval of from 1 to 3 days between certain transitory uncharacteristic symptoms and the onset of what are generally regarded as the symptoms of invasion.

Stage of Acute Paralysis.—At the close of the stage of invasion paralysis shows itself; generally at first as a decided weakness of some of the muscles, but increasing in degree and extent during 3 or 4 days. The tendon-reflexes are diminished or lost in the affected muscles. Exceptionally the development of paralysis occurs more slowly. The regions affected vary greatly. Much most frequently the loss of power is situated in one or both legs, the parts below the knee being oftener or more severely involved than those above. Not all the muscles of the limbs are affected, the peroneal muscles and the tibialis anticus and posticus being especially prone to be attacked. So, too, not all the fibres of any muscle participate in the process. Paralysis of one upper and one lower extremity is not uncommon, as is also a general paralysis of all four limbs, with some involvement of the neck and trunk. Less often one arm is attacked, the lower extremities being spared; and still less often both arms only. When the extremities of both sides of the body are involved, one side is almost always more severely affected than the other. Retention of urine is troublesome in some cases. The mind is clear, any initial drowsiness usually having disappeared.

As already stated, there may be a combination of some of the different forms of the disease. Thus it occasionally happens that paralysis of some of the extremities occurs in combination with a facial palsy. Some idea of the frequency of paralysis in different parts of the body may be obtained from the following statistics condensed from figures given by Lovett and Richardson,[3] based on 1158 cases:

TABLE 85.—REGIONS PARALYZED IN POLIOMYELITIS—AMERICA

	Per cent.
One leg only	27.97
Both legs	23.48
Both legs and arms	11.13
One leg and one arm, same side	9.49
One arm only	7.25
Both arms only	1.98
Face	6.38
Abdomen	5.78
Neck	0.94
Respiration	2.67
Deglutition	0.60

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[1] *Loc. cit.*

[2] Pub. de l'institut Pasteur de Paris, 1913. Ref., Haynes, Arch. of Pediat., 1917, XXXIV, 401.

[3] Infantile Paralysis in Massachusetts during 1910, 92.

European statistics may be compared by consulting the analysis given by Wickman,[1] but reduced to percentages, of his 868 cases with paralysis.

TABLE 86.—REGIONS PARALYZED IN POLIOMYELITIS—SWEDEN

	Per cent.
One or both legs	40.67
One or both arms	8.64
Combined paralysis of arms and legs	17.51
Legs and trunk	9.79
Arms and trunk	1.15
Trunk alone	1.04
"Whole body"	2.65
Ascending paralysis	3.69
Descending paralysis	1.50
Combined spinal and cerebral nerves	3.92
Cerebral nerves alone	2.65
Localization not defined	6.91

The *constitutional symptoms* of the stage of invasion persist during the first 3 or 4 days of the acute paralytic stage, but last very seldom more than 6 or 7 days in all from the onset, an often a decidedly shorter time. Pain, a frequent symptom, may, however, continue several weeks. There is no anesthesia. The superficial lymph-nodes are enlarged in 90 per cent. of the cases (Carr).[2] Generally after cessation of acute constitutional symptoms there is no addition to the paralysis. To this there are sometimes exceptions seen, and loss of power continues to increase for a few days longer. The condition of the spinal fluid in this stage has already been considered (p. 595).

Stationary Stage.—This follows the cessation of acute symptoms and lasts from 1 to 6 weeks. No change takes place in the paralytic condition except that atrophy rapidly develops; and no other symptoms of any sort are present, except in many cases a decided persistence of a degree of pain and of hyperesthesia. During the stationary period the loss of power seems often very extensive and complete. Paralysis of the muscles of the trunk is not infrequent; even those of the neck and abdomen and the muscles of respiration being more or less affected. In such cases of extensive involvement the child lies helpless in bed, and if lifted to a sitting position is unable to support the body or to hold the head erect. It is impossible at this period to predict how complete the paralysis will remain.

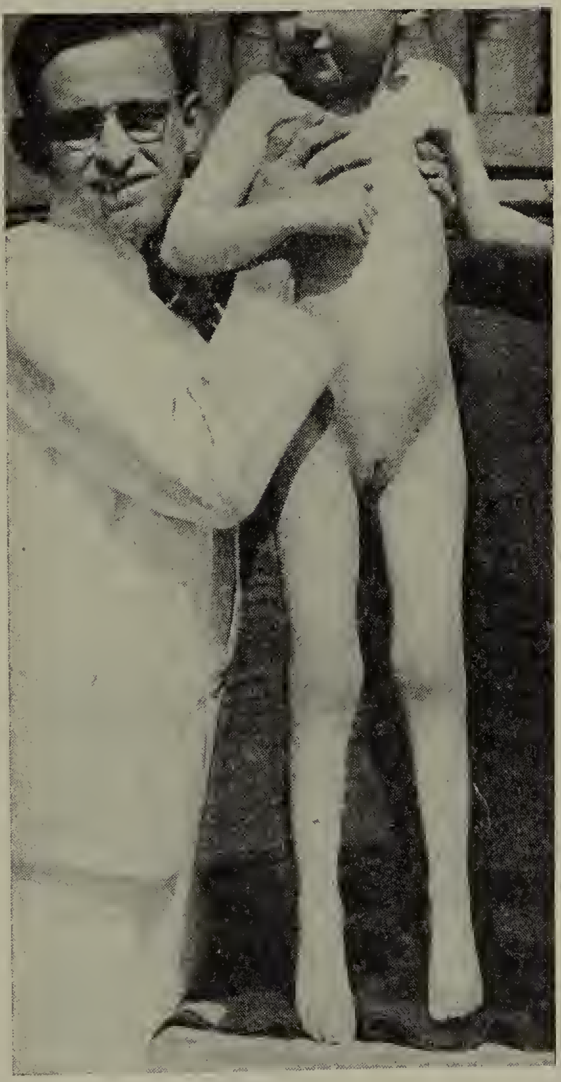

FIG. 165.—PARALYSIS IN POLIOMYELITIS.

Recent case in girl of 6 years. Complete paralysis in the lower extremities, with foot-drop. From a patient in the Children's Hospital, Philadelphia.

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[1] Die acute Poliomyelitis, 1911, 44.

[2] Arch. of Pediat., 1917, XXIV, 591.

Stage of Retrogression.—At the end of the stationary period improvement begins. The greater part of this will occur in the first 6 months, less in the next half year, but some degree takes place even up to the end of 2 years. Meanwhile atrophy becomes still more distinct in the muscular tissue which is not undergoing improvement, and by 2 months is very decided. The affected limb is then usually much smaller than normal in circumference, and peculiarly soft and flabby. There is no tactile anesthesia or analgesia. The paralysis is flaccid in nature (Fig. 165), there being diminished muscle-tonus and diminution or absence of tendon-reflexes.

Cases which at first seemed severely paralyzed improve usually greatly during this period of retrogression. Probably in these cases the muscular paralysis is dependent chiefly upon the pressure of the edema and of the cellular infiltration. Those muscle-fibres connected with ganglion-cells which have been destroyed can never regain their power. It is not possible early to determine how much of such actual destruction has taken place.

Chronic Atrophic Stage.—In the final condition the growth of the affected limb is much interfered with, the limb being both smaller in circumference and shorter than normal, the muscles greatly wasted and the ligaments much relaxed, allowing deformities to result from yielding of the articulations. The skin is cold, blue, and marbled, and the patient suffers from a sensation of coldness in the paralyzed parts. Contractions commence even during the stage of retrogression, and in the chronic stage are well-developed, the stronger muscles overcoming the weaker paralyzed ones. Talipes and lateral spinal curvature are among the commoner of the deformities. The parts of the body oftenest showing residual paralysis are one or both lower extremities, one limb being always worse than the other. Next most frequent is one upper, or one upper and one lower extremity. Thus the muscles oftenest and most severely invaded in the primary paralysis are those which are left paralyzed finally.

Electrical Reactions.—The importance of this matter makes it advisable to consider together the conditions observed in the different stages. By the beginning of the 2d week of the attack both faradic and galvanic contractility are lost in the severely paralyzed muscles. Very soon, oftenest in the course of the 2d week, there is a reappearance and then an increase in the galvanic response, with the presence of the reaction of degeneration. The galvanic contractility then diminishes, but still with the degenerative reaction which may last for some months. Finally, perhaps after 2 or 3 years, all electrical contractility of any sort disappears in the permanently paralyzed muscles. In those which recover, faradic contractility slowly reappears and the galvanic returns to a normal condition. In muscles but little affected faradic contractility may never be more than merely diminished.

2. PROGRESSIVE FORM (*Landry's Paralysis Type*).—This form, not a common one, is characterized by the rapid extension of the lesions until the medulla is involved. It was seen in 45 (4.39 per cent.) of Wickman's[1] 1025 cases. The paralysis generally begins in the lower extremities and ascends to the arms, involving the muscles of the trunk including those of respiration, and sometimes the diaphragm and even the muscles of deglutition. The course is generally rapid, death following in a few days (Fig. 166). Sometimes the arms are attacked first, and the extension is then of the descending type. It is probable that the majority

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[1] Die acute Poliomyelitis, 1911, 53.

of the cases which have been described as Landry's paralysis should be grouped here. (See Landry's Paralysis, p. 678).

3. The Bulbar or Pontine Form.—Formerly this condition was described as a distinct disease under the title of "Polioencephalitis superior or inferior," according to which of the nuclei were involved. The lesions may be limited to the nuclei of the cranial nerves, or may be combined with spinal lesions (Figs. 167, 168). The facial nucleus is the one oftenest affected, and the abducent and the hypoglossal frequently; but any of the cranial nerve-nuclei may be involved with the production of the corresponding symptoms, among them central paralysis of respiration, paralysis of deglutition, and disturbances of the heart's action. Although the bulbar paralyses generally recover completely, a paralysis, usually transitory, of some muscles of the limbs may sometimes be combined with a permanent facial palsy, and the diagnosis may later be difficult if the early history of the attack is not known. The bulbar

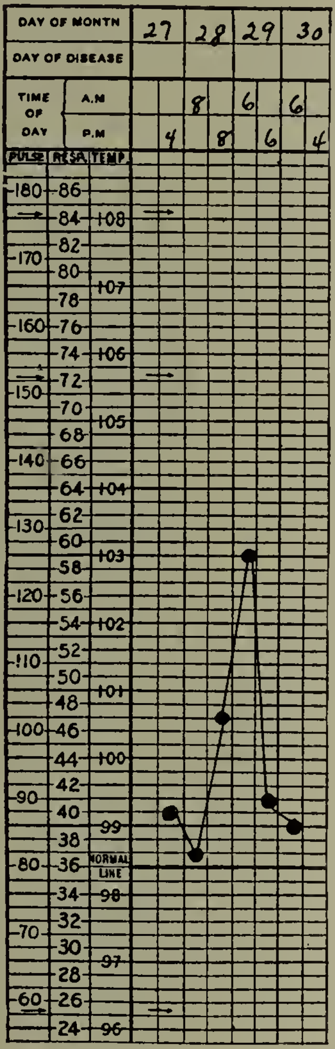

Fig. 166.

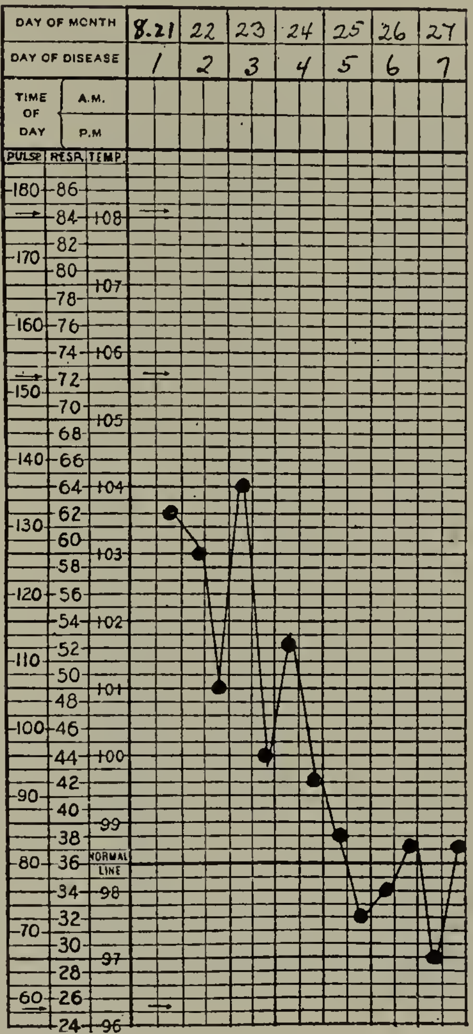

Fig. 167.

Fig. 166.—Poliomyelitis. Progressive Form.

E J., male, aged 10 years. Aug. 27, fever, malaise, headache; Aug. 28, slight restlessness; Aug. 29, nervous, head-sweating, neck rigid, fever. Weakness of left leg in morning, entirely paralyzed by noon. Right leg weak at 4 p. m., totally paralyzed at 8 p. m. Upper extremities and shoulder muscles paralyzed by evening; Aug. 30, paralysis of muscles of deglutition and respiration. Cerebrospinal fluid under increased pressure, 94 cells to the c.mm. Child conscious until 2 p. m., died at 5 p. m. *Courtesy of Dr. M. Ostheimer.*

Fig. 167.—Poliomyelitis, Bulbar Form.

Charles J., aged 4 years. Aug. 21. Tonsillitis on the 17th. Fever disappeared but returned today, with nervousness, dullness, vomiting, loss of appetite, dry tongue, constipation; Aug. 22, nearly comatose, muscular twitching, tympanites, high fever, increased knee-jerks, pain in arms and legs, slight paralysis of face. *Later.*—Little change for 5 days, except increasing paralysis of the right side of the face, and apparently some difficulty in swallowing. Then gradual improvement in all symptoms, with loss of knee-jerks. Very decided paralysis of the face remained, with slight weakness in the right leg.

form is not infrequently seen, 6.45 per cent. of Wickman's[1] 868 cases belonging here. According to McEachern[2] it may occur in epidemic form.

4. Acute Encephalitic Form.—The variety bearing this name was described by Strümpell[3] as "acute encephalitis or poliencephalitis of children," and its close relationship to, or identity with, poliomyelitis suggested. It is now generally recognized as a variety of poliomeylitis, this being confirmed by the identity in the nature of the anatomical lesions. It is one of the most unusual types of the disease. In it the

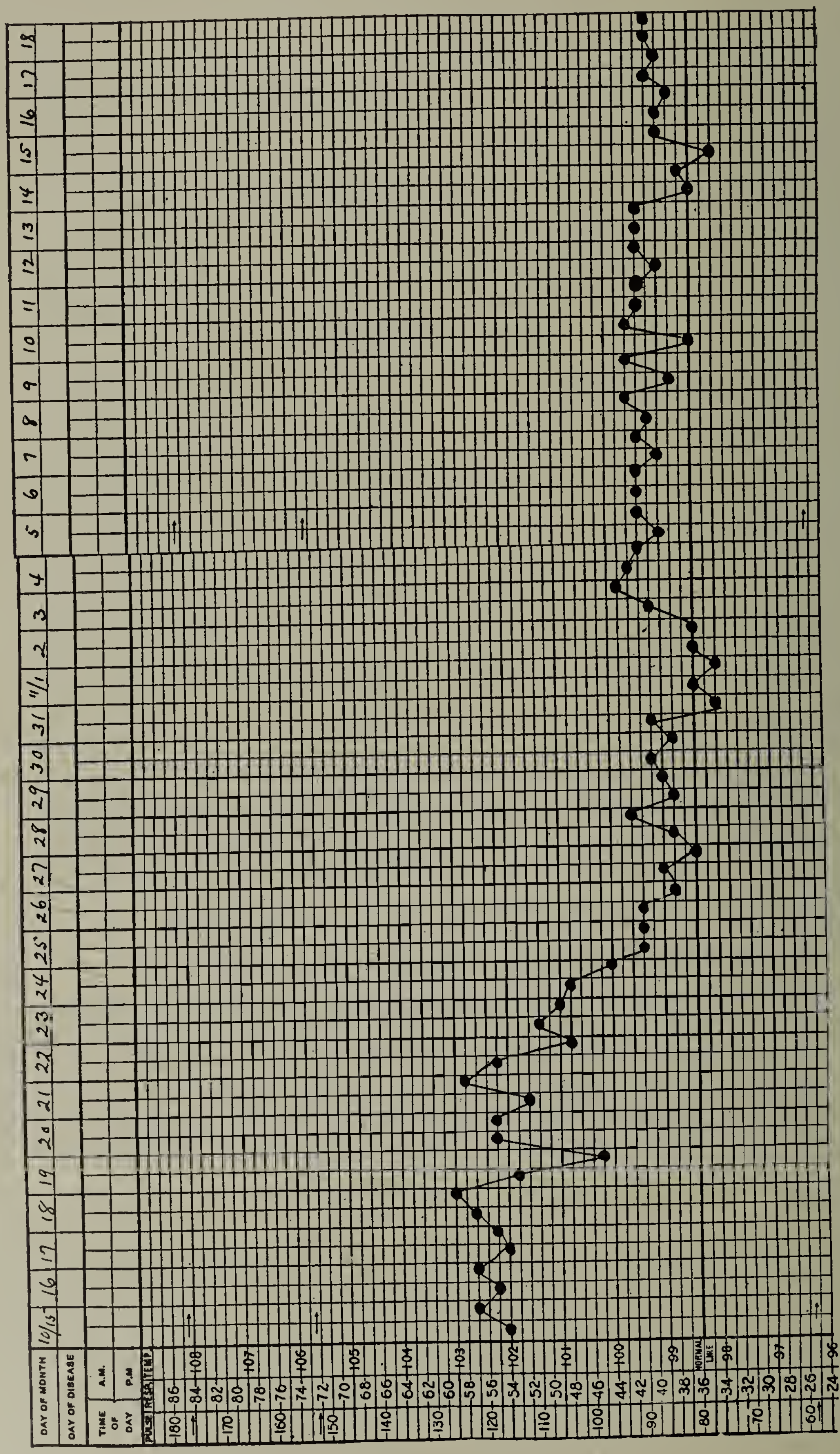

Fig. 168.—Poliomyelitis, Bulbo-spinal Form. Supposed to be Meningitis.

Betty B., aged 3½ years. Taken ill Oct. 15 with fever. Vomited on 2d day. Semicomatose for a week. Slight stiffness of neck, grinding of teeth, tache, occasional sighing respiration. Lumbar puncture done on three occasions gave slightly cloudy fluid in increased amount, increase of polymorphonuclear cells (90 per cent.), no germs. Antimeningococcic serum injected on ground that the case might be cerebrospinal fever. Not until convalescence began was there found extensive paralysis of the right upper extremity and to a less extent of the left, and transient paralysis of left external rectus and left side of face.

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[1] Die acute Poliomyelitis, 1911, 44.

[2] Jour. Amer. Med. Assoc., 1926, LXXXVI, 90.

[3] Jahrb. für Kinderheilk., 1885, XXII, 173.

grey matter of the cortex is probably involved. The early symptoms show great variation but suggest those of meningitis. The disease may begin with convulsions, which may be unilateral, somnolence develops, and a paralysis appears with *increase of tendon-reflexes.* Later the paralysis is found to be a spastic type and to some extent of hemiplegic distribution, showing its cerebral origin. This can be combined with flaccid paralysis located in other regions, and due to coincident involvement of the lower segment of the cerebrospinal system (Fig. 169). Fatal cases may occur without the development of any paralysis whatever.

5. THE ATAXIC FORM.—This is likewise an unusual form, seen in only 0.43 per cent. of Lovett and Richardson's[1] 1158 cases. In it ataxia is the chief symptom, oftenest of the lower extremities, or being more extensive; and this either occurs alone or is combined with cerebral symptoms, especially paralysis of the cranial nerves, and sometimes with a moderate degree of spinal paralysis as well.

6. THE POLYNEURITIC FORM.—The disease in this form simulates multiple neuritis so closely that its existence has been much disputed. It certainly is a rare variety. It is characterized by the severe pain and tenderness in the nerve-trunks and muscles. The pain may be present in the joints as well. The paralysis may sometimes be slight or transitory, or even overlooked, and the limbs held rigidly to guard against pain on movement. In other cases the paralysis is extensive. Disturbances of sensibility are usually absent.

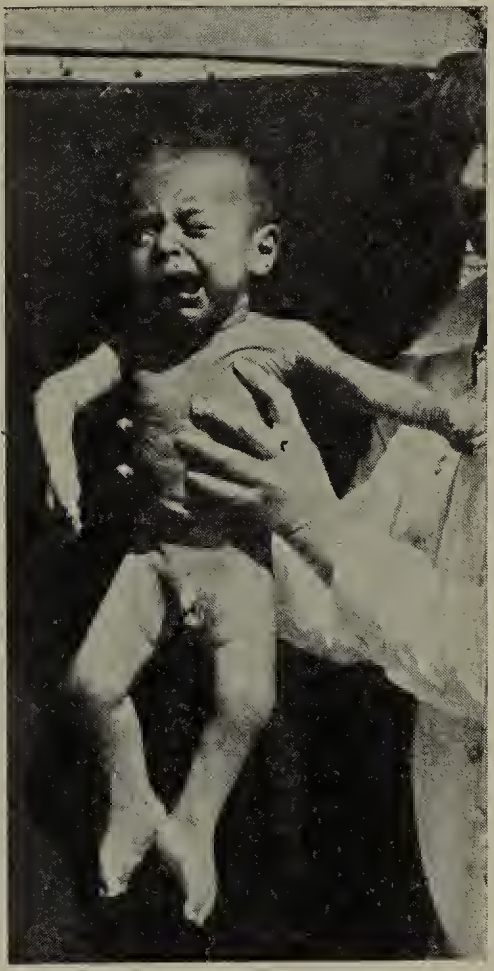

FIG. 169.—COMBINED LESIONS IN POLIOMYELITIS.

Infant of 3 months in the Children's Hospital of Philadelphia. Facial palsy, flaccid paralysis of the legs, spastic paralysis of the arms.

7. THE MENINGITIC FORM. (Fig. 170).—In this variety, not uncommon in epidemics, the very marked symptoms indicating implication of the meninges overshadow all other manifestations. There are vomiting, headache, pain and rigidity in the neck and back even with moderate opisthotonos, convulsions, delirium and coma. Not all these are present in any one case, and combinations of any sort are possible. A diagnosis can be made only if paralysis develops. The cases are often severe and death may occur in a few days.

8. THE ABORTIVE FORM (Fig. 171).—The frequency and even the possibility of such cases have come into prominence only in recent years. Their existence has been proven experimentally by Flexner and Clark,[2] and especially by Anderson and Frost[3] through the discovery of immunizing bodies in the serum of patients who had suffered from this form of the disease, and by the production of the disease in animals by the inoculation with the virus from abortive cases. There is abundant clinical evidence also, as seen in families where some cases are of the typical form and

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[1] *Loc. cit.*

[2] *Loc. cit.*, 585.

[3] Jour. Amer. Med. Assoc., 1911, LVI, 663.

others have only initial symptoms without any subsequent paralysis. The spinal fluid, too, exhibits the characteristic changes. The attack begins abruptly with the prodromal manifestations already described. There is fever of varying type, usually constipation, prostration, severe headache, and often vomiting. Frequently hyperesthesia is present with stiffness of the neck and pain here and in the back. Sore throat may be noted, or rhinitis and conjunctivitis. In some instances there is a muscular weakness or a certain unsteadiness in walking. The character of the symptoms varies decidedly, as in the initial stage of the ordinary form. Sometimes gastrointestinal manifestations prevail; sometimes respiratory; sometimes nervous. After continuing 2 or 3 days, there is rapid recovery. To this class of abortive cases might be added what E. Müller[1] has denominated the "*rudimentary form*"

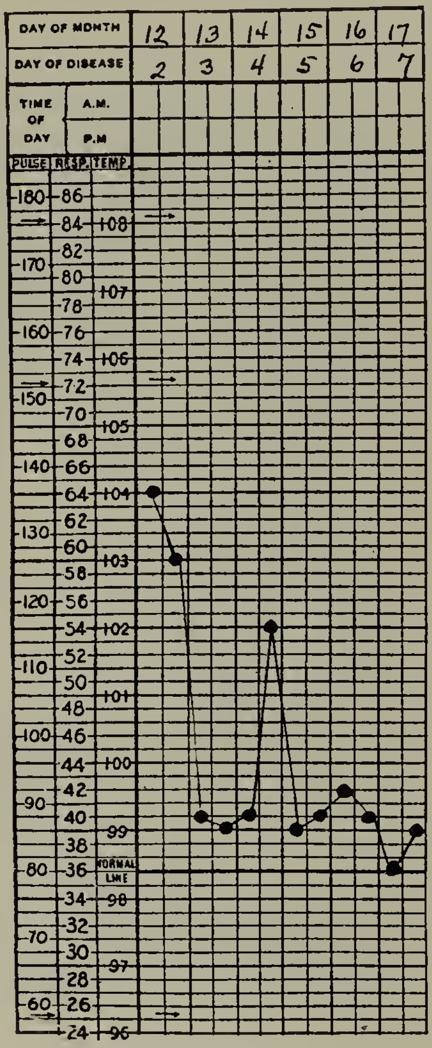

Fig. 170.

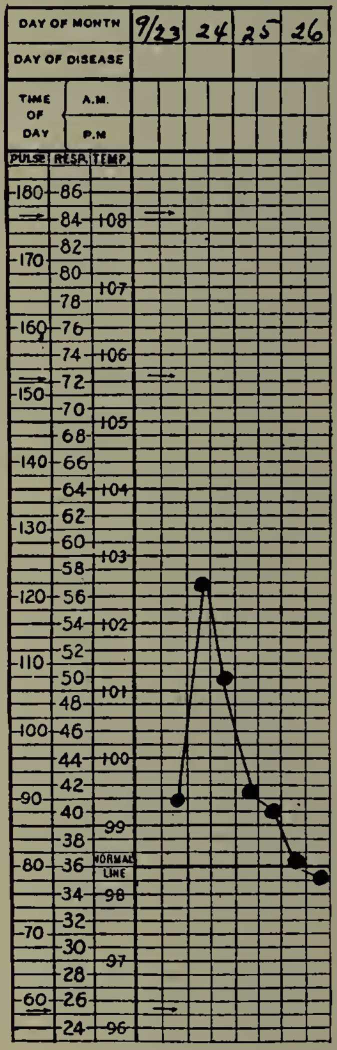

Fig. 171.

Fig. 170.—Poliomyelitis, Meningitic Form.

Boy, aged 8 years. June 11, attacks of headache and convulsions; June 12, somnolence, retraction of head, opisthotonos, pain on movement, rigidity of limbs, at times tremor, patellar reflexes appeared to be increased. *Later.*—Condition little changed for 4–5 days, then rigidity disappeared, somnolence persisted a few days longer. Recovery. No paralysis at any time. (*Wickman, Beiträge zur. Kentnss. der Heine-Medinsch. Krankheit,* 1907, 15; 100.)

Fig. 171.—Poliomyelitis, Abortive Form.

Walter B., aged 5 years. Illness began Sept. 22, during an epidemic, with fever, stiffness in feet, slight headache, constipation, and loss of appetite; Sept. 23, examination shows child drowsy, feverish, does not answer questions, throat red, slight tache, plantar reflexes slightly exaggerated, other reflexes normal. Spinal fluid under increased pressure, clear, cell-count 30, with 22 polymorphonuclear cells, no organisms; Sept. 24, some jerking of legs and arms; Oct. 2, child convalescing, but difficulty in straightening legs when sitting up, Kernig's sign present both sides, knee-jerks and plantar reflexes slightly exaggerated; Oct. 4, excellent general condition. No suggestion of paralysis.

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[1] *Loc. cit.,* 146

(Fig. 172), in which a very slight transitory paralysis is seen with temporary loss of the patellar reflexes. This is in contradistinction to cases without any evidences whatever of paralysis, which he calls the "*larval form.*"

Abortive cases are frequent in epidemics and doubtless often overlooked. Müller estimated that 50 per cent. of the cases of poliomyelitis are of this type, and of Wickman's[1] 1025 cases 157; *i.e.* 15.32 per cent., were of the abortive type. In the second great Swedish epidemic the abortive cases in some localities greatly exceeded those with paralysis (Wernstedt),[2] and the same is probably true of the 1916 epidemic in the United States.

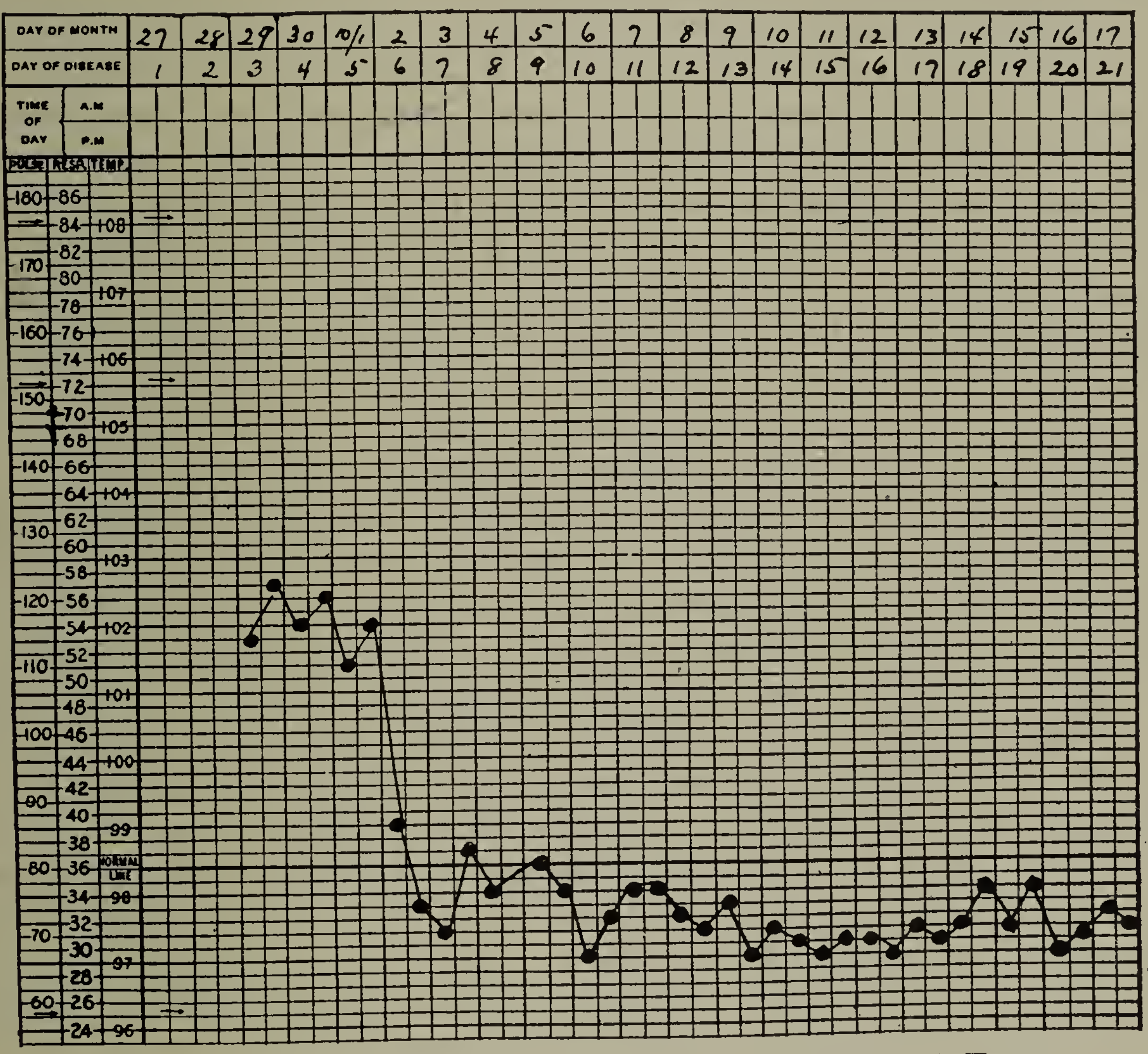

FIG. 172.—POLIOMYELITIS, ABORTIVE (RUDIMENTARY) FORM.

E. B., girl, aged 4 years. Onset with nausea, slight sore throat, headache and fever. Spinal fluid on 5th day showed no increase of pressure, leucocytes 36 to c. mm., with 63 per cent. lymphocytes. Apparently convalescent on 6th day. On 17th day slight weakness in both legs which disappeared by 21st day. *Courtesy of Dr. M. Ostheimer.*

Relapse.—Occasionally after the development of paralytic symptoms of moderate degree, there is an intermission of 6 or 7 days, after which there is renewed extension of paralysis with reappearance of other symptoms. In some cases this is probably only a later manifestation of the initial lesion; but in others there appears to be a true relapse occurring, it may be, weeks after the primary attack. Leegaard[3] reports a case probably of this nature in which there was an interval of nearly 3 weeks.

Recurrence.—A patient who has once suffered from the disease appears to be immune. Experimental work with monkeys likewise shows that the prompt injection of the blood of other monkeys or of human beings who have had an attack, will sometimes prevent or delay

[1] Beiträge zur Kenntnis der Heine-Medinschen Krankheit, 1907, 288.
[2] Jahrb. f. Kinderheilk., 1912, LXXVI, 605.
[3] Norsk. Mag. for Laegevid, 1901, XVI, 377.

the development of paralysis after inoculation. The immunizing principle has been found in the blood of individuals several years after the attack of the disease (Flexner and Clark).[1] There are, however, occasional exceptions to this rule of lasting immunity, and second attacks have been reported, as by Sanz,[2] Francis and Moncreiff,[3] Lucas and Osgood[4] and others.

Prognosis.—Formerly poliomyelitis was considered a disease not at all dangerous to life, and only to be feared on account of the lifelong crippling which it left. Experience in more recent years shows that under epidemic influences the mortality can be very decided. Thus Krause[5] reports a mortality of 15.14 per cent. in 436 cases; Zappert,[6] 10.99 per cent. in 555 cases; Lindner and Mally[7] over 16 per cent. in 96 cases; Wickman[8] 12.2 per cent. in 1025 cases or 16.7 per cent. if the 157 abortive cases are excluded; Johannessen[9] 13.08 per cent. in 1407 cases; Leegaard[10] 17.1 per cent. in 3290 cases. The American death-rate has been somewhat less. Lovett and Richardson[11] found a mortality of 7.9 per cent. in 1216 cases in Massachusetts, while in approximately 2000 cases in New York City the mortality was about 5 per cent.[12] A notable exception to the comparatively low mortality-rate was seen in the epidemic in New York State in 1916. In this there were approximately 13,000 cases, with a mortality of 25 per cent. (Nicoll).[13] The abortive cases, of course, give an entirely favorable prognosis. Those of the polyneuritic type may have paralysis persist, or there may be complete recovery. The ataxic symptoms of the form of the disease bearing this name are never more than a temporary matter. The fatal cases are seen especially in the progressive form, which nearly always ends in death, and in the acute encephalitic and the meningitic forms, both of which are serious conditions. The occurrence of complications is a not infrequent cause of death. Among these is to be mentioned particularly bronchopneumonia. In the fatal cases death is liable to occur in the first 3 to 5 days, during the continuance of the active constitutional symptoms, and probably certainly in the first 2 weeks of the attack. After this period recovery is almost assured so far as life is concerned. The influence of the age of the patient is very decided. Statistics show that the fatality in children is decidedly less than later. Wickman[14] found a mortality from 0 to 2 years of 10 per cent. and from 3 to 5 years of 11.6 per cent.; gradually increasing until from 30 to 32 years it was 33.3 per cent.

The likelihood of recovery from the paralytic condition is a matter of prognostic importance. The early paralysis of the trunk and neck seen in the spinal cases will probably disappear. The more promptly improvement begins, both with regard to paralysis and to tenderness, the better the prognosis. Not infrequently all traces of paralysis everywhere may cease to exist, and very great recovery will often take place even in cases of early, wide-spread, and apparently severe paralysis. Lovett and Richardson[1] found that in 16.7 per cent. of 150 cases analyzed by them all evidences of the disease had disappeared within 3 months. Wickman[2] was able to study 530 instances of primary paralysis from 1 to 1½ years after the attack. Of these 56 per cent. still showed paralysis and 44 per cent. none at all. The paralysis in cases of the bulbar type generally disappears completely. Very commonly, however, in the spinal cases some evidence of paralysis remains throughout life. This may vary anywhere from complete disability to a slight impairment of motion, most frequently seen as a trifling limp in walking. The ultimate degree of recovery depends often upon the faithfulness with which treatment has been carried out. The muscles of the arms and shoulders seem less liable to undergo improvement than those of the legs. Muscles in which entire paralysis and complete loss of faradic contractility are present after the acute stage is over, in the course of the 2d week, are liable to be more or less permanently paralyzed. If slight return of faradic contractility is found in the course of a few days longer, partial recovery will probably follow. If after 2 or 3 months there is much wasting and loss of power and continued loss of faradic contractility, little improvement can be expected. Muscles which have never exhibited electrical changes will probably recover completely in a few weeks or months. Even in cases seen for the first time in the chronic stage, if there is some degree of faradic contractility remaining, considerable improvement under treatment may be expected. Lovett[3] analyzed 180 cases in the 3rd year after the attack, and showed the tendency to continued improvement which was present under muscle-training and avoidance of weight-bearing. The upper extremities will improve during 4 years and the lower during 3 years. After this time there may be slight deterioration, as a result of strain put upon the deformed limbs. He further states[4] that he has not seen more than 2 or 3 cases which could not in some manner be made to walk eventually.

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[1] *Loc. cit.*

[2] El. Siglo Med., 1915, LXII, 530. Ref., Brit. Jour. Child. Dis., 1916, XIII, 56.

[3] Jour. Nerv. and Ment. Dis., 1919, XLIX, 273.

[4] Jour. Amer. Med. Assoc., 1913, LX, 1611.

[5] Deutsch. med. Wochenschr., 1909, XXXV, 1822.

[6] Studien ü. d. Heine-Medinsche Krankheit, 1911, 55.

[7] Deutsch. Zeitschr. für Nervenheilk., 1910, XXXVIII, 362.

[8] Beiträge zur Kenntnis der Heine-Medinschen Krankheit., 1907, 286.

[9] Jahrb. f. Kinderheilk., 1912, LXXVI, 603.

[10] Deutsch. Zeitschr. f. Nervenheilk., 1914–15, LIII, 222

[11] *Loc. cit.*, 78.

[12] Report of New York Collective Investigation Committee, 1910, 24.

[13] Amer. Jour. Dis. Child., 1917, XIV, 69.

[14] Beiträge zur Kenntnis der Heine-Medinschen Krankheit., 1907, 288.

Diagnosis.—It seems impossible to recognize poliomyelitis with certainty in its prodrómal stage. Regan[5] emphasizes the presence of a temporary hydrocephalus in this stage as well as later, as shown by Macewen's sign. Lumbar puncture made just before paralysis appears yields a clear or slightly opalescent fluid under pressure, exhibiting an increase of mononuclear cells and a moderate globulin reaction. A spinal fluid of this nature may be readily distinguished from that of cerebrospinal fever, but not positively from tuberculous meningitis. In the latter the albumin and globulin are generally greater in amount, the pressure greater, the sugar diminished or absent, and the cells more numerous. But to this there are so many exceptions that the differences are not trustworthy. Moreover, the symptoms of poliomyelitis are so very indefinite that it would be necessary to perform lumbar puncture on every child with manifestations of a dubious sort. It is only when an epidemic is prevailing that the beginning of poliomyelitis will be thought of and that lumbar puncture will be tried. The most suggestive of the early manifestations are hyperesthesia, sweating, and nervous irritability. The variation of the complex of early symptoms, however, makes a number of different diagnoses possible at this time. Vomiting and constipation suggest a gastrointestinal disorder; severe diarrhea, an intestinal autointoxication; severe pain in the head and stiffness of the neck and convulsions, a cerebrospinal fever or other similar form of meningitis; somnolence or coma with rigidity, a tuberculous meningitis; headache, coryza, decided prostration and rapid pulse, grippe. *Cerebrospinal fever* has symptoms very similar to cases of poliomyelitis with decided meningitic symptoms. Lumbar puncture, however, shows a cloudy or even purulent fluid, with numerous polymorphonuclear cells. The blood, too, in that disease exhibits uniformly a high leucocytosis. Fever is of longer duration in cerebrospinal fever and there is oftener coma. The reflexes are increased and a spastic condition soon develops.

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[1] *Loc. cit.*, 70.

[2] Die acute Poliomyelitis, 1911, 78.

[3] Jour. Amer. Med. Assoc., 1921, LXXVII, 1941.

[4] Jour. Amer. Med. Assoc., 1922, LXXVIII, 1607.

[5] Amer. Jour. Dis. Child., 1918, XV, 259; XVI, 13.

Tuberculous meningitis exhibits a spinal fluid very like that of poliomyelitis and the diagnosis may be impossible for some days. Then, however, the failure of flaccid paralysis to appear and the increase of the stuporous condition point to the former malady. The onset of meningitis is usually much more insidious, although to this there are exceptions. Jeans and Johnston[1] believe that the reaction in the syphilitic zone, *i.e.*, that of low-dilution, which occurs in the colloidal gold test of the cerebrospinal fluid in poliomyelitis, is of service in the differential diagnosis, since it does not take place in the fluid from tuberculous meningitis. Regan, Litvak and Regan[2] likewise found the low-dilution reaction in poliomyelitis, in contra-distinction to tuberculous meningitis, except in the early stages of the latter. (See also p. 595.) *Acute epidemic encephalitis* may easily be confused with poliomyelitis. The characteristic symptoms of lethargy, ophthalmoplegia and marked asthenia are much more likely to be present and continued in encephalitis than in poliomyelitis. Prompt appearance and spread of paralysis of the extremities is in favor of poliomyelitis. The presence of epidemics helps in the diagnosis. In the spinal fluid little difference is seen, except that evidences of meningeal irritation (increase of pressure, cells and globulin) are more marked in poliomyelitis. Increased sugar-content suggests encephalitis. As a rule, as the course of the case develops the diagnosis between these two diseases becomes clear; but this is not always the case. Some cases of the bulbo-spinal and encephalitic forms of poliomyelitis are never certainly differentiated from instances of epidemic encephalitis in which paralysis of the extremities accompanies that of the cranial nerves.

When once paralysis has developed, the diagnosis of poliomyelitis is usually apparent, although it often happens that this condition is not recognized for some time, the helpless state being supposed to be the result of prostration. Even after the stage of retrogression is over mistakes in diagnosis are still possible. There is scarcely, however, any other form of paralysis which gives the picture of flaccidity, atrophy, and flail-like freedom of movement at the articulations, unless it be *multiple neuritis.* This disease is more frequent than the polyneuritic type of poliomyelitis, the onset is slower, and there are no cerebral symptoms. It is generally much more wide-spread over the body and there is considerably more pain in the limbs; yet this last feature is not a safe diagnostic sign. A diminution of the touch-sense indicates the presence of neuritis rather than poliomyelitis. The likeness of the two diseases is certainly very close, but the anatomical identity is not yet proven. A *localized neuritis,* oftenest of one arm, as in instances of obstetrical paralysis, sometimes causes error in diagnosis. Only the history of the case can distinguish it. *Infantile scurvy* may readily simulate poliomyelitis through the development of pseudo-paralysis, the result of pain on movement. I have seen the mistake in diagnosis made in a number of instances. The associated symptoms, the history of the case, and the prompt reaction to antiscorbutic treatment soon settle the question. The pseudo-paralysis of severe *rickets* has repeatedly been supposed to be the result of poliomyelitis. A little care in observation will remove all doubt. In rickets there is the history of slow onset and the presence of associated symptoms. There exists a weakness rather than a paralysis, and this is wide-spread and not more marked on one side than the other. Electrical reactions also are different. The ordinary *cerebral paralyses* are not easily confounded. They are spastic in character, with exaggerated tendon-reflexes. Only in the uncommon acute encephalitic form of poliomyelitis is there a spastic paralysis present. The history of the attack aids in the diagnosis, as would the early employment of lumbar puncture. Even the contractions about the joints in old cases of poliomyelitis are easily recognized not to be spastic in character, but due to actual shortening of the muscles. In instances of the bulbar form of poliomyelitis, facial paralysis may readily be supposed to be a *Bell's palsy,* due to neuritis of the facial nerve. The history of the attack is, however, entirely different and the frequent combination of some paralysis of the limbs makes the matter clear. The neutralization test has been proposed as a diagnostic method in doubtful cases. In this the blood-serum of the patient is mixed with active virus in amount fatal for a monkey, the mixture incubated and this injected into the cranial cavity of the animal. If the monkey remains well, the virus has been neutralized and the patient has poliomyelitis. The test is not entirely reliable and is not a practical one, because antibodies do not form in the patient until the stage of recovery, by which time the diagnosis has probably become clear.

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[1] Amer. Jour. Dis. Child., 1917, XIII, 239.
[2] Amer. Jour. Dis. Child., 1923, XXV, 76.

Treatment. **Prophylaxis.**—Until there is greater knowledge of the method by which the disease is contracted, little can be expected in the way of preventive treatment. The fact that the virus is found at times on the nasal mucous membrane, and even may be virulent long after the attack is over, indicates the advisability of thorough frequent disinfection of this region in patients suffering with the disorder, in the hope of preventing its dissemination. This brings up, too, the subject of separation and quarantine. In spite of the absence of positive evidence of direct transmission, quarantine should be enforced, especially during an epidemic prevalence of the disease, at which time the virulence of the germ appears to be greatly increased. With a lack of knowledge of the usual period of infectiousness, the proper duration of isolation is a matter of uncertainty. A quarantine of 4 weeks would seem ample, unless inflammation of the nose and throat is present. How much longer would be necessary to ensure absolute safety cannot, as yet, be determined. A child who has been exposed may be considered safe if not developing the disease in 2 weeks. Those who have come into contact with patients should employ disinfection of the nose and throat to prevent the possibility of their acting as carriers. The value of wearing a mask of gauze or similar substance over the mouth and nose is questionable (see p. 346).

The discharges from the mouth and nose of the patient should be promptly destroyed. After the room is vacated it should be thoroughly disinfected. It is also advisable not to take children to localities where the disease is epidemic; or, if already there, to forbid their association with those who have recently had the affection, or with those who are ill with any vague symptoms, as well as to prevent their frequenting public conveyances or public places of amusement where many children are together.

The ability to immunize human beings in time of epidemics would be most desirable, and may be finally accomplished. It has sometimes been done successfully in monkeys by Flexner and Lewis,[1] Flexner and Amoss[2] and others, but as yet it is too uncertain for application to human medicine.

Treatment of the Attack.—During the initial stage of the attack there is as yet no treatment of certain avail open to us. It is questionable whether lumbar puncture is a helpful therapeutic procedure, unless there is marked increase in pressure or definite meningeal symptoms. In monkeys it was found by Flexner and Amoss[3] that it opens the way for the passage of virus from the blood into the central nervous tissues, unless the puncture was immediately followed by the injection of immune serum. In the case of monkeys it was shown by Flexner and Clark,[4] that the administration of hexamethylenamine delayed the development of symptoms. Nothing, however, of practical value has resulted from the latter experiments, but since at least no harm is likely to follow its administration, it may be given in fairly large, divided doses, of 20 to 40 grains (1.3 to 2.6) daily; but to be of any possible benefit it must be used before paralysis develops.

Based upon animal experimentation in other conditions, Meltzer[5] recommended the administration of adrenaline, giving 0.5 c.c. (8 m.) of the 1:1000 solution intraspinally. This had been previously tried by Clark[6] in monkeys, with considerable success. Netter[7] in 1910 used the injection of blood-serum from subjects who had previously suffered from poliomyelitis, and since then has employed it in 32 cases with, he believes, excellent results. In the 1916 epidemic this method of treatment was tried in a considerable number of instances, apparently with good effect. Zingher[8] used it in doses of 10 to 15 c.c. (0.34 to 0.51 fl.oz.) intraspinally every 20 to 24 hours for 2 or 3 days. He reported its employment in 418 cases and claimed a beneficial action when given in the preparalytic stage. Wells[9] advises the intravenous or intramuscular injections of 50 to 100 c.c. (1.69 to 3.38 fl.oz.) of convalescent serum daily. After the injection a spinal puncture is done and 10 to 15 c.c. (0.34 to 0.51 fl.oz.) of fluid withdrawn in order to reduce the pressure and favor circulation in the central nervous system. Amoss and Chesney[10] had good results by combining intraspinal with intravenous or intramuscular injections of convalescent serum. Aycock and Amoss[11] suggest the combination of intravenous injections of hypertonic salt solution and intraspinous ones of immune serum. Normal human serum is also used and beneficial results claimed. It is said that preservatives increase the value of serum by enhancing cellular reaction. In the preparalytic stage in experimental poliomyelitis in monkeys treatment with immune serum appears to be of undoubted value (Flexner and Amoss).[1] Rosenow[2] reports good effects from intravenous injection of an immune horse-serum, which is prepared by repeated injections of the streptococcus previously described (p. 591). Nuzum and Willy[3] have seen benefit result from the use of a serum like Rosenow's, prepared from immune sheep's and rabbit's blood as well as from the horse. Many clinicians share the opinion that treatment by serum is of value in the preparalytic stage, while others are entirely unconvinced. Peabody,[4] reporting the conclusions of the Harvard Infantile Paralysis Commission, could find no demonstrated value of any treatment in the preparalytic stage, and states that about as many cases escaped paralysis in the untreated as in the treated cases; and Lovett[5] in a study based on 5100 cases could not discover that either convalescent serum or the special streptococcic serum had established itself as serviceable. The great difficulty in determining the value of any serum-treatment rests upon the general admission that, to be of any service, it must be given in the preparalytic stage, upon the difficulty in controlling a supply of convalescent serum, and upon the impossibility at this period of determining that the disease is undoubtedly poliomyelitis, or that, if it is this, it may not be an abortive case which would have recovered in any event.

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[1] Jour. Amer. Med. Assoc., 1910, LIV, 1780.
[2] Jour. Exper. Med., 1924, XXXIX, 625.
[3] Jour. Exper. Med. 1917, XXV, 525.
[4] *Loc. cit.*, 585.
[5] Med. Rec., 1916, XC, 171.
[6] Jour. Amer. Med. Assoc., 1912, LIX, 367.
[7] Arch. de méd. des enf., 1916, XIX, 1.
[8] Arch. of Pediat., 1916, XXXIII, 872.
[9] Jour. Amer. Med. Assoc., 1916, LXVII, 1211.
[10] Jour. Exper. Med., 1917, XXV, 581.
[11] Jour. Amer. Med. Assoc., 1923, LXXXI, 474.

Complete rest in bed is imperative, but counterirritation with blisters or similar measures merely adds to the discomfort, and there is no evidence that it can be of avail at any stage of the disease. Remedies may be needed to relieve pain, restlessness, or other nervous symptoms; to control fever; and to support strength. It is not yet time for electricity and massage, which can do no good and may do harm. All such treatment should be delayed until the stationary stage is over and all tenderness has disappeared. This may not be for from 3 to 6 weeks or longer after the onset. The value of electricity and massage is to improve by this enforced exercise the tone and the power of the muscles which have been only partially or temporarily injured. Those muscle-fibres presided over by absolutely atrophic ganglion-cells can never show any improvement. The faradic current should be applied to the different muscles which have been paralyzed, continuing the entire treatment for from 10 to 20 minutes, once or twice a day. If there is no response to faradism, galvanism may be tried. Only such strength of current of either sort should be used as will produce moderate contractions without pain. Massage and active and passive movements can be commenced as soon as the acute and stationary stages are over and all hyperæsthesia and pain have disappeared. It is very important to combine with the passive exercise the encouraging of the patient to make voluntary active movements, no matter how imperfectly these are performed at first. More can be expected from this than from any other plan of treatment, and if persevered with for months or even for years remarkable improvement will often follow in cases which at first appeared most dis-

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[1] Jour. Exper. Med., 1917, XXV, 499.

[2] Jour. Amer. Med. Assoc., 1921, LXXVII, 588.

[3] Jour. Amer. Med. Assoc., 1917, LXIX, 1247.

[4] Bost. Med. and Surg. Jour., 1921, CLXXXV, 174.

[5] Jour. Amer. Med. Assoc., 1922, LXXVIII, 1607.

couraging. Confinement to bed should continue for 2 or 3 months except in the mild cases, when 3 or 4 weeks may be sufficient. As a result of the study of a large number of cases—5100—Lovett[1] emphasizes the risk attending the permitting of any weight-bearing exercise for a long period after the attack, and shows that there is danger of increasing the degree of residual paralysis already present. Very little walking should be permitted during the 1st year even with braces. Exercises under water are employed with success. Phillips and Garland[2] believe that the *x*-ray applied over spinal segments stimulates diseased but viable anterior horn cells. In residual paralyses they have seen improvement by this treatment when other methods failed. It is serviceable, too, for the relief of pain in the hyperaesthetic cases. Borden,[3] also, maintains its value for the restoration of the function of the ganglion cells.

Mechanical Treatment.—It is important to prevent deformity by the employment as early as necessary of mechanical apparatus. Even during the early part of the stage of retrogression one must guard against beginning deformity. Sand-bags at the soles of the feet, the wearing of a shoe with elastic extending from the toe to the hip or the application of a light splint will aid in preventing the contraction of the tendo Achillis, which is so likely to occur. A proper position in bed is necessary, too, in many cases to avoid the production of lateral curvature. As soon as the patient is allowed to sit up in bed an abdominal corset may be needed. While the use of braces is to be deprecated on the ground that it adds to weight to be moved and removes the necessity for the paralyzed muscle to do its work completely; yet, on the other hand, it is still worse to allow deformities of any part to develop. The need for their employment must be determined for the individual case. Sole dependence upon crutches is harmful, as it permits the muscles to remain in an entirely unused condition. Yet, as already stated, allowing the child to bear weight upon the partially paralyzed and unsupported legs is one of the surest ways of increasing deformity and diminishing muscular power by over-strain. Tenotomies and other operations will be required less frequently if measures are early undertaken to prevent contractions forming. In some cases tendon-transplantation may be performed with benefit. This, as a rule, is best deferred for at least 2 years. In other cases artificial anchylosis may give necessary rigidity to an otherwise useless joint. These matters are discussed in treatises upon orthopedic surgery. The great principle of treatment in the chronic paralytic stage is never to abandon it, since slow improvement may finally come, and aid may be given in some way in cases at first most unpromising.

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[1] Jour. Amer. Med. Assoc., 1917, LXIX, 168; 1921, LXXVII, 1941; 1922, LXXVIII, 1607.

[2] Jour. Amer. Med. Assoc., 1924, LXXXII, 1847.

[3] Arch. Radiol. and Electrother., 1921, XXVI, 215.

CHAPTER XX

EPIDEMIC ENCEPHALITIS

Terminology.—Among the confusing number of terms used to describe this condition may be mentioned Lethargic encephalitis, Epidemic stupor, Epidemic somnolence, Sweating sickness, Acute infectious ophthalmoplegia, Sleeping sickness, Acute encephalitis, Focal hemorrhagic encephalitis, Post-influenzal encephalitis, Myoclonic encephalitis, and Nona. To all of these there are objections. Some of them emphasize symptoms and relationships which are not always present, and Sleeping sickness is, in addition, to be avoided on account of a possible confusion with the African disease caused by the trypanosoma. Nona is a term used a number of years ago in Italy, Bavaria and Russia to describe epidemics which resembled, and probably were identical with, the epidemic encephalitis of today; but the name was apparently also employed to denote somnolence due to any cause. The term Epidemic encephalitis seems preferable, because the disease usually occurs in epidemic form and its pathological basis is an inflammation of the brain-substance.

History.—In spite of the fact that epidemic encephalitis was first recognized and probably first appeared in America in the year 1918 (Tilney and Riley;[1] Bassoe[2]), there is no reason to call it a new disease. The attention of the medical profession was first aroused by the writings of von Economo[3] describing the epidemic in Austria which began in 1916. The malady spread from that country to France, then to England, and later to America and elsewhere; but if the difference in terminology is considered, and the symptoms of earlier reported cases studied, it is evident that epidemics of this disorder have visited Europe for some hundreds of years. The literature has become so voluminous that it is impossible in what follows to do more than give credit to a few of the many excellent publications. The history of encephalitis may well be consulted in Crookshank's[4] contribution. The articles of Barker, Cross and Irwin[5] and of Happ and Mason[6] contain comprehensive bibliographies.

Relation to Other Diseases.—It is but natural that *poliomyelitis* and encephalitis should be confused, and that argument should arise concerning the identity of the two diseases. In both the seat of the pathological change is in the central nervous system. In both epidemic influence and infectious origin are definite, and there are also some symptoms common to both. The consensus of opinion, however, is that the weight of evidence in history, pathology, symptoms and experiment indicates that each is a distinct entity. In the history of epidemics it can be seen that the two do not occur together; undoubted cases of poliomyelitis are common in the summer and fall, while epidemic encephalitis is a disease of winter. There are differences in pathology and also in symptomatology which will be discussed later. On the other hand, it has been argued that these differences are only those dependent upon the situation attacked by the virus (Burrows;[1] Maggiore and Sindoni[2]) and there are some who maintain that no proof has as yet been offered which clearly separates the diseases in a clinical, epidemiological or pathological way (Crookshank).[3] Although it has been shown experimentally by Amoss[4] that the blood-serum of recently convalescent cases of epidemic encephalitis was devoid of the power of neutralizing the virus of poliomyelitis, some experiments of Neustaedter, Larkin and Banzhaf[5] appear to prove that this protective power does exist, thus indicating some relationship between the two disorders.

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[1] Neurol. Bull., 1919, II, 106.
[2] Jour. Amer. Med. Assoc., 1919, LXXII, 971.
[3] Wien. klin. Wochenschr., 1917, XXX, 581.
[4] Bost. Med. and Surg. Jour., 1920, CLXXXII, 34.
[5] Amer. Jour. Med. Sci., 1920, CLIX, 348.
[6] Johns Hopk. Hosp. Bull., 1921, XXXII, 137

It has been claimed that there is a close relationship between *influenza* and encephalitis, some authors maintaining that the virus causing the two is the same; others only that influenza prepares the ground for encephalitis or, in other words, activates the encephalitic virus. Apparently there is no experimental work which establishes any such connection. The idea arose from the fact that in some instances epidemics of encephalitis have followed epidemics of influenza; or that in sporadic cases symptoms of the former have been preceded by those suggesting the latter. Yet in neither epidemic nor sporadic cases of encephalitis can the occasional association be viewed as more than coincidental. From some reports it seems very probable that meningitis following influenza has been supposed to be encephalitis.

Very interesting is the fact that a virus capable of causing *herpes* in man and a herpetic keratitis in animals will produce, when inoculated into such animals as rabbits, symptoms identical with encephalitis, and that, furthermore, material from the brains of these animals will in turn produce herpetic keratitis in other animals inoculated (Edel;[6] Doerr and Berger;[7] Lauda;[8] Flexner[9]). This matter will be alluded to again when considering the etiology.

Because of histological examinations and animal inoculations, some writers claim that certain cases of acute febrile *chorea* are instances of epidemic encephalitis (Harvier and Levaditi).[10] Dubini's electric chorea has been regarded as identical with it (Cruchet;[11] Litvak[12]). This confusion results from the similarity in symptoms between the choreoid and myoclonic forms of encephalitis and the diseases in question. Certainly there can be no reason to regard classical Sydenham's chorea as epidemic encephalitis. Wilson and Winkelman[13] have found that there are no pathological processes in the cerebrospinal system in chorea resembling those of encephalitis. Other diseases have been thought to possess a near relationship, among them being hiccup appearing in epidemic form, and a disorder entitled Australian X. The latter bears a closer resemblance to poliomyelitis than it does to encephalitis (Flexner).[1]

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[1] Arch. Intern. Med., 1920, XXVI, 477.

[2] La Pediat., 1921, XXIX, 682.

[3] Jour. Amer. Med. Assoc., 1923, LXXX, 1473.

[4] Acute Epidemic Encephalitis. Trans. Assoc. for Research in Nervous and Mental Dis., 1921, 225.

[5] Amer. Jour. Med. Sci., 1922, CLXIII, 715.

[6] Nederlandsch, Tijdschr. v. Geneesk., 1922, II, 263.

[7] Schweiz med. Wochenschr., 1922, LII, 863.

[8] Centralbl. f. Bacteriol., 1924, XCI, 159.

[9] Jour. Amer. Med. Assoc., 1923, LXXXI, 1688.

[10] Bull. et mém. soc. méd. d. hôp. de Paris, 1920, XLIV, 583

[11] Med. Rec., 1920, XCVIII, 225.

[12] La riforma med., 1920, XXXVI, 322.

[13] Arch. Neurol. and Psychiat., 1923, IX, 170.

Etiology. Predisposing Causes.—The disease may occur at any *age.* At least 2 instances have been reported in new-born infants whose mothers were suffering from the malady during pregnancy (Mercier;[2] Klippel and Baruk[3]). That is it common in infancy and childhood is attested by such reports as those of Heiman,[4] Comby,[5] Moore,[6] Neal,[7] Hofstadt[8] Duzár and Baló[9] and many others. In some epidemics at least 40 per cent. of the patients have been children under 10 years of age (Neal)[10] and about 50 per cent. under 15 years (Smith).[11] We believe that encephalitis is more common in infancy and childhood than some statistics would indicate. Many at this time of life probably have it in a mild and unrecognized form; and in others it is only after sequels develop that the true nature of the original illness becomes manifest. *Sex* seems to have little if any influence in childhood. This is in contradistinction to adults, in whom males are more often attacked. *Season* plays a definite part, in that, while cases develop throughout the year, they are much more numerous in the winter and spring months. (Fig. 173). The strong *epidemic influence* has been alluded to. Since encephalitis first appeared in this country in the winter of 1918–19, up to and including the year 1923, cases have appeared in sufficient numbers to constitute an epidemic, although usually of mild form. Sporadic cases are uncommon. There seems to be an almost universal *geographical distribution,* cases being reported from every civilized country. Many

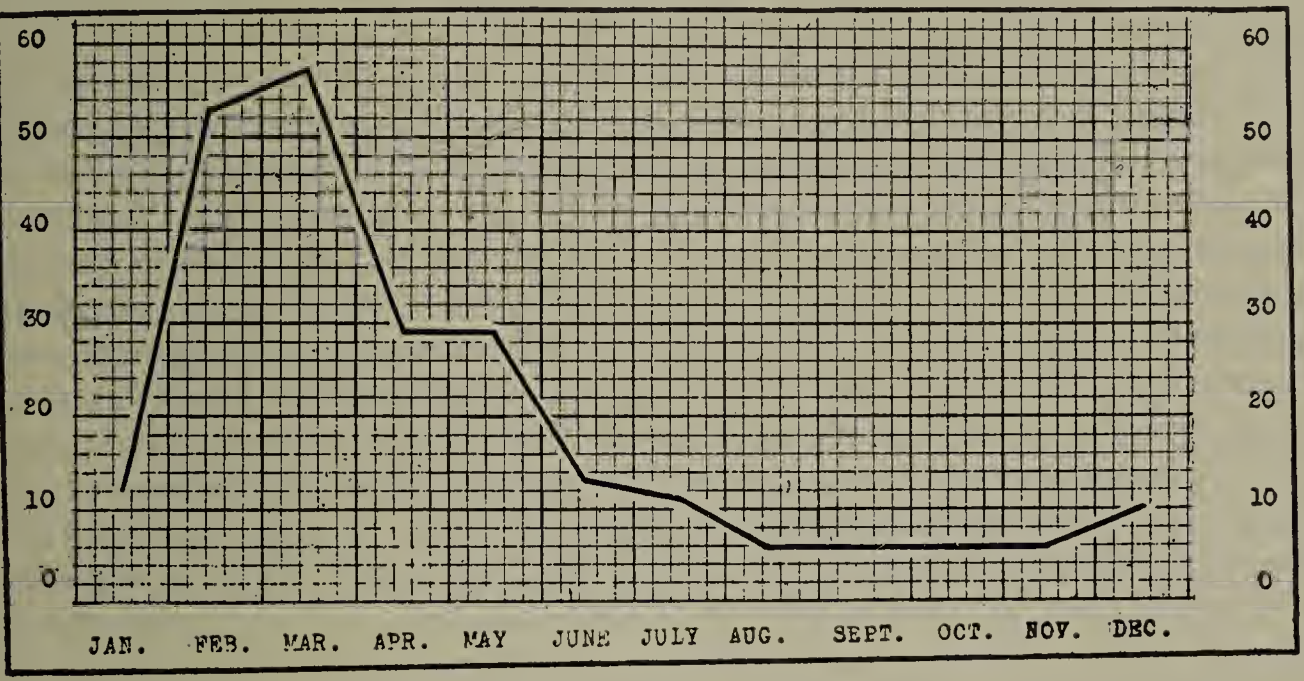

FIG. 173.—EPIDEMIC ENCEPHALITIS.

Monthly mortality in New York City during the epidemic of 1920. Indicates the greater prevalence of the disease in the winter-months. The figures denote the number of deaths. (*Wynne, in Acute Epidemic Encephalitis. Proceedings of Association for Research in Nervous and Mental Diseases,* 1921; 4.)

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[1] *Loc. cit.*
[2] Jour. Amer. Med. Assoc., 1921, LXXVII, 135.
[3] Rev. neurol., 1923, XXXIX, 381.
[4] Amer. Jour. Dis. Child., 1919, XVIII, 83.
[5] Bull. et mém. soc. méd. d. hôp., de Paris, 1920, XLIV, 161.
[6] Northwest Med., 1921, XX, 176.
[7] Jour. Amer. Med. Assoc., 1921, LXXVII, 121.
[8] Zeitschr. f. Kinderheilk., 1921, XXIX, 190.
[9] Jahrb. f. Kinderheilk., 1922, XCIX, 209.
[10] *Loc. cit.*
[11] U. S. Pub. Health Rep., 1921, XXXVI, 207.

individuals must be immune to encephalitis since its *communicability* is very slight. Some authors have suggested that a temporary or permanent substandard state of the nervous system is necessary for the action of the virus (Lépine;[1] Villinger[2]). Whether or not this is true, it is certain that the majority of those exposed do not contract it. In the report of Smith[3] it was noted that 153 patients exposed 900 individuals in their families, none of whom developed the disease. Neither Hunt[4] nor Happ and Mason[5] could find instances of home or hospital contagion. In over 100 cases Neal[6] observed only 1 instance, and Ebaugh[7] another, of 2 cases in the same family. Roger[8] records 174 instances of contagion in 10,000 cases. A few additional reports illustrate the possibility of direct infection, such as those of Claude and Laulerie,[9] Roger and Blanchard,[10] Hofstadt[11] and others; but with many such mere coincidence would explain the occurrence. Kling,[12] who had the unique opportunity of observing an epidemic in a thinly populated section of Lapland, found that direct personal contact-transmission occurs, and believes that he has also demonstrated the existence of carriers. It would appear, too, from experimental work that the virus may be disseminated through the naso-pharyngeal secretion. It may well be that abortive and unrecognized cases, with general symptoms only and without evidence of lesions of the central nervous system, occur more frequently than has been supposed and are sources of infection to others. This would make the infrequency of direct transmission more apparent than real.

Exciting Cause.—Loewe, Hirshfeld and Strauss[13] have made important studies on the active cause of encephalitis. Their work has been confirmed by Levaditi and Harvier,[14] McIntosh and Turnbull,[15] Thalhimer[16] and others. The results of the investigations may be summarized as follows:—Berkefeld filtrates of brain-material, nasopharyngeal mucous membrane and nasal washings, spinal fluid and blood from cases of epidemic encephalitis have produced in rabbits and monkeys lesions typical of this disease. By means of special cultural methods a minute, filterable organism was discovered in these tissues and secretions, and this same organism later recovered from the brain and nasopharyngeal mucous membrane of animals that had been inoculated with it. The cultures of this recovered organism have again produced the disease when injected into other animals, and the organism has again been isolated. A certain amount of doubt attaches to some of the animal investigations because rabbits and monkeys in captivity are subject to epizootic encephalitis (Lucke;[17] Flexner[18]) and according to Levaditi, Nicolau and Schoen[19]

[1] Bull. acad. de méd. de Paris, 1920, LXXXIV, 225.
[2] Münch. med. Wochenschr., 1921, LXVIII, 913.
[3] *Loc. cit.*
[4] Jour. Amer. Med. Assoc., 1923, LXXXI, 1352.
[5] *Loc. cit.*
[6] *Loc. cit.*
[7] Amer. Jour. Dis. Child., 1924, XXVII, 230.
[8] La médecine, Paris, 1920, II, 194.
[9] Bull. et mém. soc. méd. d. hôp. de Paris, 1921, XLV, 36.
[10] *Ibid.*, 40.
[11] *Loc. cit.*
[12] Norsk. Mag. f. Laegevidensk., 1922, LXXXIII, 843.
[13] Jour. Amer. Med. Assoc., 1919, LXXIII, 1056.
[14] Ann. de. l'inst. Pasteur, 1920, XXXIV, 911.
[15] Brit. Jour. Exp. Path., 1920, I, 89.
[16] Arch. Neurol and Psych., 1922, VIII, 286.
[17] Arch. Neurol. and Psych., 1923, X, 212.
[18] Jour. Amer. Med. Assoc., 1923, LXXXI, 1785.
[19] Ann. de l'inst. Pasteur, 1924, XXXVIII, 651.

the inoculation of the material of epidemic encephalitis into one of these animals simply so reduces local resistance that the virus of the spontaneous epizootic type of the disease can act.[1] Von Wiesner,[2] House,[3] Morse and Crump[4] and Rosenow and Jackson[5] have reported microorganisms other than the type just described. The relation between herpetiform viruses and that of encephalitis has already been referred to (p. 612). A possible explanation for this is that these strains are found only by chance in association with material employed for the experimental transmission of encephalitis[6] (Parker).[7]

Pathological Anatomy.—The pathological changes in the central nervous system have been described by a number of investigators, among them Buzzard and Greenfield,[8] Calhoun,[9] Tilney and Howe,[10] Ayer,[11] and Boyd.[12] Usually there is little gross or microscopical evidence of pathological change in the meninges, although there may be edema and congestion of the pia-arachnoid, and in rare instances thrombosis of the meningeal vessels. The brain itself may show gross evidence of edema or congestion, or may appear normal until microscopic studies are made. The essential lesions found are (*a*) congestion of vessels and hemorrhagic areas which may be visible grossly, but usually are minute and microscopic; (*b*) marked cellular infiltration, consisting of small and large mononuclear and plasma cells which are present in the perivascular and the Virchow-Robin spaces; (*c*) degenerative alterations such as cloudy swelling, neuronophagia and satellitosis in the nerve-cells, and (*d*) proliferation of the neuroglial cells. While changes may be discovered throughout the brain and even the white matter be involved, it is the gray matter of the mid-brain, the basal ganglia, the pons and the medulla which suffer most frequently and most intensely. The spinal cord, the nerve-roots and the dorsal root-ganglia may be the seat of perivascular lymphocytic infiltration. Numerous spherical homogeneous hyaline bodies are found in the white and gray matter in some cases. Cloudy swelling, congestion, and petechial hemorrhages may be observed in other organs, notably the kidney and the serous lining of the pericardium, pleura and peritoneum.

This description applies to acute cases. In patients dying some time after the active process has ceased there are no characteristic findings that serve to distinguish epidemic encephalitis from any other form (Gilbert and Gurd).[13]

Symptoms.—As might be expected from the pathological anatomy the symptomatology is extremely diversified. While in epidemics there is a tendency for a particular type to be prevalent, even then other forms may appear. There are several ways in which the disease may be classified. It may be divided, as is done by Tilney and Howe,[14] into meningitic, cerebral, cerebellar, cranial-nerve, spinal, and peripheral types. The chief symptoms may serve to differentiate types into the lethargic, myoclonic, cataleptic, Parkinsonian, polyneuritic, psychotic, choreiform and other varieties. There are other elaborate classifications proposed. Because of the multiplicity of the clinical types and their overlapping, the disease will be discussed in this chapter from the point of view of symptoms, after the method followed in the valuable paper of Happ and Mason.[1]

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[1] See thorough review in Medical Science, 1924, X, 355.

[2] Wien. klin. Wochenschr., 1917, XXX, 933.

[3] Jour. Amer. Med. Assoc., 1920, LXXIV, 884.

[4] Jour. Lab. and Clin. Med., 1920, V, 275.

[5] Jour. Infect. Dis., 1923, XXXII, 144.

[6] Medical Science, *loc. cit.*

[7] Jour. Med. Research., 1924, XLIV, 289.

[8] Brain, 1919, XLII, 305.

[9] Arch. Neurol. and Psych., 1920, III, 1.

[10] Epidemic Encephalitis, 1920.

[11] Acute Epidemic Encephalitis, Trans. Assoc. for Research in Nerv. and Ment. Dis., 1921, 159.

[12] *Ibid.*, 189.

[13] Amer. Jour. Med. Sci., 1924, CLXVII, 781.

[14] *Loc. cit.*

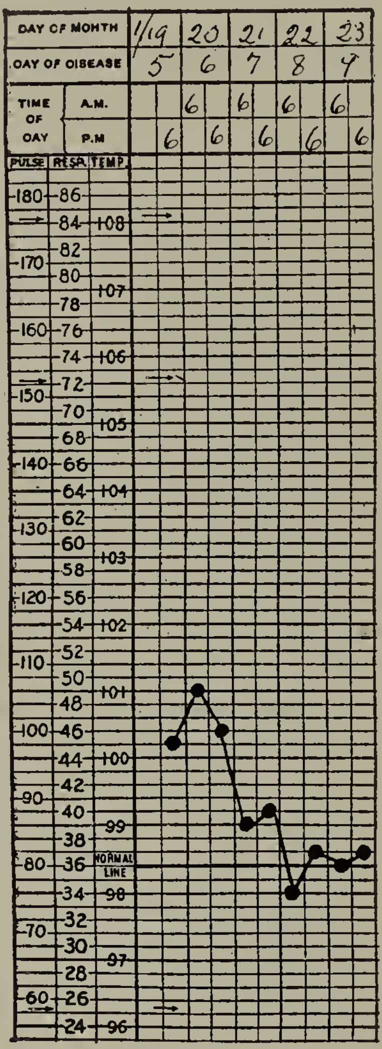

Fig. 174.

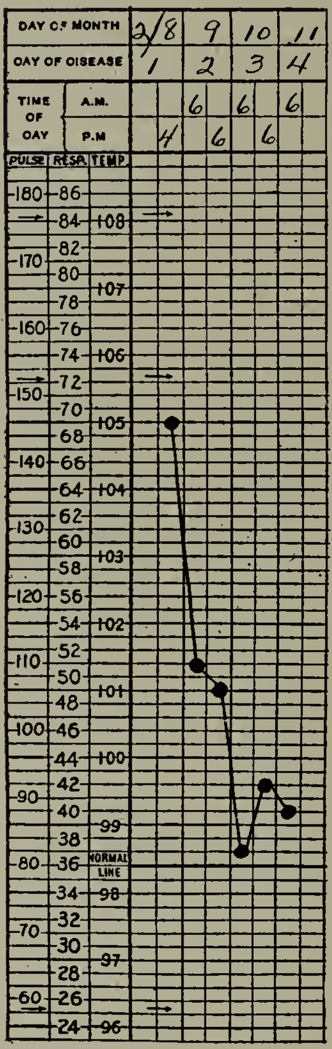

Fig. 175.

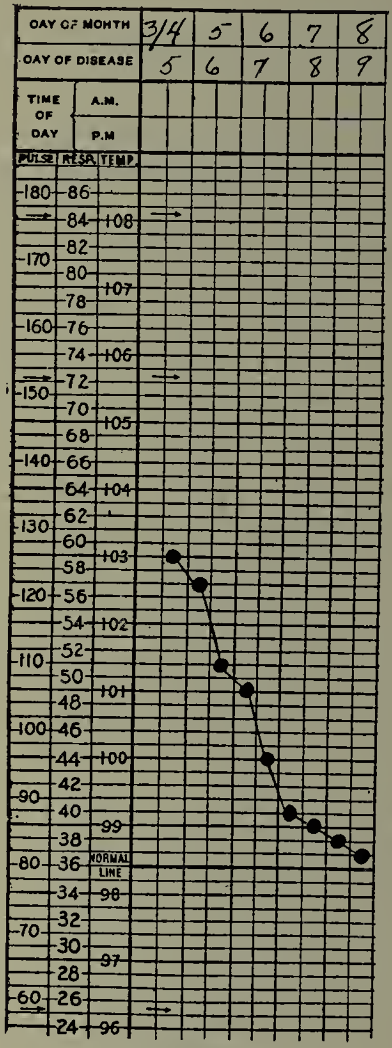

Fig. 176

Fig. 174.—Encephalitis with Short Febrile Course but Persistence of Sequels.

Albert C. Aged 7 years. Admitted to the Cincinnati General Hospital, Pediatric Ward, 5 days after onset. Exhibited slight rigidity of neck; lethargy; left-sided facial paralysis, including tongue; slurring of speech; ankle-clonus; no ocular paralysis. Spinal fluid negative. Four weeks later still ankle-clonus; patellar reflexes normal; no tremor; slurring of speech still present; mentality normal.

Fig. 175.—Epidemic Encephalitis; High Fever; Short Course.

Nick R. Aged 10 years. Admitted to Cincinnati General Hospital, Pediatric Ward, Feb. 8, 7 hours after onset. Exhibited convulsions; stupor; rigidity. Spinal fluid negative except for increased pressure. Four days later still lethargic; patellar reflexes sluggish. Recovery.

Fig. 176.—Epidemic Encephalitis; Short Febrile Course; Involvement of Cranial Nerves.

Jane F. aged 7 months. Taken ill Feb. 29th with great somnolence; arousing slightly for nourishment.—*March* 2. Somnolence continued; paralysis left external rectus.—*March* 3. Paralysis right side of face; ptosis of right eyelid.—*March* 4. Symptoms unchanged; spinal fluid clear; pressure slightly increased; 90 leucocytes to the cmm.; trace of globulin.—*March* 7. Drowsiness less; no facial paralysis discoverable.—*March* 11. Apparently normal, except for a little paralysis of right external rectus.

Incubation.—The duration of the period of incubation is doubtful. According to Kling[2] it is 10 days. Roger[3] states that it is 1 to 2 months but may be several months. In experimental animals it is as short as 5 or 6 days.

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[1] *Loc. cit.* [2] *Loc. cit.* [3] *Loc. cit.*

Stage of Invasion.—There may be sudden onset with the appearance of the characteristic triad of symptoms; lethargy, asthenia and ocular nerve paralyses; or these symptoms may be preceded for several days by such evidences of general disturbance as fever, malaise, anorexia, vomiting, constipation, headache, body-pains and irritability. It is noticeable that the asthenia is much more marked than the fever and other symptoms warrant. In some cases there is an interval of several weeks between the onset of general symptoms and the manifestations denoting central nervous lesions. In cases with slow onset there occur defective memory, lassitude and drowsiness. The temperature is usually moderately elevated and may continue so in an irregular manner throughout the acute course of the disease. The accompanying charts (Figs. 174–177) illustrate the uncharacteristic nature of the fever and the lack of accord with the other symptoms present. In fatal cases hyperpyrexia may occur before death. In some instances there is little or no febrile reaction at any time. Many observers have reported initial symptoms suggesting infection of the upper respiratory tract. This

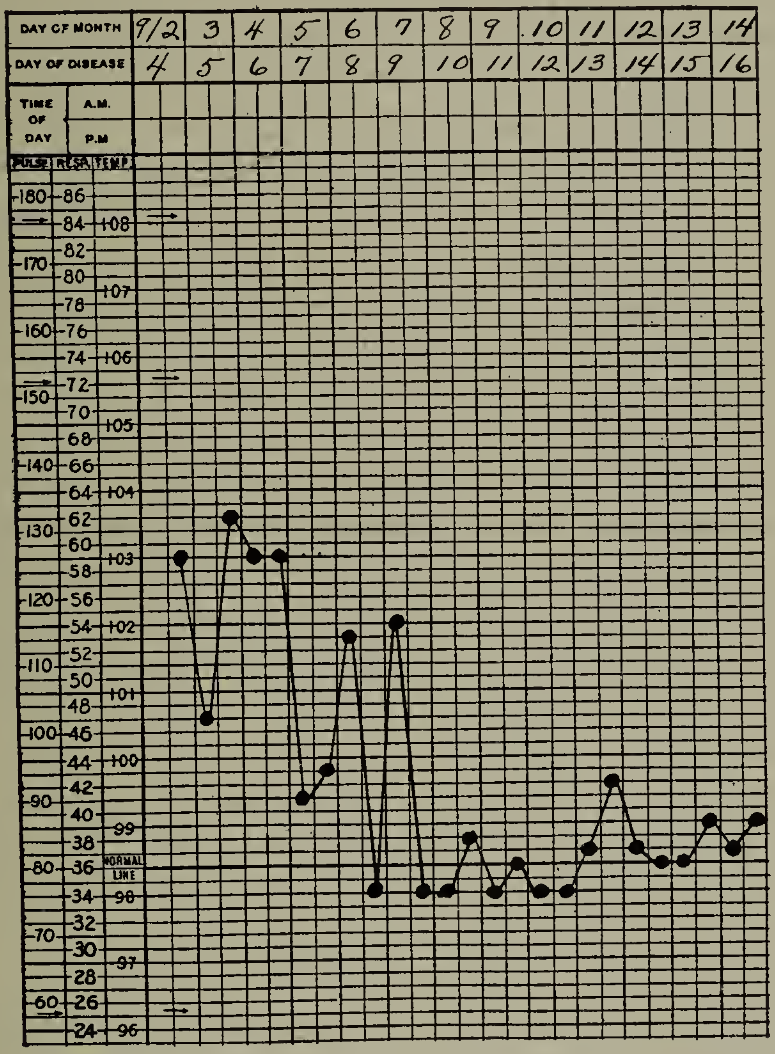

FIG. 177.—EPIDEMIC ENCEPHALITIS. MODERATELY MILD COURSE PSYCHIC DISTURBANCES.

Florence Q. Aged 4 years. Taken ill Aug. 30th with fever and headache. Later convulsions and coma. Admitted to the Children's Hospital of Philadelphia *Sept.* 2. Under observation exhibited unconsciousness; at times restlessness; twitching and rigidity of muscles of neck; bilateral Kernig's and Babinski's signs; twitching of muscles of body; eye-grounds normal; slight lateral nystagmus; internal strabismus of right eye. Leucocytes 15,900. Spinal fluid under increased pressure and showed 40 and later 120 leucocytes per cmm., chiefly lymphocytes; globulin present.—*Sept.* 10. Spoke today; somnolence has disappeared; great irritability; still slight rigidity of neck and paralysis of 6th nerve.—*Sept.* 16. Happy and mentally bright, but moral sense altered; uses foul language and at times seems silly. Residual paralysis of 4th and 6th nerves on right side.

has been one of the arguments that encephalitis results from influenza. It has also led to the interpretation that the virus is localized in the nasopharyngeal passages and from that point is diffused throughout the body, (Abrahamson)[1] a hypothesis which is not supported by the experience of others, since in most cases respiratory symptoms are not present or are so slight as to escape notice.

Disturbances of Consciousness and Mental Symptoms.—Disturbance of consciousness, which may be so mild as simply to constitute apathy or so severe that the patient is lethargic or comatose, is a most characteristic symptom. Like other symptoms it varies with epidemics, in some of which it is present in all cases and in others in from 30 to 80 per cent. Usually the patient can be aroused to answer questions or obey commands, but immediately relapses into the somnolent state. In some instances an exactly opposite condition of marked insomnia is found, either as a preliminary state to, or instead of, lethargy. Even at an early stage in the disease there may be a reversal of the sleep-habit, producing diurnal somnolence and nocturnal wakefulness; but this type of disturbance is usually a later phenomenon. Speech is apt to be slow and monotonous, and answers given at a distinct interval after questions. A mask-like facies may accompany the lethargic state, the effect often being heightened by ptosis of the lids and facial paralysis. Any of these symptoms may be present for a few days only, but usually persist for weeks or even months. A confused mental condition, in which the patient is delirious or irrational or excessively talkative, is sometimes seen instead of, or in addition to, somnolence. Disturbances of emotion with periods of crying or laughing, delusions and maniacal states are not infrequently observed during the acute stage.

Ocular Disturbances.—These constitute the second of the triad of characteristic symptoms, the cranial nerves involved usually being the 3rd and 6th. Transient diplopia is very common (Smith,[2] 83 per cent.; Holden,[3] 56 per cent.; Dunn and Heagey,[4] 47 per cent.), and may occur as an initial symptom. Ptosis of the upper eyelids is also frequently noted and is apt to be continued. In some cases it is slight and seems to be due to hypotonia of the muscles rather than to paralysis of the cranial nerves, the difficulty in distinguishing between the two causes perhaps accounting for the difference in incidence given by different observers (Smith, 95 per cent.; Dunn and Heagey, 18 per cent.). The 4th nerve may also be involved and assist in causing diplopia by paralysis of the superior oblique muscle of the eyeball. The 2nd nerve is affected in some cases (Holden, 4–5 per cent.), and there may be optic neuritis or rarely choke-disc (Spiller).[5] Nystagmus has been observed in some epidemics with considerable frequency (Holden, 32 per cent.), and paralysis of accommodation occasionally.

Disturbances of Other Cranial Nerves.—Involvement of the 5th nerve may occur (Dunn and Heagey, 7.8 per cent.), causing pain in the head and face; that of the 7th nerve is common (Holden, 73 per cent.; Dunn and Heagey, 13 per cent.), producing facial paralysis, and that of the 8th (tinnitus and deafness) unusual (Friesner).[1] Rarely there are symptoms which can be ascribed to affection of the 9th nerve (alteration in taste and dysphagia), the 10th (tachycardia), or the 11th (paralysis of the sterno-mastoid and trapezius muscles). Involvement of the 12th nerve (paralysis of the tongue) seems to be fairly common (Dunn and Heagey, 11 per cent.).

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[1] Acute Epidemic Encephalitis, Trans. Assoc. for Research in Nerv. and Ment. Dis., 1921, 23.

[2] *Loc. cit.*

[3] Acute Epidemic Encephalitis, Trans. Assoc. for Research in Nerv. and Ment. Dis., 1921, 59.

[4] Amer. Jour. Med. Sci., 1920, CLX, 568.

[5] Jour. Amer. Med. Assoc., 1923, LXXX, 1843.

Motor Disturbances.—Some type of motor disorder is frequent (10 to 30 per cent.). While this may occur alone, it is more usual to find associated symptoms such as lethargy and ocular disturbance;—the latter being, of course, essentially motor in origin. Localized paralyses of the tongue, palate, larynx and pharynx have been noted in connection with affections of the cranial nerves. In some epidemics as many as 10 or 15 per cent. of the cases have shown other definite paralyses, such as monoplegias, paraplegias or hemiplegias. A generalized spasticity of the muscles may be present, but this is not so common as hypotonia. Catatonia and catalepsy have been mentioned in a number of reports. The literature on this subject has been reviewed by Musser and Bennett.[2]

Of distinct diagnostic importance are the frequently-seen *hyperkinetic phenomena.* These consist of quick, jerking movements which are involuntary and non-rhythmical in character. They may be only fibrillary, or they may involve an entire muscle or group of muscles; a favorite situation being around the mouth or eyelids. Sometimes the twitchings are wide-spread. Sicard[3] called attention to rhythmic contrations (myoclonia) which are of frequent occurrence. These are usually bilateral and may be either generalized or limited to the extremities. Reilly[4] described a rhythmic convulsive twitching of the muscles of the abdomen in the region of the 8th and 9th ribs. This is present in many cases in some epidemics, while in others it is seldom witnessed. Fine or course tremors of the fingers, bilateral and non-intentional in character, are common (McCarthy,[5] 10 per cent.; Dunn and Heagey,[6] 30 per cent.). Tremors of the paralysis agitans type, athetoid movements, and ataxia may occur during the acute stage of the disease, but are more commonly sequels. Movements of the choreiform type may be observed. Convulsions are not infrequent at the onset in children. Hiccup is sometimes associated with clear-cut cases of encephalitis. At other times it occurs as a solitary phenomenon about the time of epidemics of this disease, and has been thought by some observers to be due to the same virus and to represent diaphragmatic myoclonia.

Disturbances of Sensation.—These are not frequent. Generalized hyperesthesia and pains may be early and transient symptoms and may presage the development of hyperkinetic phenomena. Numbness or paraesthesia of the trunk, head or extremities is occasionally witnessed. The pain due to the involvement of the sensory portion of the 5th cranial nerve has already been mentioned, and this may be persistent after the acute stage is over (Dunn and Heagey). Headache is complained of early in some cases and is then usually toxic in origin.

Other Symptoms and Signs.—It is not uncommon to have profuse and continued sweating as a prominent symptom. Marked salivation is seen at times, and frequently-repeated yawning in other cases. Alteration in the respiratory rate and rhythm with hyperpnea or perhaps with suffocation and cyanosis has been described in a number of reports. The peripheral nerves may be involved giving the picture of a polyneuritis. The superficial reflexes may be unaltered or absent and the Babinski reflex absent or present; the part of the central nervous system involved deciding the matter. In the meningeal type the usual signs of rigidity will be found.

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[1] Acute Epidemic Encephalitis, Trans. Assoc. for Research in Nerv. and Ment. Dis., 1921, 50.

[2] New York Med. Jour., 1923, CXVIII, 399.

[3] La presse méd., 1920, XXVIII, 213.

[4] Jour. Amer. Med. Assoc., 1920, LXXIV, 735.

[5] Penna. Med. Jour., 1921, XXIV, 449.

[6] *Loc. cit.*

Special Types.—Some of the more common symptoms have been described and an idea of their frequency given. The determination of the latter is somewhat difficult, owing, as has been stated, to the variation of symptoms in epidemics and localities. It should be remembered that in some cases special features will be prominent, and in some epidemics special types will predominate, the terminology used explaining which symptoms are the most marked. Usually, even in the special types, there will be found other symptoms. Consequently in, for example, the spinal type, or in the meningeal type, or in the cerebellar type there also will be periods when lethargy or ocular paralyses are present. In some instances the initial symptoms may be so mild that the term abortive is justified in designating the variety, and it may be only after mental or motor sequels appear that the true nature of the initial illness is disclosed.

Laboratory Findings.—The *blood-examination* shows slight leucocytosis up to 10,000 or 12,000, although in some typical cases this may rise as high as 20,000 or even 30,000. A slight polymorphonuclear increase is usually noted. The pressure of the *spinal fluid* is slightly increased in about 10 per cent. of the cases. The fluid is practically always clear, and the number of cells normal or not infrequently increased to 20 or 30 per c.mm. and occasionally to 100 or 150. Exceptionally cell-counts of 500 or more have been reported in cases which have been diagnosed encephalitis (Neal).[1] There is a slight increase in albumin and globulin. The amount of sugar is normal or increased, the latter condition being due to, or at least coincident with, a hyperglycemia (Thalhmer and Updegraf).[2] Studies by Halliday[3] would indicate that the amount of sugar is normal provided the spinal fluid is examined after a 12-hour fast. The Wassermann reaction of the spinal fluid is negative, but the colloidal gold test may be positive in the syphilitic zone; *i.e.*, the color-changes are most marked in the lower dilutions. A characteristic finding given by some observers is "cytoglobulin-dissociation;" *i.e.*, cell-count high in proportion to the amount of globulin (Eskuchen).[4] Pellicle-formation is rare. It should not be forgotten that while the various changes described may be present in encephalitis, it is quite common for the spinal fluid to be perfectly normal or to show nothing but a slight pleocytosis. Marked abnormality of the fluid suggests meningeal involvement.

Complications and Sequels.—**Complications,** such as nephritis and respiratory infections, are occasionally seen during the acute course of encephalitis, as in any febrile disease. It is sometimes difficult to determine whether to classify certain end-results as complications or as sequels. They may begin during the acute febrile stage, but may not terminate until after many months or may even be permanent. In other instances a considerable time may elapse between the initial symptoms and the onset of sequels, the patient in the meantime seemingly having returned almost to normal. This late appearance of sequels and the recrudescence of symptoms accompanying this, which at times occurs after a period of quiescence, has been explained on the theory that the virus in the nervous tissues is not destroyed and may resume activity (von Economo;[1] Chiray and Lafourcade[2]). **Sequels** of some kind occur in from 80 to 90 per cent. of the cases and in about ⅓ are serious and either long-continued or permanent. The permanency depends upon the amount of damage which the cells of the central nervous system have undergone. If they are merely diseased, improvement takes place; if they have been destroyed, symptoms do not disappear.

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[1] *Loc. cit.*

[2] Arch. Neurol. and Psych., 1922, VIII, 15.

[3] Quart. Jour. Med., 1926, XVIII, 300.

[4] Zeitschr. f. d. ges. Neurol. u. Psychiat., 1922, LXXVI, 568

Mental and psychic disturbances are common, the usual figures being for all ages about 50 to 60 per cent. of the sequels observed. (Grossman.)[3] In some epidemics in children this may be as high as 75 per cent. of the total number of cases. (Paterson and Spence.)[4] Among minor manifestations are marked irritability, emotional instability, inability to fix attenton, defective memory, delusions, abnormal drowsiness, and fear and compulsion-neuroses. Insomnia (agrypnia) may be very marked, or there may be nocturnal wakefulness and diurnal drowsiness. Wakefulness at night is often associated with hyperkinetic phenomena and with whistling, singing or constant talking. Disturbance of sleep is a frequent disorder (Grossman,[5] 55 per cent. of sequels; Kennedy,[6] in children, 31 per cent. of sequels). In some cases the excessive desire to sleep may be combined with muscular weakness (narcolepsy) (Spiller).[7] Changes in personality and disposition are common (Kennedy,[8] 38 per cent. of sequels) and are shown by anti-social tendencies, lying, stealing, pugnacity and incorrigibility; or the reversal may be demonstrated by amiability and dociliness in a child previously difficult to manage. More serious mental complications are the development of idiocy, manic depressive insanity or that of the type of dementia praecox, and epileptiform convulsions with mental retardation. Mental and motor sequels are liable to be combined.

Among neuro-muscular sequels the so-called Parkinsonian syndrome should be mentioned. Anderson[9] observed it in only 1 instance out of 40 children with encephalitis, and Paterson and Spence[10] in but 1 of 25 children. In our experience it has been uncommon, at least in a typically developed form. Kennedy[11] however, reports the syndrome in 34 per cent. of his 61 children exhibiting sequels; those with symptoms at first not well-developed finally presenting the typical syndrome in its entirety. Tremors, muscular twitchings and choreiform movements are common (Grossman,[12] 58 per cent. of sequels). Athetoid movements are occasionally seen. Residual injuries from involvement of the cranial nerves, especially of the 3rd and 7th, resulting in ophthalmoplegia and facial paralysis, occur in over 50 per cent. of the cases (Grossman,[13]

[1] Münch. med. Wochenschr., 1919, LXVI, 1311.

[2] Bull. et mém. soc. méd. d. hôp. de Paris, 1923, XLVII, 406.

[3] Arch. Neurol. and Psychiat., 1921, V, 580.

[4] Lancet, 1921, II, 491.

[5] *Loc. cit.*

[6] Amer. Jour. Dis. Child., 1924, XXVIII, 158.

[7] Jour. Amer. Med. Assoc., 1926, LXXXVI, 673.

[8] *Loc. cit.*

[9] Quart. Jour. Med., 1923, XVI, 173.

[10] *Loc. cit.*

[11] *Loc. cit.*

[12] *Loc. cit.*

[13] *Loc. cit.*

64 per cent. of sequels). Blindness and deafness are much less common. Such paralyses as monoplegia, paraplegia and hemiplegia are rare. A peculiar respiratory syndrome with hyperpnea lasting for a few seconds or a minute and followed by apnoea is sometimes seen. Parker[1] reports 7 such cases in children, apparently the result of encephalitis. A generalized spasticity has been observed.

Many of these conditions which have been mentioned as sequels may also have been present as symptoms during the acute course of the disease, and this is true of other symptoms as well. Among these are excessive salivation; yawning; talkativeness; precocious sexual sensations, which are not infrequent; and endocrine disorders of the adiposogenital type, instances of which have been reported by Hofstadt,[2] Ebaugh[3] and several others. Disturbances of speech may be observed (Pfaundler)[4] associated with, or independent of, the Parkinsonian syndrome. Alteration of the reflexes persist in 30 per cent. of the cases with sequels (Grossman).[5] There may be pupillary changes and exophthalmos.

Further discussion on the sequels as seen in children will be found in the contributions of Happ and Blackfan,[6] Leahy and Sands,[7] Hohman,[8] Collin and Réquin[9] and others.

Course and Prognosis.—The consideration of this subject involves not only the mortality during the acute stage, but that observed later also, as well as the end-results of complications and sequels. The early mortality is usually high, in many reported series equalling about 20 to 30 or even 50 per cent. It is sometimes only 10 per cent. or lower, as in the series of mixed adults and children of Happ and Mason[10] or in Comby's[11] series in children. In Neal's[12] statistics the death-rate was the same in the patients under 15 years as in those older. In young infants the mortality may be very high, Duzár and Baló[13] reporting 10 deaths in 11 infants. Sudden onset indicates a bad prognosis according to Smith,[14] in whose 153 cases of all ages, there was a total mortality of 29 per cent., and one of 60 per cent. in those with sudden onset.

It is well to remember that the statistics given above may lead to a false idea of the mortality. They are taken from studies on hospital cases, and as such represent mostly the severer forms of the disease. Many of the milder and of the abortive cases, which probably constitute a fairly large proportion, are not included because not diagnosed. In others, as already mentioned, the children are seen for the first time only after signs of residual injury of the brain indicate that an encephalitis had in reality been existent. If the unrecognized cases of both these classes were added to the recognized severer instances, the mortality-percentage would be much lowered.

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[1] Arch. Neurol. and Psychiat., 1922, VIII, 630.
[2] Jahrb. f. Kinderheilk., 1921, XXIX, 272.
[3] Amer. Jour. Dis. Child., 1922, XXV, 89.
[4] Jahrb. f. Kinderheilk., 1920, XCIII, 379.
[5] *Loc. cit.*
[6] Jour. Amer. Med. Assoc., 1920, LXXV, 1337.
[7] Jour. Amer. Med. Assoc., 1921, LXXVI, 373.
[8] Johns Hopk. Hosp. Bull., 1922, XXXIII, 372.
[9] Arch. de. méd. des enf., 1923, XXVI, 265.
[10] *Loc. cit.*
[11] Arch. de. méd. des enf., 1921, XXIV, 457.
[12] *Loc. cit.*
[13] Jahrb. f. Kinderheilk., 1922, XCIX, 209.
[14] *Loc. cit.*

The *duration* of symptoms in uncomplicated, acute cases varies from 2 to 8 weeks. Of those who survive the acute stage some die later as a result of complications and sequels, and in any event the prognosis should be very guarded as to final complete recovery of health. This is because of the absence of reparative process characteristic of brain-tissue. A hopeful attitude may be taken in some cases because, while it is true that about ⅔ of the survivors will develop sequels, not all of these will exhibit progressive nervous or mental disorder. The severity of the initial illness seems to bear some relation to the prognosis of functional recovery. A short illness with a few days of lethargy usually eventuates in complete restoration of health. It is difficult to give figures as to the number of patients with sequels who will improve. According to Ebaugh[1] 50 per cent. of children whom he observed over a long period were well or improving. Improvement in the presence of serious sequels usually does not begin until after 6 months and may continue for 2 years. Kennedy's[2] statistics were not so favorable. Of the 16 children with the Parkinsonian syndrome none improved; of 23 with change in personality 6 showed improvement; of 19 with disturbance of sleep 9 recovered and 3 improved; and in 17 with the respiratory syndrome 2 recovered and 3 improved.

Diagnosis.—A typical case of encephalitis with lethargy, asthenia, fever and ophthalmoplegia, especially if occurring at the time of an epidemic, offers no great trouble in diagnosis. The chief diagnostic difficulties will be with poliomyelitis and tuberculous meningitis. In *poliomyelitis* of the encephalitic type a clinical differentiation is not possible. In the ordinary type of this disease the predominance of spinal symptoms, the hyperesthesia, the prompt appearance and rapid extension of paralysis, the absence of marked lethargy, the rarity of ocular paralyses, and the prevalence in the summer-season will help to distinguish it from encephalitis. There is so little difference in the cytology of the spinal fluids in the two conditions that differential diagnosis is seldom helped by this examination. In *tuberculous meningitis* the onset is more gradual, there is lower fever at the onset, the symptoms run a more rapid course with increasing severity and more marked signs of rigidity, there may be found evidence of tuberculosis elsewhere, as in the lungs or the choroid, and the age of greatest incidence is in infancy. A positive tuberculin-reaction is of little aid, since it may sometimes be absent in tuberculous meningitis or present in a tuberculous individual with epidemic encephalitis. The differences in the spinal fluids in the two diseases should settle the differential diagnosis, except in an early case of tuberculous meningitis before meningeal reaction has become marked. In tuberculous meningitis the increased pressure, the definitely increased cell-count, the film-formation, and later the diminished or absent sugar are all characteristic findings which are not liable to be present in encephalitis. The discovery of tubercle-bacilli in the fluid is of course at once conclusive. The white cells in the blood in tuberculous meningitis are more likely to be increased than in either encephalitis or poliomyelitis, but dependence cannot be placed upon this. *Syphilis* of the central nervous system has an insidious onset and a chronic course, and other evidences of the disease will be found; optic nerve changes are common, and the Wassermann reaction of the blood and spinal fluid is almost invariably positive. *Intracranial tumor* is a comparative uncommon affection in childhood, is afebrile in its course, and early produces choke disc and eventually localizing symptoms. *Cerebral abscess* causes septic fever, localizing symptoms, a high leucocytosis and possibly a positive blood-culture. A focus of infection may be found, usually in the ear. *Uremia* should be diagnosed by evidences of renal disease and the examination of the urine. *Meningitis* other than tuberculous will readily be distinguished by the study of the spinal fluid. *Botulism* develops within a few hours to a day or two after eating infected food. There is no fever, sensory disturbances are absent, there is no mental impairment although drowsiness may be marked, and there is paralysis without spasticity, especially in the muscles of the neck. The pupils are widely dilated. Toxins or the bacillus botulinus may be discovered in the suspected food. In *poisoning by hypnotic drugs*, such as veronal, cocaine, morphine, methanol, alcohol, etc., there may be a history of the ingestion of the drug, there will be subnormal temperature or at least no elevation of it, the coma may be profound, and other findings characteristic of the action of the drug may be present, such as odor to the breath, pupillary changes, slow respiration, etc. *Hysteria* may be ruled out by the persistence of symptoms, a hysterical patient not showing such continuance of tremors, local paralyses, lethargy or disturbed mental state, and lacking ocular paralyses. *Sydenham's chorea* differs from the choreiform type of encephalitic sequel in its more acute course, in the frequent association with rheumatic manifestations, and in the absence of lethargy, ophthalmoplegia or other encephalitic symptoms. In *plumbic encephalitis* there will be the history of exposure to that metal, the lead-line in the gums, the characteristic blood-picture with stippling of the cells, the absence of fever and the presence of frequent convulsions. *Encephalitis following acute infections*, such as measles, scarlet fever, pertussis or vaccination, may be suspected by the history of its relation to some of these conditions. Loewe and Strauss[1] have suggested a means of diagnosis in which Berkefeld filtrates of nasopharyngeal washings or cerebrospinal fluid from suspected cases are injected intracranially into rabbits. Characteristic lesions are said to be produced in these animals in about 75 per cent. of the cases. Sato and Yoshimatsu[2] claim that the myeloid leucocytes present in epidemic encephalitis always give a negative peroxidase reaction with the copper method, in contradistinction to all other diseases, the reaction being positive in these.

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[1] Amer. Jour. Dis. Child., 1923, XXV, 89.

[2] *Loc. cit.*

Treatment.—In the acute stage there is indicated the usual attention given to patients suffering from any infectious fever. Rest and quiet are important and care should be taken to keep up the fluid intake and nourishment, gavage being resorted to if necessary. In hyperactive conditions the intake of food must be greater than the average in order to balance the increased metabolism. Hot packs and warm baths have a soothing effect in hyperactive and excitable states, and are of more service than are sedative drugs. In the cases with ocular paralyses there should be protection of the eyes and frequent flushings with boric acid solution; and if the cornea becomes dry from exposure mineral oil should be instilled into the conjunctival sac. The skin should be guarded against irritation and pressure, and the bladder watched for retention. There is a difference of opinion concerning the advisability of lumbar puncture. Dercum[3] believes that the sudden reduction in intracranial pressure thus caused favors the diffusion of virus throughout the brain. Many clinicians, however, perform lumbar puncture at intervals of 3 or 4 days. In those cases in which there is an increase in pressure we found that an improvement in the condition follows the relief of this; and with any meningeal symptoms puncture is definitely indicated. In fine, the entire treatment of the acute stage of encephalitis is symptomatic, and will remain so until the cause is definitely known and specific therapy can be evolved.

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[1] Jour. Amer. Med. Assoc., 1920, LXXIV, 1373

[2] Amer. Jour. Dis. Child., 1925, XXIX, 301.

[3] New York Med. Jour., 1923, CXVIII, 397.

The *treatment of sequels* is also mainly symptomatic. In the mental sort rest and protection against nervous excitement are necessary. Hydrotherapy has more effect than hypnotics on insomnia. In some instances when inversion of the sleeping-hours is marked, keeping the patient awake during the day may eventually cause a return to a normal habit. In the respiratory syndrome medication usually has no effect, although occasional cases are apparently aided by luminal. *X*-ray treatment of the parotid gland has an inhibiting influence in excessive salivation.

Special training should be carried out when special senses are involved, and active and passive motion in paralytic states and also in hyperkinetic conditions is advised by different clinicians. On the latter, too, atropine seems to have some effect.

Certain *special forms of treatment* recommended should be mentioned. During the acute stage of the disease benefit has been claimed from several procedures. Helmholz and Rosenow[1] utilized a serum from a horse, which had been inoculated with a streptococcus derived from the nasopharyngeal mucous membrane and brain of cases of encephalitis. This was injected intravenously and intramuscularly. Piticariu[2] gave intravenous injections of the patient's own spinal fluid, and Moore[3] injected the patient's own blood-serum intraspinally. Convalescent's blood-serum has also been used (Grünewald),[4] as well as intraspinal injection of convalescent's spinal fluid. Carnot and Blamoutier[5] injected sodium salicylate intravenously. Bucks[6] gave hypertonic salt-solution intravenously, thus reducing intracranial pressure. During the acute stage we have had seemingly excellent results from hypertonic solutions of glucose given intravenously. Sison[7] claimed good results from the injection of convalescent serum, and Strecker and Willey[8] believed that benefit was obtained in the sequels by the use of intravenous injections of a solution of neutral acriflavine. Later studies by Strecker[9] indicate that the most good is to be expected in the treatment of the acute cases. With regard to these various forms of intraspinal and intravenous therapy in encephalitis it is evident that further investigation is indicated.

Some physicians have made attempts to influence the cells of the central nervous system in the presence of persistent sequels, perhaps with the hope of stimulating those which are diseased but not destroyed. With this in view, Leiner,[10] administered sodium nucleinate; Lust[11] used intramuscular injections of milk for insomnia, and Roch[1] gave intraspinal injections of casein to produce a reactive aseptic meningitis. The advantage of any of these procedures seems very doubtful.

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[1] Jour. Amer. Med. Assoc., 1922, LXXIX, 2068.

[2] Wien. klin. Wochenschr., 1922, XXXV, 441.

[3] Calif. State Jour. Med., 1922, XX, 387.

[4] Deutsch. med. Wochenschr., 1920, XLVI, 1243.

[5] Paris méd., 1923, XIII, 177.

[6] Jour. Oklahoma Med. Assoc., 1922, XV, 308. Ref. Quarterly Cumulative Index, 1922, 632.

[7] Phillippine Isl. Assoc. Journ., 1924, 306.

[8] Amer. Jour. Psychiat., 1925, IV, 631.

[9] Personal communication.

[10] Jour. Amer. Med. Assoc., 1923, LXXXI, 1284.

[11] Deutsch. med. Wochenschr., 1921, XLVII, 1545.

CHAPTER XXI

TUBERCULOSIS

Tuberculosis affects many different parts of the body, the local symptoms usually greatly preponderating over the general ones, and producing clinical pictures essentially different from each other. On this account, and for the sake of greater convenience of study, some, at least, of the special forms of tuberculosis will be discussed more in detail in different sections. In the present chapter tuberculosis as a whole will be considered, especially as it affects children, avoiding repetition of the subject-matter of other chapters as far as possible.

Etiology. **Frequency.**—As far back as the history of medicine extends tuberculosis has existed as one of the most frequent of diseases. It affects many different varieties of animals, although to unequal degrees; being most prevalent in man, monkeys, cattle and swine, as well as in poultry, although in the last it is a decidedly different disorder. It is rare in sheeps, goats, horses, dogs and cats. Guinea-pigs and rabbits are very susceptible if inoculated. The actual frequency of tuberculous infection, apart from the occurrence of lesions found at autopsy, is difficult to determine. Not all cases which show post-mortem lesions have died of this disease. In many it is only a contributory or an accidental matter. On the other hand, tuberculous infection exists in many cases which do not come to autopsy. Of 4388 children in the schools of Christiania, as reported by Fröhlich,[2] 2900 were examined clinically and by the cutaneous tuberculin-reaction. The clinical study revealed evidences of tuberculosis in 5.15 per cent., and the tuberculin-test in 83.8 per cent. The influence of the age of the subject upon the frequency of the disease will be considered later (p. 627).

Predisposing Causes.—Much discussed in this connection is the influence of heredity, the difficulty being to distinguish between the effect of heredity and the occurrence of exposure. While direct inheritance of the germ is rare, that of the disposition to develop tuberculous disease is very common. Any debilitated condition of the parents diminishes the resistance in the offspring, but the existence of parental tuberculosis is by far the most powerful factor. The acquiring of the disease by association with tuberculous parents and not on account of an inherited tendency is to be carefully excluded in drawing conclusions. The effect of association is illustrated by the studies of Armand-Delille and Famin[3] who found that, of a series of children remaining in contact with tuberculous parents, 60 per cent. became infected and 40 per cent.

[1] Presse méd., 1923, XXXI, 496.

[2] Norsk. Mag. f. Laegevidensk., 1914, LXXV, 137. Ref. Monatsschr. f. Kinderheilk. Ref., 1915, XIV, 379.

[3] Bull. acad. de méd. Paris, 1924, XCI, 479.

died of acute tuberculosis before the death of the parents occurred. Race exerts some etiological influence, but the effects attributed to it depend upon other associated conditions. The Indians, Irish and Negroes appear especially predisposed in the United States. The people of primitive races, who have not had tuberculosis as an endemic disease, readily become infected on exposure and develop rapid forms. Climate, locality and altitude are prominent factors. With some exceptions damp regions predispose, and dry, elevated localities are unfavorable to the occurrence of the malady. The frequency of tuberculosis in children on the continent of Europe would appear from statistics to be much greater than in the United States, and in certain cities the number of instances of infection developing in early life seems to be particularly high. Unhygienic conditions in general are of great importance. The crowding of children in dirty buildings, the lack of fresh air and sunlight, insufficient nourishment, and the like, are prominent predisposing causes. The influence of poor hygiene and improper food became evident in the increase of tuberculosis in Europe following the World War (Emerson).[1] Figures given by Pape,[2] based on a study of 10,840 histories from 1910 to 1920, show very strikingly the increase in the number of deaths during the war-time, especially marked in the early years of life; the mortality-rate increasing for instance, in the 1st year from 63 per cent. to 94.4 per cent. Previously impaired health also predisposes, especially derangements of the respiratory mucous membrane, and, to a less extent, those of the alimentary tract. Many other disorders predispose, particularly to be mentioned here being measles, pertussis, grippe, repeated attacks of bronchitis, and the existence of hypertrophied tonsils and of adenoid growths. These various agents may act either by rendering the subject liable to the entrance and development of the germs, or by decreasing the local or general resistance of the organism and thus allowing an infection already present in the body in a quiescent condition to assume an active form.

Age exerts a very positive influence. Congenital tuberculosis is occasionally seen, as was proven by Schmorl and Birch-Hirschfeld[3] and others, and a considerable number of cases have been reported (Pehu and Chalier).[4] The occurrence is very possibly more common than generally supposed. The subject has been reviewed by Weber[5] and by Grulee and Harms,[6] and more recently by Whitman and Green,[7] who estimate there are on record 113 authenticated instances and a much larger number with more uncertainty attached. Tuberculosis is, however, exceptional in the first 3 months of life, but increases rapidly in frequency of occurrence after this period. Cornet[8] gives the following figures based on 1542 autopsies on children under 5 years of age:

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[1] Amer. Rev. Tub., 1924, IX, 191.
[2] Beiträge z Klin. d. Tuberc., 1921, XLVIII, 24.
[3] Beiträge z. path. Anat. u. z. allg. Path., 1891, IX, 429.
[4] Arch. de. méd. des enf., 1908, XI, 1.
[5] Brit. Jour. Child. Dis., 1916, XIII, 321; 359.
[6] Amer. Jour. Dis. Child., 1915, IX, 322.
[7] Arch. Int. Med., 1922, XXIX, 261.
[8] Nothnagel's Encycl. Pract. Med., Amer. Ed., Tuberculosis, 307.

Table 87.—Incidence of Tuberculosis as Derived from Autopsies

	Autopsies	Tuberculous	Percentage
Birth to 2 mos	486	0	0
2 to 3 mos	33	2	6.06
3 to 6 mos	76	8	10.53
6 to 9 mos	88	15	17.05
9 to 12 mos	65	18	27.69
Total under 1 yr	748	43	5.75
1 to 2 yrs	311	83	26.69
2 to 3 yrs	189	56	29.63
3 to 4 yrs	160	51	31.86
4 to 5 yrs	134	30	22.39
Total	1542	263	17.5

There exists, however, considerable variation in many of the published statistics, as may be seen in the following selected examples: In 752 autopsies in children reported by Feldmann[1] tuberculosis was present in 19.6 per cent.;—under 3 months, 6.3 per cent. of all autopsies at this age; 3 to 6 months, 22.8 per cent.; 7 to 9 months, 29.1 per cent.; 10 to 12 months, 22.4 per cent.; 1 to 2 years, 43.1 per cent.; over 2 years, 71.1 per cent. Schlossmann[2] gives 6.8 per cent. tuberculous in 532 autopsies in infants under 1 year. According to Hamburger[3] of 848 autopsies in children in Vienna, 39.5 per cent. showed tuberculosis. In the first 3 months it was present in 4 per cent.; 4 to 6 months, 18 per cent.; 7 to 12 months, 23 per cent.; in the 2d year, 40 per cent.; 3 to 4 years, 60 per cent.; 5 to 6 years, 56 per cent.; 7 to 10 years, 63 per cent.; 11 to 14 years, 70 per cent. The percentages in the records of the Children's Hospital of Philadelphia (Hand)[4] are also high; 115 of 332 autopsies, or 34.6 per cent., revealing tuberculous lesions, ½ of these cases being in subjects under 2 years of age. More favorable are the figures of Wollstein and Spence,[5] who in 2024 autopsies in children of 5 years or less found tuberculous infections in only 9 per cent. Harbitz[6] for Norway found tuberculous lesions in 40 per cent. of 484 autopsies in children from 1 to 15 years of age. The Paris mortality is shown by the statistics of Comby[7] upon 1675 autopsies with 638 (38 per cent.) tuberculous. The division according to age was: Of autopsies under 3 months, tuberculous 1.82 per cent.; 3 to 6 months, 18.18 per cent.; 6 to 12 months, 26.25 per cent.; 1 to 2 years, 40.72 per cent.; 2 to 5 years, 60 per cent.; 5 to 10 years, 67.15 per cent.; 10 to 15 years, 71.23 per cent. In 499 autopsies on infants in the first 2 years of life, Barbier[8] observed tuberculosis in 39 per cent.

The figures as given represent, however, only the finding of tuberculous lesions at autopsy. They do not always indicate that the death was due to this disease, nor do they show how often children of different ages, who do not come to autopsy, are subjects of tuberculous infection. In fact, the older the child, the oftener is the tuberculous lesion a latent or healed process. The question of the frequency of tuberculous infection at different periods as shown by the tuberculin-test (see p. 651) has been carefully studied in many quarters. The figures obtained vary somewhat with the locality, as may be seen in the following table:

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[1] Budapesti orvosi ujśay, 1906. Ref., Jahrb. f. Kinderheilk., 1906, LXIV, 763.

[2] Beiträge z. Klin. d. Tuberc., 1906, VI, 229.

[3] Wien. klin. Wochenschr., 1907, XX, 1069.

[4] Arch. of Pediat., 1903, XX, 247.

[5] Amer. Jour. Dis. Child., 1921, XXI, 48.

[6] Norsk. Mag. f. Laegevidensk., 1913, 1. Ref. Monatsschr. f. Kinderheilk., Ref., 1914, XIII, 429.

[7] XVII Intern. Cong. of Med., 1913, Sect. X, 38.

[8] Paris méd., 1917, VII, 109.

TABLE 88.—INFLUENCE OF LOCALITY ON AGE-INCIDENCE OF TUBERCULOUS INFECTIONS

Lapage,[1] Manchester			Veeder and Johnston,[2] St. Louis		
Age	Number of cases	Positive reactions, per cent.	Age	Number of cases	Positive reactions, per cent.
0–2 yrs	103	32.0	Under 1 yr	202	1.5
2–5 yrs	209	51.2	1–2 yrs	109	5.5
5–10 yrs	446	60.6	2–4 yrs	163	19.0
10–14 yrs	242	60.8	4–6 yrs	172	23.0
			6–8 yrs	152	29.0
			8–10 yrs	126	30.0
			10–12 yrs	107	34.0
			12–14 yrs	94	38.0
Total	1000	55.7	Total	1125	21.0

Hamburger and Monti,[3] Vienna			M'Neil,[4] Edinburgh		
Age	Number of cases	Positive reactions, per cent.	Age	Number of cases	Positive reactions, per cent.
Under 1 yr	23	0.0	Under 1 yr	64	14.1
1–2 yrs	46	9.0	1–2 yrs	61	29.9
2–3 yrs	56	20.0	3–4 yrs	75	46.6
3–4 yrs	75	32.0	5–6 yrs	52	28.8
4–5 yrs	50	52.0	7–10 yrs	79	51.9
5–6 yrs	63	51.0	11–14 yrs	40	55.0
6–7 yrs	46	61.0			
7–8 yrs	30	73.0			
8–9 yrs	35	71.0			
9–10 yrs	26	85.0			
10–11 yrs	29	93.0			
11–12 yrs	19	95.0			
12–13 yrs	17	94.0			
13–14 yrs	17	94.0			

The British statistics differ from the others in the much higher percentage of positive cases during the 1st year. Midway come the figures of McLean and Jeidell[5] with 7.35 per cent. positive Pirquet reactions in the 1st year. The figures of Gittings and Donnelly[6] on 1219 cases more nearly approach to those of M'Neil for the early years; viz: under 1 year, 15 per cent., 1 to 2 years 22.33 per cent., and 2 to 4 years 24.75 per cent.

The cases of Lapage showed in some instances clinical evidences of tuberculosis; those of Veeder and Johnston and of Hamburger and Monti did not; those of M'Neil were unselected. The cutaneous test was the only one employed except in the Vienna cases, where the subcutaneous

[1] Brit. Jour. Child. Dis., 1912, IX, 493.
[2] Amer. Jour. Dis. Child., 1915, IX, 478.
[3] Münch. med. Wochenschr., 1909, LVI, 449.
[4] Edinburgh Med. Jour., 1912, I, 324.
[5] Amer. Jour. Dis. Child., 1922, XXIV, 73.
[6] Arch. of Pediat., 1921, XXXVII, 78.

was used in cases not responding to the cutaneous. The statistics show in general that the older the child the more liable is a positive tuberculin-reaction to be present. This indicates an infection, but not the existence of what may be called tuberculous disease. The comparatively small number of instances of even tuberculous infection in the 1st year of life is noteworthy, and would seem to indicate a certain degree of immunity at this period. Barchetti[1] studied 51 infants born of tuberculous mothers. Of those born healthy and nursed by the mothers over a considerable period 27 per cent. failed to give a positive reaction.

The obtaining of a positive tuberculin-reaction is not a proof that the clinical manifestations which the child may exhibit necessarily depend upon tuberculosis, but only that this infection exists somewhere in the body, perhaps as a small, entirely inactive focus. Vonessen[2] tested 550 children of school-age for the Pirquet reaction. Although finding it in 57 per cent., manifest tuberculosis was observed in only 2 instances. It is probable that children of the better classes, and not examined when suffering from any symptoms, would not give a positive reaction so often. Brüning[3] in the examination of 350 children in private practice, from infancy to late school-age, obtained a positive cutaneous reaction in only 26 per cent. Whatever other bearing these various statistics may have, the frequency with which tuberculous lesions are found in children dying from other disorders, and the large number of cases in which a positive tuberculin-reaction is obtained in those apparently without tuberculous disease, appears to favor the view of Schlossmann[4] and of Hamburger[5] that tuberculosis is to a large extent a children's disease, acquired in childhood and often recovered from at this time; although it may in other cases lie dormant and break out actively in later life. This raises the question of the existence of an immunity to tuberculosis. Wassermann,[6] summing up our knowledge of the subject, states that this exists, but only in those who already have tubercle-bacilli present in the body, and that the immunity to reinfection lasts only so long as the local lesions stay dormant; while Köffler[7] adopts the view that if such an individual is exposed to massive doses of bacilli, serious reinfection may take place. The tuberculin-reaction is, then, an indication of the resistance of the organism to reinfection (Hamburger).[8]

Exciting Cause.—That the disease is an infectious one was believed from early time, but was first clearly proven by Villemin[9] in 1865. That it was dependent upon the tubercle-bacillus was first demonstrated by Koch[10] in 1882. Since the studies of Theobold Smith,[11] most authors agree that there appear to be 2 varieties of the bacillus capable of producing the disease in man: the bovine and the human. The relationship and relative importance of these is still a subject of much discussion. It appears to be proven that the human bacillus can occasionally cause tuberculosis in cattle. In like manner, the bovine germ can certainly produce the disease in man, but the large majority of cases owe their origin to the human variety. This would seem, at least, to be the most frequent experience, although certain investigators, notably of Scotland, among them Mitchell,[1] Fraser,[2] and others, insist on the etiological importance of the bovine bacillus especially in children, and chiefly in the production of tuberculosis of the cervical and mesenteric glands and of the bones and joints. There would appear to be a difference in the prevalence of the two forms of the bacillus in different countries, since in the United States, at least, infection by the bovine bacillus is very much less frequent. Park and Krumwiede's[3] statistics show that tuberculosis of the lungs, meninges and osseous system in children is nearly always of human origin, but that the disease in the intestines and the cervical glands is not infrequently caused by the bovine type; and that there is a greater tendency for bovine tuberculosis to develop in children than in adults. Their statistics are based upon their own and collected reports of cases, and equal 1511 examinations in all, or 545 in subjects up to 16 years of age. The following table is taken from their publication.

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[1] Monatsschr. f. Kinderheilk., 1921, XXI, 563.
[2] Beitr. z. Klin. d. Tuberc., 1922, XLIX, 357.
[3] Jahrb. f. Kinderheilk., 1921, XCVI, 286.
[4] Münch. med. Wochenschr., 1909, LVI, 398.
[5] Münch. med. Wochenschr., 1908, LV, 2702.
[6] Zeitschr. f. Tuberc., 1921, XXXV, 1.
[7] Arch. f. Kinderheilk., 1921, LXX, 95.
[8] Pfaundler and Schlossmann's Handb. d. Kinderheilk., 1923, II, 505.
[9] Gaz. hebdom, 1865, II, 795.
[10] Berlin. klin Wochenschr., 1882, XIX, 221.
[11] Jour. Exp. Med., 1898, III, 451.

Table 89.—Incidence of Human and Bovine Tuberculosis, Respectively

Diagnosis	Children 5 to 16 years		Children under 5 years	
	Human	Bovine	Human	Bovine
Pulmonary tuberculosis	14	..	35	1
Tuberculous adenitis (axillary or inguinal)	4	..	2	..
Tuberculous adenitis (cervical)	36	22	15	24
Abdominal tuberculosis	8	9	10	14
Generalized tuberculosis of alimentary origin	3	4	17	15
Generalized tuberculosis	5	1	74	7
Generalized tuberculosis including meningitis; alimentary origin	1	..	5	10
General tuberculosis including meningitis	10	..	76	1
Tuberculous meningitis	3	..	28	4
Tuberculosis of the bones and joints	41	3	27	
Genito-urinary tuberculosis	2			
Tuberculosis of the skin	4	6	2	
Miscellaneous	..	1	1	

Not all investigations are in accord with these statistics. For example, Gordon and Brown[4] record that of 30 cases of tuberculosis in children, ⅓ were due to the bovine type. Rosenau[5] in 1040 collected cases, many of whom, it must be stated, were selected, found the bovine type as follows:—16 years and over, 1.3 per cent.; 5 to 16 years, 25 per cent.; under 5 years, 49.1 per cent.

The germs are found in tuberculous lesions wherever situated and in the secretions and excretions from the affected tissues, particularly when the process is active. Outside of the body they are widely diffused and very abundant, the chief source being the sputum, in which they are present in enormous numbers. They are capable of living and remaining virulent for weeks in a dried state in the dust from rooms occupied by consumptive patients (Cornet),[6] but are killed by a temperature of 60°C.

[1] Edinburgh Med. Jour., 1914, XIII, 209.
[2] Jour. Exper. Med., 1912, XVI, 432.
[3] Jour. Med. Res., 1912–13, XXVII, 109.
[4] Amer. Jour. Dis. Child., 1923, XXV, 234.
[5] Preventive Med. and Hyg., 1916, 138.
[6] Zeitschr. f. Hyg., 1888, V, 191.

(140°F.) continued for from 15 to 20 minutes or of 70°C. (158°F.) for 5 minutes (Brown).[1] The general opinion is that they are seldom to be found in the blood, their presence there being a variable and transitory phenomenon.

Mode of Transmission and Portal of Entry.—Except for the rare cases of congenital tuberculosis in which the infection is through the blood, and the exceptional cases of tuberculosis by direct inoculation, as by ritual circumcision or through other lesions of the skin, the germ enters the body either by the respiratory or the alimentary tract. When infection is by the inspired air, the tubercle-bacilli, often attached to particles of dust or to fine, moist drops expelled by the coughing of some patient, are deposited upon the mucous membrane of the nasopharynx, larynx, trachea, bronchi or lungs. If any catarrhal or other lesion exists the penetration of this membrane is rendered much easier, but it can take place without a lesion being present. It has been believed that a tuberculous infection either may develop at the site of invasion, or that the germs may pass through the mucous membrane without injury to it. It is probable, however, as maintained by Parrot,[2] Küss,[3] Albrecht,[4] Ghon[5] and others, that the primary tuberculous lesion is always situated at the portal of entry, although it may be small and undiscovered except by the most careful search. Canti[6] observed this primary pulmonary lesion in 10 out of 16 cases of tuberculous lesions in children, in 8 of them single, and in all just below the pleura; and Fischl[7] discovered it in 35 out of 40 autopsies. Later investigations by Ghon and Wertheimer[8] found the primary lesion, next to the lung, oftenest in the alimentary tract, especially the intestine. There is a tendency here to healing with scar tissue, never calcification. Primary lesions outside of the lung were, however, comparatively uncommon; only 5 in 184 cases in the Vienna material, and 9 of 395 cases in that from Prague. The primary pulmonary lesion suffers the usual changes of caseation, calcification, softening or cicatrization, and from this focus the bacilli travel by way of the lymphatic vessels to the regional lymphatic glands, viz. the tracheobronchial. These act as sieves and may arrest the further progress of the germs, which can lie dormant here for an indefinite time, as shown by the investigations of Loomis,[9] Pizzini,[10] Beitzke[11] and others, exerting no influence whatever on the general health, although the condition is an ever-present menace. Infection of the organs, or of the system at large, is the result of the inflammatory process increasing in the primary focus or in the lymphatic glands, and the bacilli extending thence by way of the lymphatics; or by the process finally involving the blood-vessels and the germs then entering the blood-current.

When the infection is by the alimentary tract the bacilli enter from the pharynx, tonsils, esophagus, stomach, or intestine, being attached to the food, or obtained from toys, cooking utensils and the like, or even from the infant's fingers. As with the respiratory tract there is usually a tuberculous lesion, although small, at the portal of entry. When the intestine is the seat of the primary lesion, the bacilli pass thence into the mesenteric glands and finally to other parts; when entrance is by way of the tonsillar tissue the cervical glands generally arrest further extension of the disease.

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[1] Lancet, 1923, II, 317.
[2] Compt. rend. soc. de biol. de Paris, 1876, III, 308.
[3] De l'hérédité parasitaire de la tuberculose humaine, Paris, 1898. Ref. Ghon.
[4] Wien. klin. Wochenschr., 1909, XXII, 327.
[5] Der primäre Lungenherd bei der Tuberkulose der Kinder, Berlin, 1912.
[6] Quart. Jour. Med., 1919, XIII, 71.
[7] Arch. f. Kinderheilk., 1922, LXXI, 6.
[8] Med. Klinik, 1923, XIX, 26.
[9] Med. Rec., 1890, XXXVIII, 689.
[10] Zeitschr. f. klin. Med., 1892, XXI, 329.
[11] Virchow's Archiv., 1912, CCX, 173.

Whether infection by the respiratory or the alimentary tract is the more frequent in children has been, and still is, a subject of widespread-discussion. Except in the British statistics there is little question, as post-mortem examination has repeatedly shown, that primary lesions are much more often found in the region of the respiratory tract, including the tracheobronchial lymph-glands (see p. 647), and a logical conclusion would appear to be that the gland-filter situated nearest to the portal of entry will be the one first affected. On the other hand, it has been claimed by those who maintain that infection by way of the intestine is the more common, that the bacilli are not filtered out by the mesenteric glands, but are carried to the general circulation by the thoracic duct and finally are deposited in the lungs and the tracheobronchial glands, which are the parts most susceptible to the disease in early life; and that consequently apparently primary lesions of these glands do not exclude the possibility of alimentary infection.

Even experimental evidence is contradictory. Thus, as illustrating the opposing views, Straus[1] showed that tubercle-bacilli injected into the stomach readily cause a general tuberculosis, and Calmette, Guerin and Déléarde[2] that respiratory involvement could be produced in this way without any affection of the mesenteric glands. On the other hand Cornet,[3] Pfeiffer and Friedberger,[4] and Findlay[5] demonstrated the great ease with which respiratory tuberculosis could be brought about by inhalation experiments, and that injection of germs into the stomach produced tuberculosis with difficulty. Nearly all recent clinical and experimental evidence is in favor of the very much greater frequency of infection by inspiration, especially of droplets containing bacilli.

Directly bearing upon this matter is the question of the danger of milk from tuberculous cattle. That this exists has been practically denied by Koch,[6] but affirmed by von Behring[7] and other investigators. The preponderance of the evidence at the present time seems to indicate that infection with the bovine bacillus contained in milk from tuberculous cattle is a possibility, but comparatively infrequent; and that the danger is much over-rated. Cattle which react to tuberculin but reveal no tuberculous foci usually have no bacilli in their milk. The same is true of infection by the human bacillus in the milk of tuberculous mothers. Wang and Coonley[8] examined the milk of 28 tuberculous women and obtained entirely negative results except in 1 questionable case. More is to be feared from the contamination of cows' milk by the *human* bacilli which may enter it from the air, just as they may contaminate any other article which enters the infant's mouth. At the same time it is unwise to employ milk from tuberculous cattle; and whether or not the milk from a tuberculous mother is dangerous, the intimate association of the infant with her certainly is so, and should be prevented. As regards the flesh of tuberculous cattle, infection by it is perhaps possible, but certainly unlikely.

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[1] Arch. méd. expér., 1896, VIII, 689.
[2] Compt. rend. acad. sci., 1906, CXLII, 1136.
[3] Nothnagel's Encyclop. Pract. Med. Tuberc., 98; 143.
[4] Deutsch. med. Wochenschr., 1907, XXXIII, 1577.
[5] Zeitschr. f. Kinderheilk., Orig., 1913, VII, 503.
[6] Brit. Med. Jour., 1902, II, 1885.
[7] Deutsch. med. Wochenschr., 1903, XXIX, 689.
[8] Jour. Amer. Med. Assoc., 1917, LXIX, 531.

Pathological Anatomy. The Histology of Tubercle.—The basis of the pathological changes of tuberculosis is the miliary tubercle, which is produced by the irritation of the rapidly multiplying bacilli. It consists of a grey, translucent body about 1/20 to 1/5 inch (0.127 to 0.5 cm.) in diameter surrounded by a reticulum of connective tissue, and is composed of lymphoid and epithelioid cells the result of proliferation of the tissue-cells, and of leucocytes, at first polymorphonuclear, later chiefly mononuclear, migrating from the neighboring blood-vessels. Bacilli are present in some of the epithelioid cells. In some of the tubercles giant-cells are also found, their number being inversely proportional to the number of bacilli present. The tubercles may remain scattered and mostly separated from each other, or may be grouped in large masses. As growth progresses degenerative changes take place. Caseation is the most common, the tubercle becoming yellow, containing many bacilli, and finally softening. This is followed by an inflammation of the surrounding tissue and the formation of pus through a mixed infection. Less frequently calcification occurs, seen oftenest in the lymphatic glands. Sometimes, especially if the tubercles remain discrete and miliary, sclerosis takes place, a firm fibrous structure resulting.

Regions Oftenest Found Affected by Tuberculosis at Different Periods of Infancy and Childhood.—*Age* is a prominent factor in this connection. It is to be borne in mind that the process is seldom limited to any one locality, and that the region most frequently attacked does not necessarily produce the most important symptoms. In fact, tuberculosis may be only a post-mortem finding, death being dependent upon some other disease. Taking infancy and childhood as a whole, the statistics compiled by Freeman[1] in 2288 autopsies in tuberculous children reported by different observers show involvement of the lungs in from 71 to 100 per cent.; of the bronchial lymph-nodes in from 76 to 99 per cent.; of the intestines in from 23 to 55 per cent.; and of the mesenteric lymph-nodes in from 16 to 40 per cent.

In the *first 2 years of life* the regions oftenest found diseased are the bronchial lymphatic glands, lungs, and pleura. The tubercles may be scattered over the surface of the lung or may involve a small part of its parenchyma. Most frequently there develop the lesions of acute or subacute tuberculous bronchopneumonia, and less often those of miliary tuberculosis of the entire lung. There may be numerous small cavities; but the formation of large cavities, as seen in phthisis, is less common at this period. The bronchial glands are more or less enlarged and cheesy, and not infrequently suppurate. Sometimes only a few are affected; sometimes large masses of much enlarged glands are present. Involvement of the mesenteric glands is less frequent than that of the bronchial; that of the cervical glands not as common as later. Tuberculous meningitis is very frequent, seen oftenest in combination with other clinical manifestations, especially those of bronchopneumonia or of a general tuberculosis. Stirnimann[2] found the latter in 54.4 per cent. of 42 cases in 591 autopsies in the 1st year of life. Tuberculosis of the peritoneum, the intestine, and the bones is not frequent in infancy. At this period of life there is especially to be noted the tendency for tuberculosis to become wide-spread. Raczynski[1] states that in 611 cases of tuberculosis, as seen at autopsy, up to 3 years about 50 per cent. had a general tuberculosis.

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[1] Med. News, 1905, May 27.

[2] Jahrb. f. Kinderheilk., 1906, LXIII, 130.

In *early childhood*, from the 3d to the 5th year inclusive, general tuberculosis and meningitis remain very common, and involvement of the cervical and mesenteric glands, and of the intestine, peritoneum and bones increases in frequency. Tuberculosis of the lungs is, however, probably the most common form, with that of the pleura and bronchial glands nearly as often seen as this.

In *later childhood* tuberculous meningitis, although still frequent, is not so often observed as in earlier childhood. Cervical adenitis and involvement of the bones and joints remain common, while tuberculous peritonitis is increasingly often seen.

The kidneys, liver and spleen may show miliary tubercles on their surface at any age in cases of general miliary tuberculosis. The larynx is seldom involved. The pancreas, thyroid, thymus, heart, and genito-urinary tract are regions only exceptionally attacked in early life. In general, there is a predisposition at this age to involvement of the glands, while in adults the mucous membranes are oftener attacked.

Seat of the Primary Lesions.—This, of course, can be merely surmised in many instances. Reference is not made here to the original lesion at the portal of entry, often small and undiscovered, but to those lesions readily seen and having the appearance of having existed longest, and from which the extension of the process could be considered to have taken place. In 200 autopsies on tuberculous children, reported by Northrup[2] and by Bovaird,[3] this oldest lesion was in the respiratory tract, including the bronchial glands, in 148, and in the intestines and mesenteric glands in 3. In 119 autopsies on tuberculous children published by Holt[4] the oldest lesion was in no instance in the intestinal tract, and in 63 per cent. of the cases there was no intestinal involvement whatever. In 115 autopsies on tuberculous children in the Children's Hospital of Philadelphia, recorded by Hand,[5] apparently primary lesions of the intestine or mesenteric glands were found in 10. In 335 autopsies on children with tuberculous lesions reported by Hamburger[6] there was no certain case of primary intestinal or mesenteric tuberculosis. Albrecht[7] in 1060 autopsies showing tuberculosis in children found only 0.66 per cent. exhibiting undoubted primary intestinal tuberculosis. All these figures indicate the great preponderance of lesions in the respiratory tract as generally observed.

On the other hand, British statistics show somewhat different figures. J. W. Carr[8] found in 120 autopsies 79 with primary respiratory lesions, and 20 with primary intestinal or mesenteric glandular involvement; and Still[9] in 269 cases noted the primary localization as follows: Lungs 138; intestines 63; bones and joints 5; ear 15; unknown situation 46. The contrast between British and American experience is well illustrated in the collected statistics published by Bovaird[10] as follows:

[1] Jahrb. f. Kinderheilk., 1901, LIV, 67.
[2] New York Med. Jour., 1891, LIII, 201.
[3] New York Med. Jour., 1899, LXX, 1.
[4] Med. News, 1896, LXIX, 656.
[5] Arch. of Pediat., 1903, XX, 248.
[6] Wien. klin. Wochenschr., 1907, XX, 1069.
[7] Wien. klin. Wochenschr., 1909, XXII, 327.
[8] Lancet, 1894, I, 1177.
[9] Pediatrics, 1899, VIII, 335.
[10] Sixth Internat. Cong. Tuberc., 1908, II, 4, 451.

Table 90.—Intestinal Tuberculosis; English and American Statistics

	Cases	Primary intestinal	Per cent.
English	1161	236	20.33
American	620	21	3.39

In addition is to be mentioned the occasional occurrence of the primary localization in the tonsils or adenoids, Wood[1] finding tuberculosis in 5.2 per cent. of 1671 collected cases in which examination was made of the tonsillar tissue removed from individuals apparently free from the disease in other respects. Doubtless these organs are oftener the primary seat than has usually been supposed.

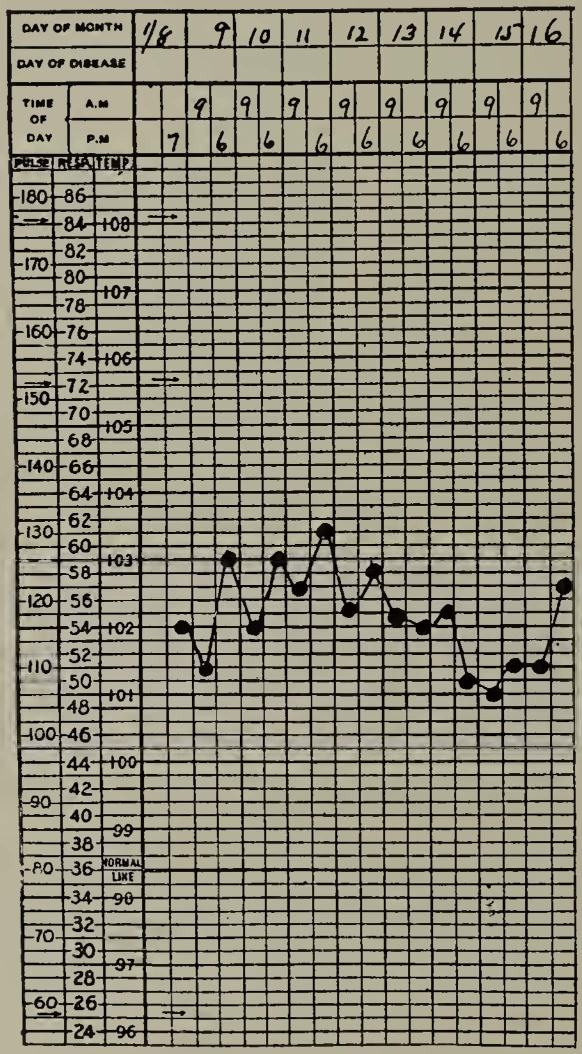

Fig. 178.—General Tuberculosis, Typhoid Form.

George D., aged 5½ years, colored. Jan. 8, in bed for a week with fever and debility. Is apathetic, weak, tongue coated. No physical signs in chest or abdomen. Typhoid fever suspected. Jan. 14, gradually increasing somnolence, slight, varying rigidity of limbs, leucocytosis 7410, negative Widal reaction; Jan. 16, undoubted slight signs of consolidation in right lung. Death. Autopsy showed general miliary tuberculosis.

Lesions Oftenest Producing Death.—It by no means follows that the parts oftenest affected are those the disease of which occasions the death of the patient. Thus, in infancy, although pulmonary involvement, usually in the form of bronchopneumonia, is the most frequent cause of death, in many pulmonary cases the fatal issue depends actually upon a general miliary tuberculosis, or, oftener, a tuberculous meningitis. Involvement of the bronchial lymphatic glands, although so frequent, rarely produces fatal symptoms. In early and later childhood meningitis, secondary to involvement of the lungs, bones, or lymph glands, very often occasions the fatal termination. Shennan[2] found this true in 44.5 per cent. of 413 cases of tuberculous disease of various sorts in children. Tuberculous peritonitis is also a not infrequent cause of death in later childhood, while tuberculous pleurisy, although common, is rarely in itself fatal.

Duration of Incubation.—A distinction is to be made here between the biological and the clinical period of incubation. The first is the time elapsing between a known infection of a previously tuberculosis-free child and the time when the tuberculin-test responds positively. This is somewhat variable, but averages for the cutaneous reaction from 4 to 6 weeks (Hamburger).[3] The determination of the length of clinical incubation is usually difficult and uncertain. It may extend from a few weeks to several years before symptoms of tuberculosis make their appearance.

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[1] Jour. Amer. Med. Assoc., 1905, XLIV, 1425.

[2] Sixth Internat. Cong. Tuberc., 1908, II, 4, 367.

[3] Pfaundler and Schlossmann's Handb. d. Kinderheilk., 1923, II, 608.

Clinical Forms of Tuberculosis. (*A*) GENERAL TUBERCULOSIS.—This form consists in a wide-spread development of tubercles secondary to some small caseous area. Through the entrance of large numbers of bacilli into the blood the disease is spread more or less widely throughout the body, the extent varying greatly with the case. Sometimes the various organs are crowded with tubercles, especially well seen in the lungs; sometimes these are much more scattered and fewer. The symptoms and the rapidity of the course of the disease vary accordingly. The liver is oftener affected in children than in adults. The tubercles are commonly larger in the child than in the adult and in the less rapid cases assume a considerable size through confluence, many of them becoming caseous and breaking down. This variety of the disease may exhibit tself in two forms, (1) the *Typhoid* and (2) the *Marantic.*

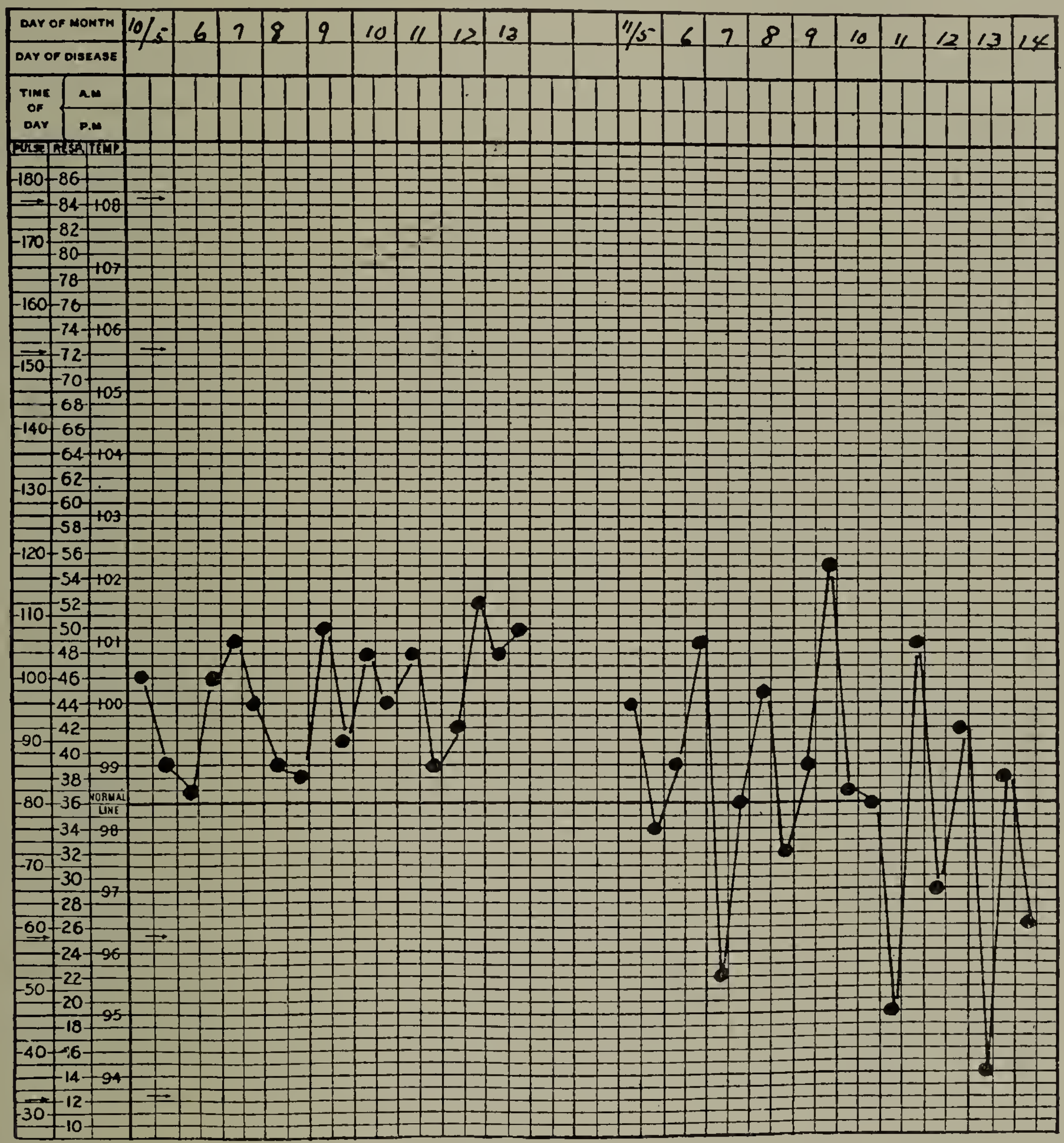

FIG. 179.—GENERAL TUBERCULOSIS, MARANTIC FORM. FINAL BRONCHOPNEUMONIA AND MILIARY TUBERCULOSIS.

Chas. McL., aged 1 year. Said to have been ill for 6 months. Been suffering latterly from fever, loss of weight, slight cough, poor appetite and loose stools. *Examination.*—Greatly emaciated; anemic; enlarged cervical, axillary, inguinal and abdominal glands; possible slight impairment of percussion-resonance, and harsh respiration; distended abdomen. Later positive evidence of pulmonary involvement. Had daily a moderate number of soft or liquid undigested stools. Death about 1 month later. *Autopsy.*—Tuberculous bronchopneumonia of left lung and root of right lung; miliary tuberculosis both lungs and of the spleen; tuberculous alteration of intestine; adenitis of mesenteric, retroperitoneal and bronchial glands.

1. The Typhoid Form; Acute Miliary Tuberculosis.—This is seen chiefly in infancy and early childhood. The initial symptoms are very vague, consisting of loss of appetite, debility, loss of weight, and other evidences of a general impairment of health. Fever of an irregular and uncharacteristic type soon develops; debility increases; the tongue becomes dry; the pulse and respiration are accelerated; there is apathy, dullness, sometimes delirium and finally coma. The tympanites and enlarged spleen may, with the continued fever and other symptoms, strongly suggest typhoid fever (Fig. 178). In other cases the temperature is almost too irregular for this disease, and often only slight or, exceptionally, not at all elevated. No cause for the symptoms can be found. Temporary improvement may occur, although the emaciation does not disappear nor the strength greatly increase. Finally some localizing symptoms may develop, especially those of pulmonary consolidation or of meningitis; less often of peritonitis. The course of the disease is rapid, death occurring usually in a few weeks. The differential *diagnosis* is extremely difficult. Suggestive of tuberculosis is the more irregular fever, the tendency to rapid respiration, the failure of the fever to lessen at the end of 3 or 4 weeks, and especially the persistent absence of the Widal reaction. Finally the development of localizing symptoms may make the diagnosis of tuberculosis almost certain; but this occurs generally only a few days before death.

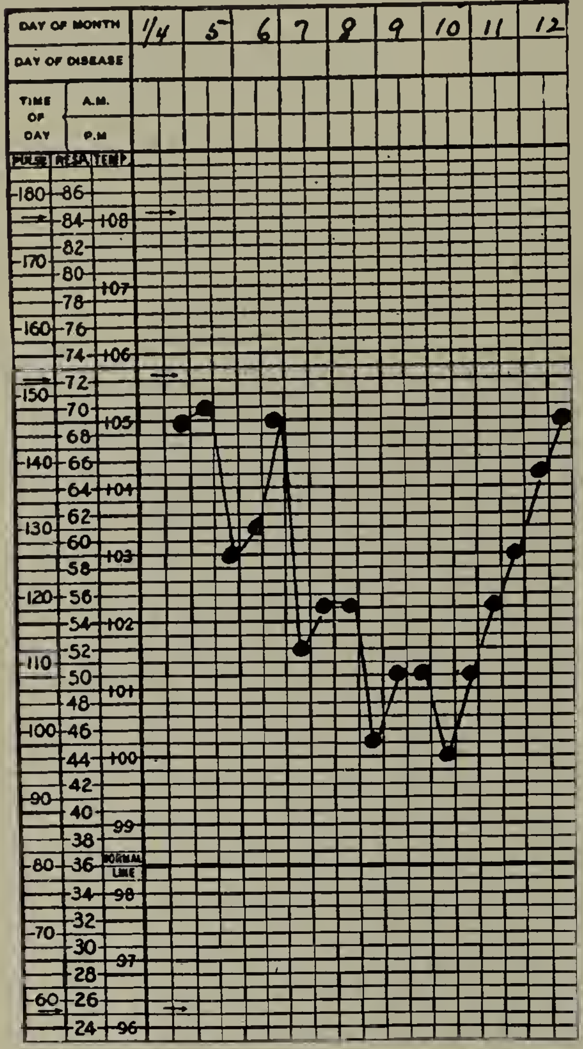

Fig. 180.—Acute Miliary Tuberculosis of the Lungs.

Bertha D., aged 3 years, admitted to Children's Hospital of Philadelphia, Jan. 4. About Dec. 15, developed cough and fever which continued. *Examination on admission.* Dyspnea, rapid respiration, bases of lungs filled with fine râles, and bronchophony heard. Child toxic; Jan. 11, been growing worse, convulsive movements, unconscious, harsh breathing and numerous scattered râles over both lungs in front and behind, no bronchial respiration or dullness on percussion; Jan. 12, death. *Autopsy.*—Lungs crowded with miliary tubercles throughout. Moderate enlargement of the tracheobronchial glands. Moderate development of tubercles on surface of liver and spleen and on the surface of the cerebrum.

2. The Marantic Form.—Infants not infrequently exhibit a somewhat more chronic form of tuberculosis, closely simulating marasmus. The symptoms are entirely uncharacteristic, progressive wasting and anemia being the principal ones. There is no fever, or occasional and irregular elevations, and no respiratory or gastrointestinal disturbances sufficient to account for the condition. The disease can in no way be distinguished from other marantic states, unless the case comes to autopsy. In other instances there develops a few weeks before the end of life more or less constant but moderate fever of irregular type, with slight symptoms suggesting bronchopneumonia (Fig. 179). The respiration is somewhat accelerated yet seldom decidedly dyspneic, there is slight cough, and the physical signs in the lungs are usually not well marked, or only those of bronchitis. Sometimes vomiting or, especially, diarrhea may become troublesome, or the symptoms of meningitis close the scene. Generally, however, death appears to be due to progressive exhaustion. The course of the case, although decidedly more prolonged than in the typhoid form, is brief; at longest a few weeks after continued fever or localizing symptoms appear, but often much longer from the first beginning of signs of illness. The *diagnosis* is always difficult and usually impossible, until, perhaps, shortly before death. Even the development of signs of bronchopneumonia is not conclusive, since non-tuberculous bronchopneumonia is so frequently a terminal condition in infantile atrophy. The principal diagnostic aid is to be sought in the history. The entire absence of discoverable reason for the continued wasting, the absence of evidences of chronic or repeated intestinal autointoxication, and the fact that diarrhea, cough, and vomiting have followed emaciation rather than preceded it, are reasons for suspecting tuberculosis. The tuberculin-test may be of value in some cases; in others it is negative on account of the overwhelming nature of the attack.

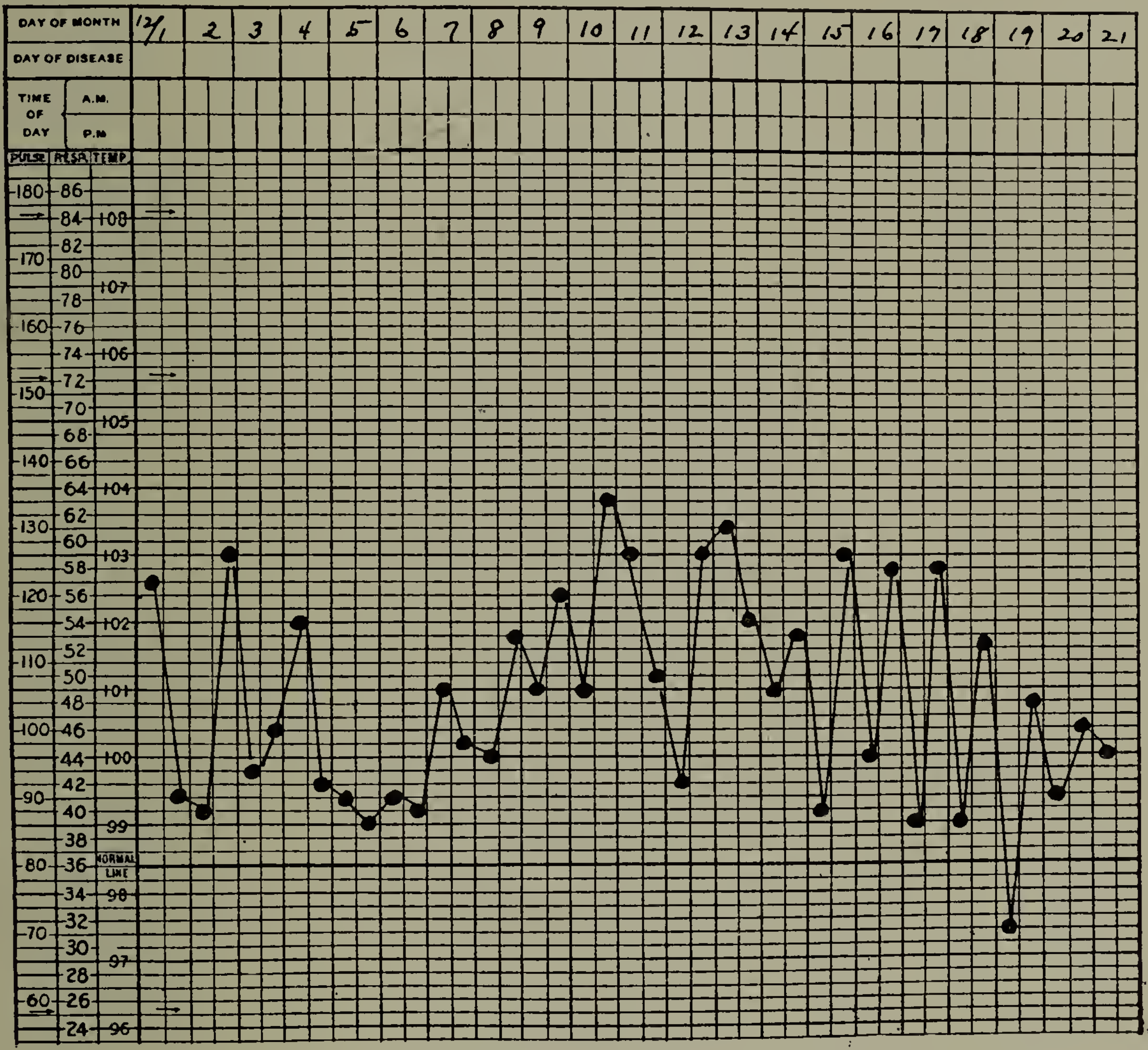

FIG. 181.—TUBERCULOUS BRONCHOPNEUMONIA.

Charles T., aged 1 year. Is said to have been ailing 6 weeks before seen. In the Children's Hospital nearly 1 month. Increasing impairment of percussion-resonance and of bronchial respiration over both lungs, with numerous râles. Increasing weakness and loss of weight, petechiæ, cough, anorexia, d'Espine's sign. Tubercle-bacilli found in sputum. *Autopsy.*—Bronchial and mesenteric glands much enlarged and tuberculous. Caseous pneumonia of both lungs. Cavity size of walnut in right lower lobe. Miliary tuberculosis of spleen and of pleura.

(*B*) TUBERCULOSIS OF SPECIAL REGIONS.—Here are to be included a large number of forms of tuberculosis in which the disease is confined to, or preponderates in, certain regions or organs of the body. Many of the conditions will be simply mentioned briefly, the fuller description of them being found elsewhere.

1. Tuberculosis of the Lungs.—Several varieties of this may be seen: (*a*) *Acute Miliary Tuberculosis of the Lungs* (Fig. 180).—This is the form of the general miliary tuberculosis described in which the localization is most prominent in the lungs from the onset. It is most common after the age of infancy. There is persistent fever of irregular but not hectic type; rapid respiration, which is sometimes dyspneic; prostration; rapid pulse; cough; and sometimes cyanosis. The physical signs in the lungs are often poorly marked and uncharacteristic. Later the evidences of tuberculosis elsewhere may show themselves, the child dying, possibly, from meningitis. The course of the case is short. At autopsy the lungs are found filled with miliary tubercles.

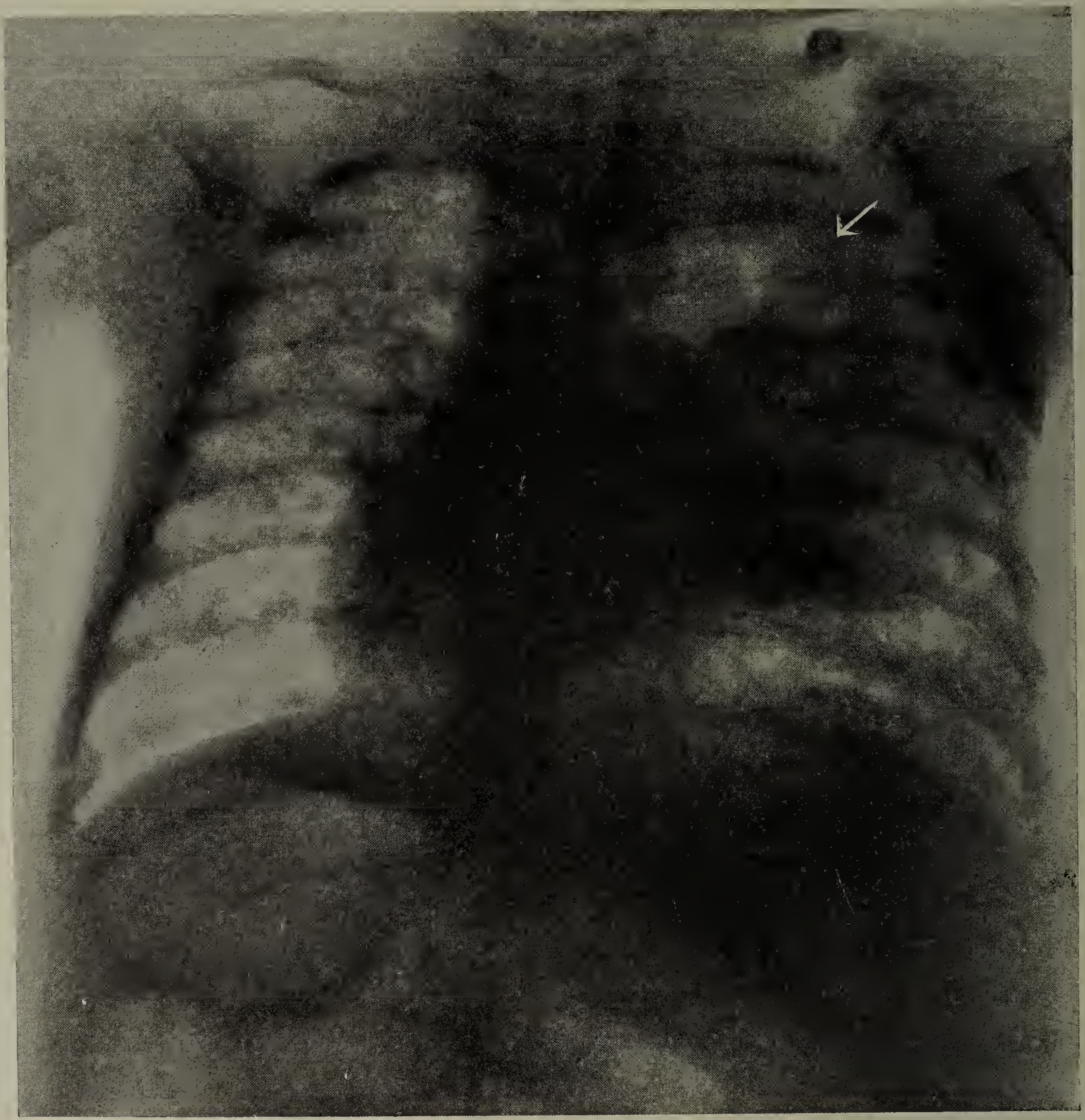

Fig. 182.—Radiograph of Acute Tuberculous Bronchopneumonia; with Cavity-formation.

Mary S.; colored; aged 10 months. More or less bronchitis since April. Pneumonia began 4 days before admission to the Children's Hospital of Philadelphia June 10. Physical signs of consolidation of upper part of right lobe suggested croupous pneumonia. In hospital 5 weeks. *X*-ray July 5th showed consolidation of right lung with cavity at the apex. Autopsy disclosed lesions of tuberculosis. Cavity about 2.5 cm. in diameter with ragged walls; tubercle-bacilli in smear from its contents.

(*b*) *Tuberculous Bronchitis.*—This is, strictly speaking, rather an obstinate bronchitis complicating and dependent upon the presence of tuberculous foci somewhere in the lungs. Sometimes it is the first stage of a tuberculous bronchopneumonia. In other instances it is of a chronic nature, and probably depends upon reflex or direct irritation from the tuberculous focus or its secretions. Sometimes the râles are scattered; oftener they are limited to a certain area upon one side. There are no other physical signs. The general symptoms are entirely uncharacteristic and those only of bronchitis, except in the cases which progress unfavorably, when fever finally develops and the condition assumes the characteristics of a chronic bronchopneumonia.

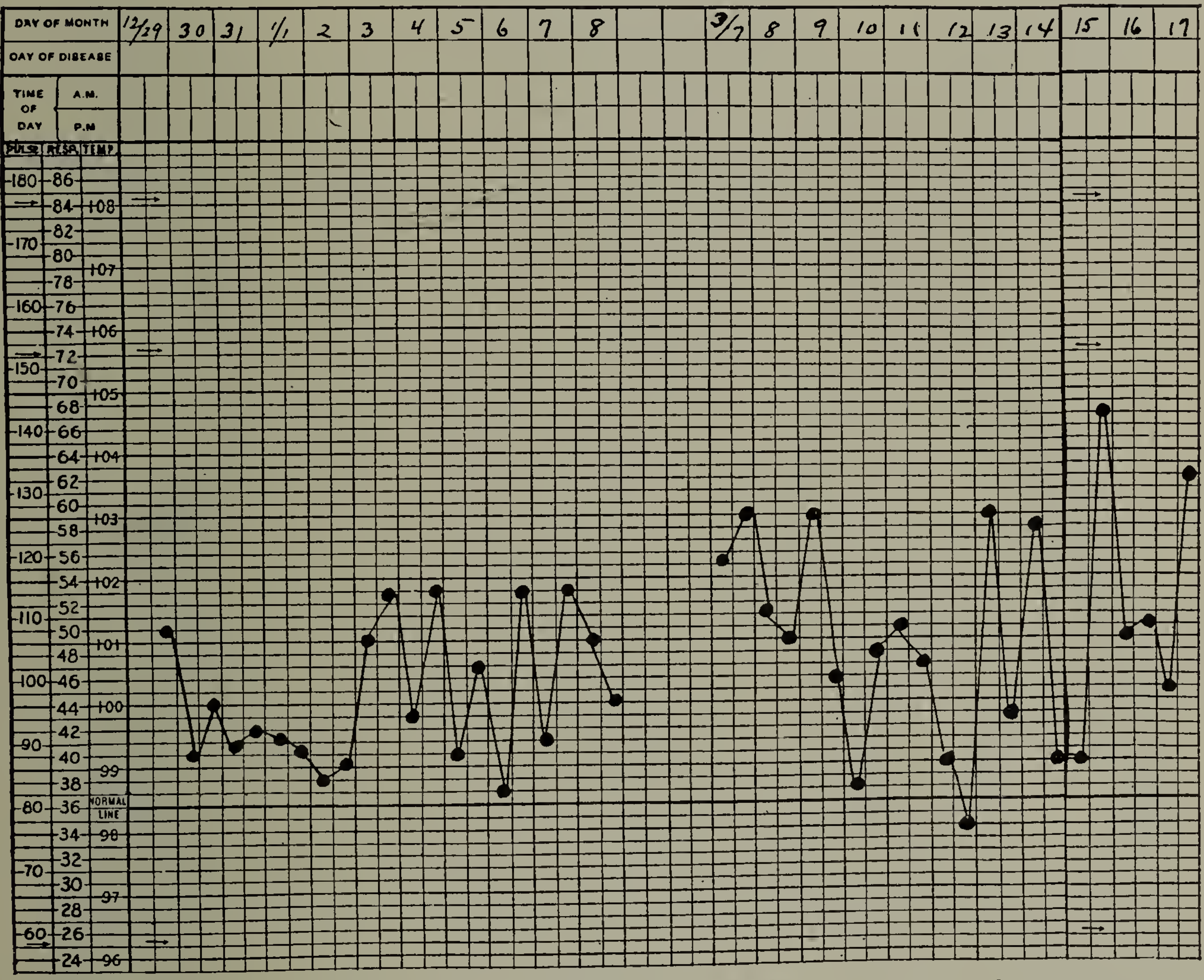

FIG. 183.—SUBACUTE TUBERCULOUS BRONCHOPNEUMONIA WITH CAVITY-FORMATION. Chart shows the first and last 11 days of observation in hospital

William K., aged 11 months when admitted to the Children's Hospital, Dec. 26, 1916. Died Mar. 18, 1917, aged 14 months Cough and debility since age of 3 months. While under observation considerable dullness on percussion throughout the right lung; many crackling râles and feeble breath-sounds, not bronchial. Before death there developed bronchial respiration over the upper part of the right lung; distinctly cavernous at the apex. *Autopsy* showed enlarged bronchial glands especially on the right side; the right pleura firmly adherent; right lung tuberculous throughout, a cavity the size of an orange occupying the whole of the upper lobe.

(c) *Acute Tuberculous Bronchopneumonia.*—This is one of the most frequent manifestations of tuberculosis in children. It is seen oftenest in infancy and especially, in early childhood; may be primary in the lungs or secondary to tuberculosis in some other part of the body, such as the bones, pleura, peritoneum, and particularly the bronchial lymphatic glands; or may follow some other disease, especially pertussis, grippe, measles, or even bronchitis or simple bronchopneumonia. The pathological lesions are the same as those of non-tuberculous bronchopneumonia, with the addition of the presence of tubercle-bacilli and the development of tubercles and of the degenerative changes which subsequently take place in these, and which result in the formation of many smaller and larger caseous areas and often finally of cavities (Fig. 182) if life continues a sufficient time; generally small and centrally located, but sometimes of considerable size. I have seen a cavity as large as an average orange, occupying the entire upper right lobe in a child of 7 months, and Crespin and Athias[1] report a cavity containing tubercle-bacilli in an infant of 20 days. Occasionally rupture into the pleural sac takes place. The large, slowly developing, encapsulated cavities characteristic of phthisis in the adult are usually absent in childhood until the age of puberty is approached. With the tuberculous process in the lungs is always associated similar disease of the bronchial lymph-nodes. The clinical picture does not differ materially from that of simple bronchopneumonia. (See Bronchopneumonia, Vol. II, p. 296.) Very frequently, however, the onset is more gradual, the principal early symptoms being cough, loss of weight, debility, increased rapidity of respiration and pulse-

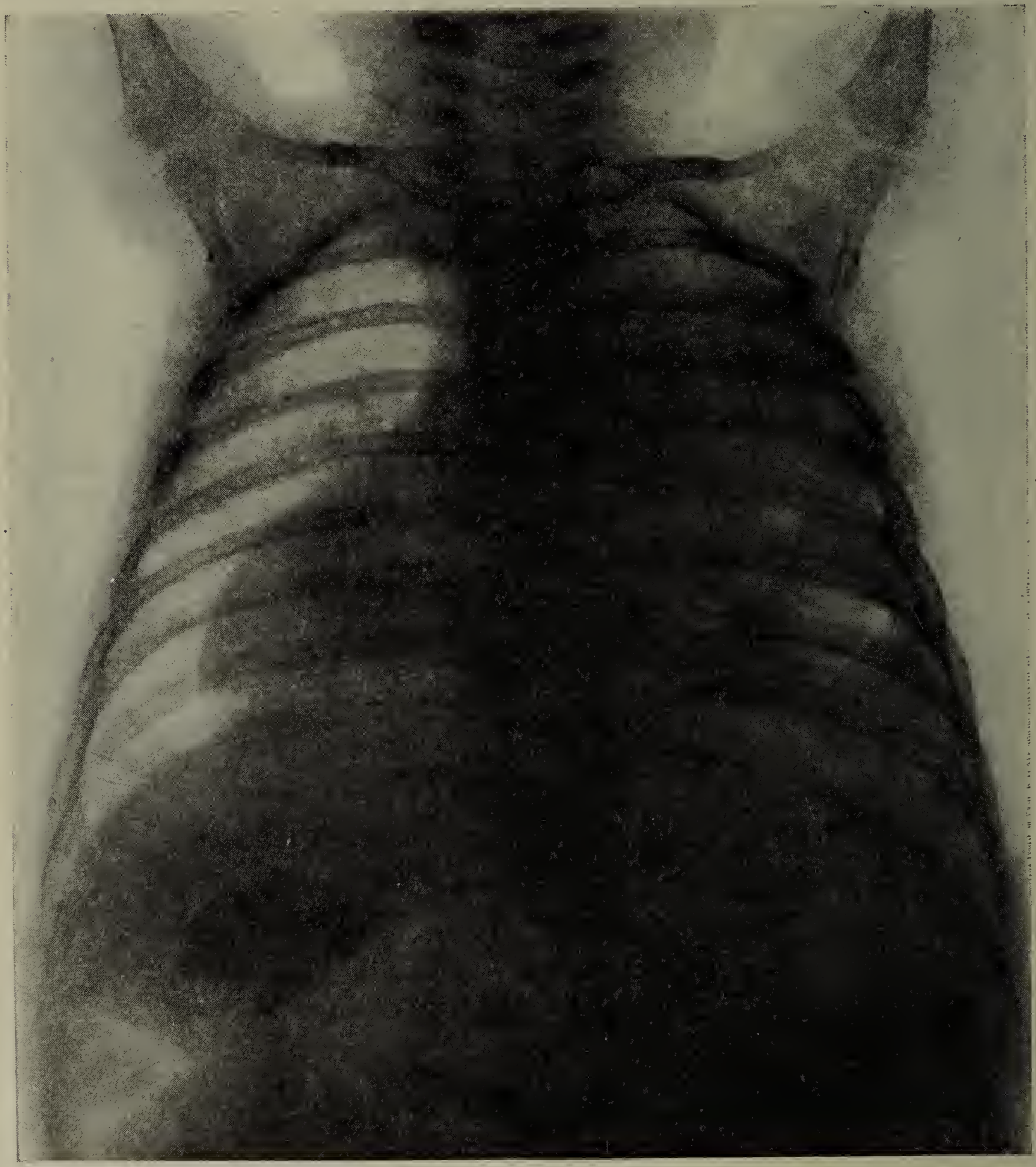

FIG. 184.—RADIOGRAPH OF TUBERCULOUS BRONCHOPNEUMONIA.

Same case as in Fig. 183. Shows advanced lesions of tuberculous bronchopneumonia in the right lung and to a less extent in the left. Viewed from behind.

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[1] Bull. soc. de pédiat., 1919, Jan.-June, 48.

rate, and moderate fever of an irregular type (Fig. 181). Later dyspnea becomes decided; the cough worse; the temperature higher, and there is cyanosis and increasing weakness. The physical signs are the same as those of simple bronchopneumonia, the lesions usually being scattered to a varying extent throughout both lungs, although most apparent in one. The pseudolobar form is less often seen. When localization occurs it is oftener in the upper lobe and toward the hilus than is the case with simple bronchopneumonia. Yet there is such variation possible in the symptoms and physical signs that the *diagnosis* of the tuberculous nature of the case is often impossible. Suggestive of tuberculosis is the development of bronchopneumonia during convalescence from measles or pertussis, or after a period of wasting and ill-health such as occurs in the general tuberculosis of infants; the discovery of tuberculosis elsewhere in the body; the failure of convalescence to begin at the time which may reasonably be expected in simple bronchopneumonia; and the consequently longer course, continuing perhaps for several weeks. Tubercle-bacilli may often be found in the secretion from the lungs (see p. 651) or the tuberculin-reaction may be obtained. In rare instances hemoptysis

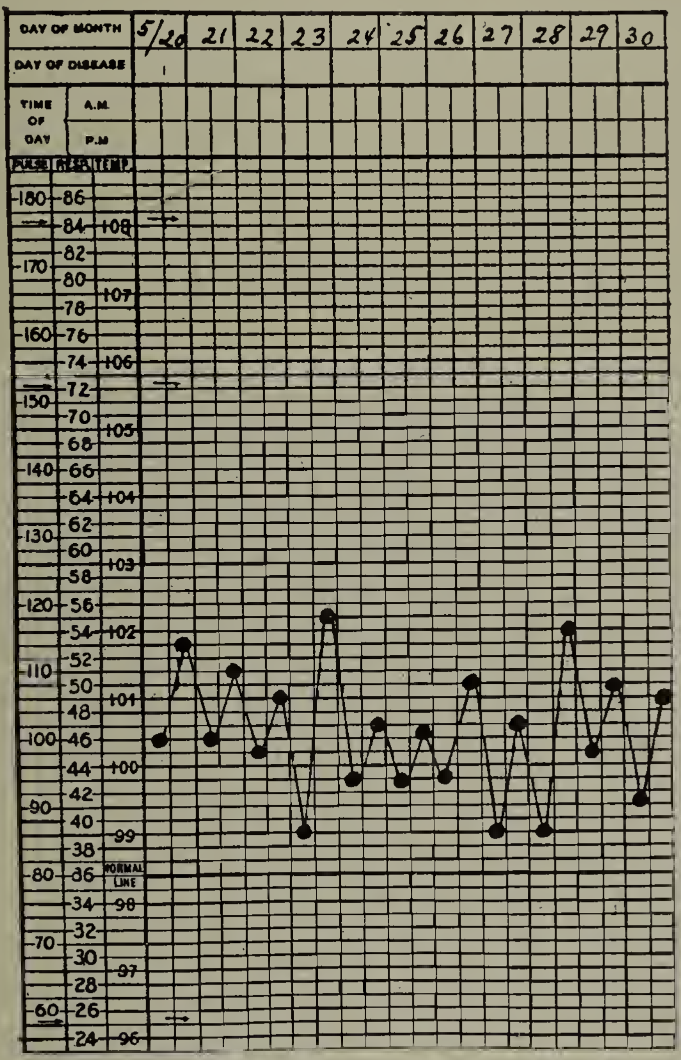

FIG. 185.—CHRONIC TUBERCULOUS BRONCHOPNEUMONIA.

George T., 2½ years old. Taken ill in summer of 1916 with general poor health. In the autumn symptoms of ileocolitis. Recovered from this, but never regained strength. Had dry cough, sometimes troublesome; accelerated pulse and respiration; irregular fever, the last certainly from December. First seen Apr. 4, 1917. Under observation was emaciated, good appetite, bowel-movements generally normal, slight cough, nasal obstruction with adenoids, respiration 30 to 40, fretful, debilitated, fever daily, positive Pirquet, no tubercle-bacilli found in secretion obtained from throat, tuberculide under chin, enlarged glands each side of neck. Repeated physical examinations of chest showed nothing abnormal, except deficient expansion; but x-ray examination (Fig. 186) revealed numerous scattered lesions. Went to seashore May 30, 1917, with condition unchanged, and died July 24.

may occur. I have seen a profuse hemorrhage fatal in a few minutes in an infant of 14 months.

(*d*) *Subacute and Chronic Pulmonary Tuberculosis.*—This may show itself in several forms in children.

(*α*) *Subacute and Chronic Tuberculous Bronchopneumonia* (Figs. 183–186).—The subacute type of this condition constitutes merely a protracted form of acute tuberculous bronchopneumonia and is directly continuous with it. It may last for some months and terminate fatally, or may, less often, pass into the chronic form. The chronic form may also develop without being preceded by any well-marked attack of bronchopneumonia. In some cases there may have been only attacks of what was supposed to be a bronchitis. Whatever the mode of onset, the recovery is not complete and the child is left with debility and cough. Repeated exacerbations occur at intervals and gradually decided physical signs of chronic bronchopneumonia develop, if not present previously. (See Chronic Pneumonia, Vol. II, p. 341.) Exceptionally the disease may begin insidiously, with wasting, continued fever, and more or less cough. The lesions may not be discoverable, or only unsatisfactorily so by physical examination; but the *x*-ray may show numerous scattered areas (Fig. 186). The case may continue for months and ter-

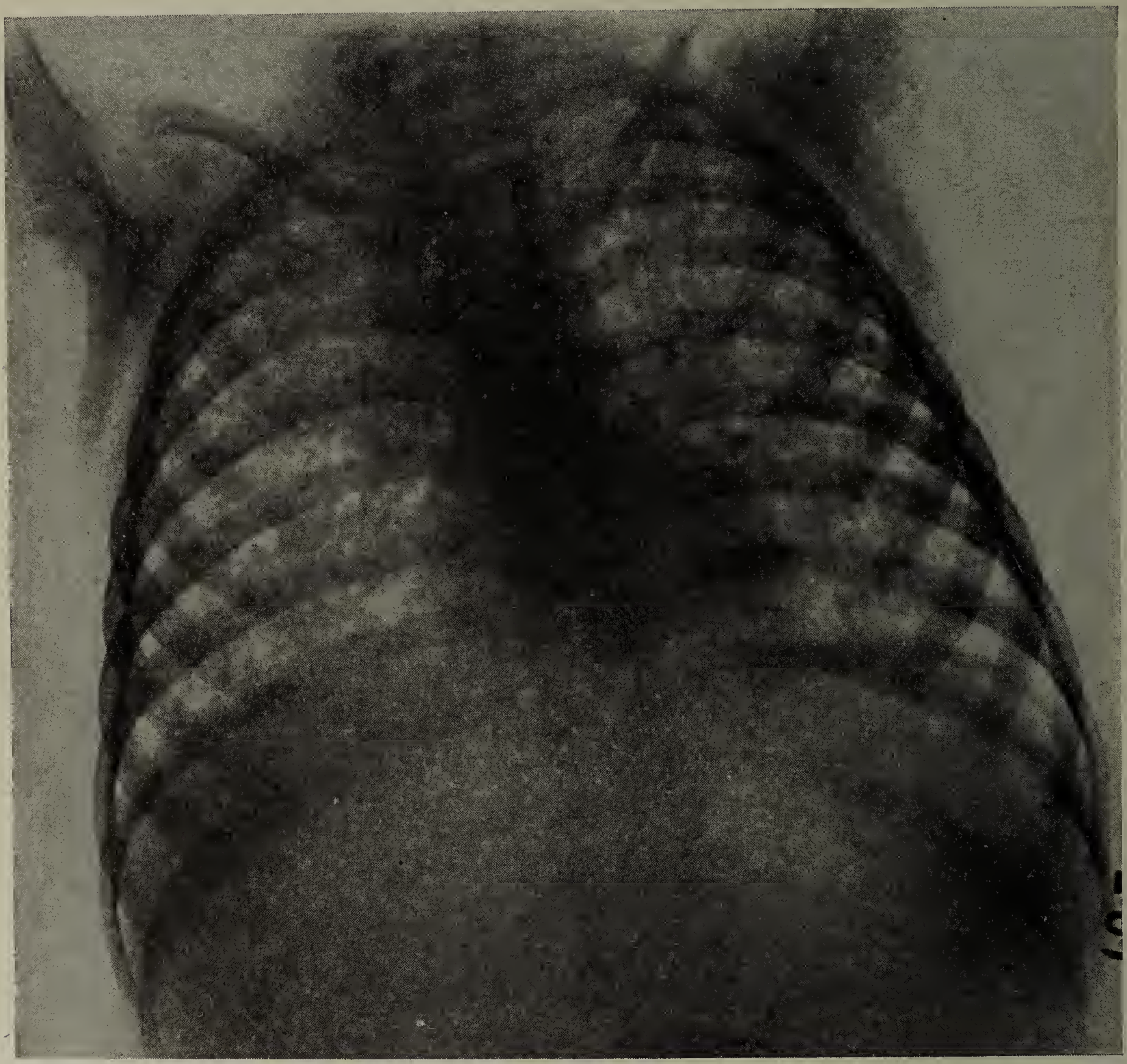

FIG. 186.—RADIOGRAPH OF CHRONIC TUBERCULOUS BRONCHOPNEUMONIA. Same case as FIG. 185. Shows wide-spread infilteration with numerous scattered lesions.

minate finally by exhaustion or through the development of some acute tuberculous process.

(β) *Hilus Tuberculosis.*—This is a pulmonary condition arising in connection with tuberculosis of the tracheobronchial glands, especially those adjacent to the hilus of the lung. It may develop at any period of early life, even infancy. The onset is insidious and the symptoms vague, consisting of malaise, diminished appetite, debility, and possibly evening rise of temperature. There is a positive tuberculin-reaction, but an absence of the ordinary physical signs. The involvement of the pulmonary tissue of the hilus cannot be distinguished by physical examination from that of the glands of the hilus which accompanies it. The x-ray examination may show fine lines radiating from the root of the lung (Fig. 187), indicating probably an involvement of the pulmonary parenchyma of the hilus-region (Stoll and Heublein).[1] The condition runs the chronic course of tuberculosis of the tracheo-bronchial glands.

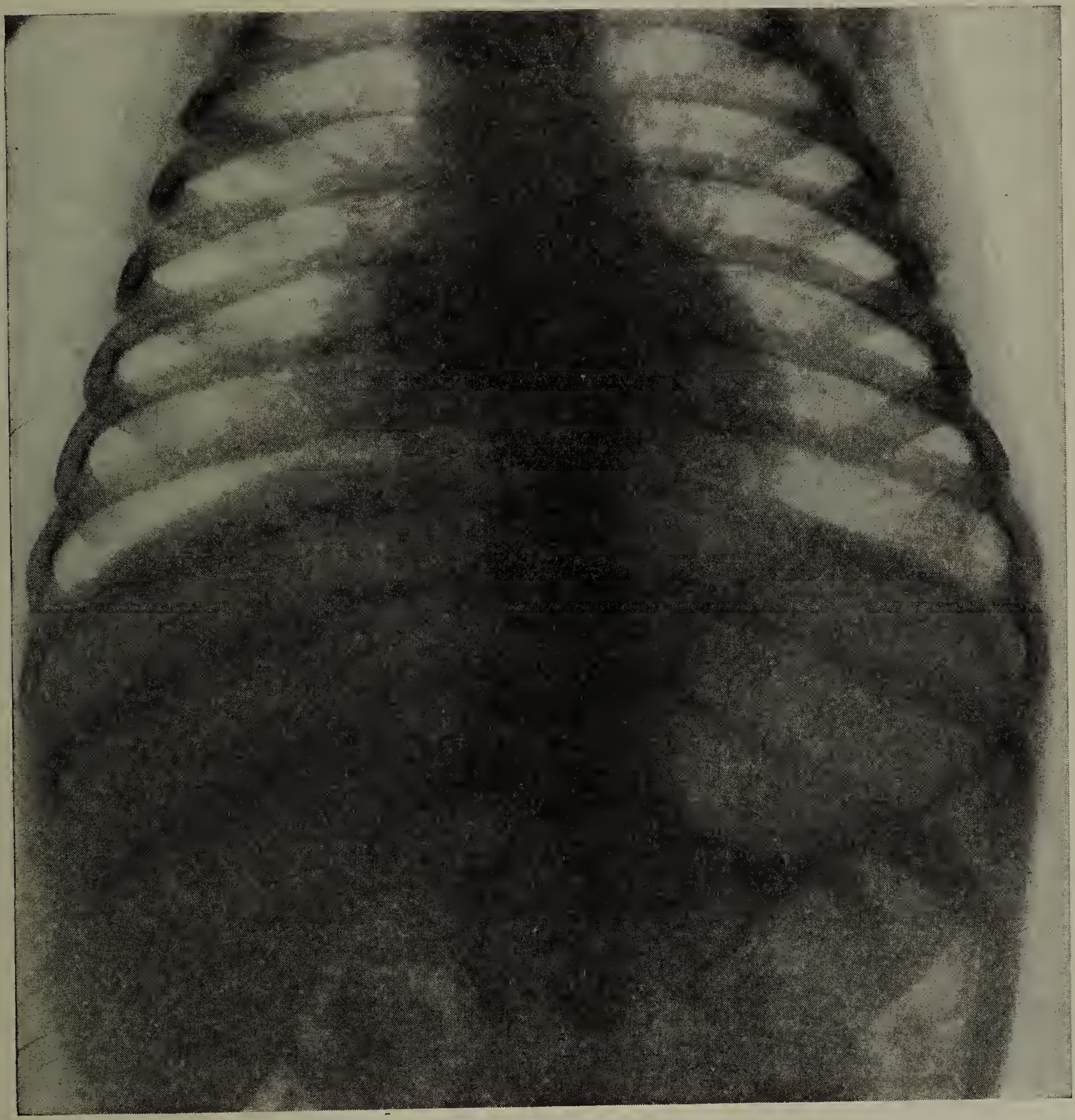

Fig. 187.—Radiograph of Tuberculosis of the Bronchial Glands and of the Hilus of the Lungs.

The ray-like shadows indicate the infiltration along the course of the bronchial tubes.

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[1] Amer. Jour. Med. Sci., 1914, CXLVIII, 382.

(γ) *Primary Pulmonary Foci of Ghon* (See also p. 632).—The name of Ghon[1] is commonly associated with these lesions, although they were earlier described by others. They consist of small, round foci from that of a millet seed to that of a hazelnut or even larger in dimensions (Albrecht),[2] but oftenest of the size of a pea. They are generally single or but few in number and situated in different parts of one or both lungs. The lesions represent the seat of primary pulmonary involvement. Any portion of the lung may be attacked, although there is a somewhat greater disposition to involve the upper lobe. They may occur at any period of early life, but with diminishing frequency as infancy is past. The lesions may become caseous, calcified, or shrunken, and may continue for an indefinite time without the production of symptoms, although the neighboring lymphatic glands are always secondarily involved. The condition in this form is of importance only as indicating the primary source of the extension of tuberculosis which later sometimes takes place.

It is probable, however, that following another course, and with increasing frequency in proportion to the youth of the patient, the foci instead of becoming dormant, increase in size and remain moderately active, the child exhibiting irregular or no fever, debility, loss of weight and a chronic cough; physical signs being absent except perhaps some scattered râles. The tuberculin-reaction is positive and in some cases tubercle-bacilli may be found in the sputum. The lesions at autopsy consist of the few foci described, which perhaps have become broken down and formed cavities; the combined involvement of the tracheo-bronchial glands; and the evidences of fresh tuberculous processes in the neighborhood of the foci or elsewhere. The prognosis in these more active cases is uncertain. The condition is liable to eventuate in an acute tuberculous bronchopneumonia or a more wide-spread tuberculosis, but especially in tuberculous meningitis.

(δ) *Phthisis.*—This is the form of the disease so common in adult life. It is rarely found in infancy and early childhood, and even in later childhood it is very much less frequent than after this period. In our experience it occurs much oftener in children of the colored race. Statistics vary considerably, but it is interesting to note that Sawyer[3] in a physical examination of 8000 children under 15 years of age found only 15 in which a diagnosis of phthisis could be made with reasonable certainty. Its symptoms, physical signs and treatment are much the same as those of phthisis in adult life. Only to be noted here is the lesser frequency of cough, involvement of the larynx, dyspnea, and of hemoptysis. The disease is to be distinguished chiefly from chronic bronchiectasis, and from those cases of chronic simple bronchopneumonia in which the occurrence of thickening about a large bronchus strongly suggests the presence of a cavity. Abscess of the lung, too, may closely simulate it. The obtaining of the tubercle-bacilli from the sputum or the presence of the tuberculin-reaction may settle the diagnosis. The infrequency with which cavity-formation takes place in the cases in children denominated phthisis by many observers raises the question whether, strictly speaking, many of those included in statistics should not be considered a chronic disseminated tuberculous bronchopneumonia, rather than phthisis as it occurs in adult life. No reference is intended here to the rapid production of cavity so frequent in acute tuberculous bronchopneumonia.

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[1] Der primäre Lungenherd bei der Tuberkulose der Kinder, 1912.
[2] Wien. klin. Wochenschr., 1909, XXII, 327.
[3] Brit. Jour. Child. Dis., 1909, VI, 205.

2. Tuberculosis of the Lymphatic Glands. (*Scrofula, Tabes Mesenterica, etc.*).—The involvement of the lymph-nodes is one of the most common forms of tuberculosis in early life, the frequency of its occurrence being much greater than in adults. That of the internal glands is often combined with evident tuberculosis of other parts, the symptoms of which give the clinical evidences of tuberculous infection.

(*a*) *Tuberculosis of the Tracheo-bronchial Lymphatic Glands* (Fig. 187). These glands include those about the trachea and the main bronchi and those following the major subdivisions of the bronchi at and beyond the hilus of the lungs. The process may be distinctly secondary to lesions in the lungs; or apparently primary, although, in reality, dependent upon some small undiscovered pulmonary tuberculous focus. There are, as a rule, few if any clinical manifestations, and the condition is recognized only post-mortem. This is especially true of infancy, but even after this period the clinical picture is generally indefinite. In some cases distinct evidences of pressure or of rupture of a suppurating gland become manifest. The symptoms will be discussed under Adenitis. (See Vol. II, p. 816.) There must be emphasized here the need of precaution in making a diagnosis of tuberculosis of the tracheo-bronchial glands, based solely upon the existence of x-ray shadows in the neighborhood of the hilus.

(*b*) *Tuberculosis of the Mesenteric Glands* (*Tabes Mesenterica*).—This is a common localization of tuberculosis in children, although, as already stated, much less so than is that in the bronchial glands. It may be combined with tuberculosis of the intestine, or less frequently occur without evident lesions there. As a rule there are no clinical manifestations apart from those of the other portions of the body, especially the intestine. The symptoms will be discussed under Adenitis. (See Vol. II, p. 818.)

(*c*) *Tuberculosis of the Cervical Glands.*—Tuberculosis of the external glands is exhibited in the cervical group with much the greatest frequency. It is especially common in early and later childhood, less so in infancy. The group of symptoms often associated with and frequently preceding it has for many years been described under the title "Scrofulosis," and the term is still employed in many text-books. The adenitis is usually unaccompanied by discoverable evidences of tuberculosis of the bones, lungs, or other viscera. The portal of entry is probably the tonsils, adenoid growths and chronic lesions of the pharynx (see p. 632); or there may occur much less often an extension of infection from the bronchial glands. In 106 cases in which the tonsils were removed for tuberculosis of the cervical glands, Mitchell[1] records infection of the tonsils in 41. Deterioration of the general health or attacks of acute infectious diseases, especially measles and pertussis, act as predisposing causes. The bacilli may be either human or bovine in type. (See p. 631.) The process may extend to the axillary and sometimes to other external glands. The bronchial lymph-nodes have been found involved in most of the cases coming to autopsy. Apart from the glandular affection, symptoms called "scrofulous" are described. The subject will be further discussed under Diseases of the Lymphatic Glands. (See Vol. II, p. 815.) Here only may be said that since it is certain that the involvement of the glands in "scrofula" is invariably tuberculous, and very possible that the other local conditions are in reality tuberculous lesions also, the retaining of the word in the nomenclature of diseases appears to be unnecessary and a source of confusion.

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[1] Jour. Pathol. and Bacteriol., 1916–17, XXI, 248.

(*d*) *General Tuberculous Adenitis.*—This condition without other clinical manifestations is of unusual occurrence at any period of life. In it one set of glands after the other may be attacked without other discoverable lesions. The disorder is to be distinguished from Hodgkin's disease. It has been especially studied by Lesage and Pascal.[1]

3. Tuberculosis of the Alimentary Tract.—The disease, as stated, not infrequently attacks the tonsils and adenoid tissue and spreads thence to the cervical glands. White[2] estimates the presence of tuberculosis in 5 per cent. of adenoids, and Weller[3] in 8679 cases of tonsillectomy observed tuberculosis of the tonsils in 2.35 per cent. The stomach is but rarely the seat of tuberculosis. The most frequent localization of alimentary infection in children is in the intestine, where the disease assumes the form of ileocolitis. (See Intestinal Ulceration, Vol. II, p. 172.) This is not common in early life after the age of infancy. In the majority of cases it is secondary to involvement of the lungs, and is usually associated with tuberculosis of the mesenteric glands. The small intestine is the region oftenest involved, the colon is sometimes secondarily so, and occasionally the cecum is the only part attacked. The diagnosis from other forms of ileocolitis depends chiefly upon the discovery of tubercle-bacilli in the stools or of tuberculosis elsewhere in the body.

4. Tuberculosis of the Genito-urinary Tract.—While miliary tuberculosis of the kidney is frequent in cases of general tuberculosis, clinical manifestations of tuberculous renal disease are rare in children. Tuberculosis of the testis is occasionally seen and more rarely of the female genitals. That of the penis has followed ritual circumcision. Of this occurrence Reuben[4] has collected 42 reported instances.

5. Tuberculosis of the Nervous System.—Apart from involvement of the meninges, the nervous system is not often attacked. Large, solitary tubercles are sometimes found in the brain or spinal cord. These may become cheesy or even calcareous, and may give rise to localizing symptoms, as in the case of tumors of other nature.

6. Tuberculosis of Serous Membranes.—This may occur wide-spread as one of the manifestations of general tuberculosis, or may predominate in, or be confined to, certain of the serous membranes, especially the meninges, the peritoneum, and the pleura. Meningitis is very common and a frequent cause of death. It is nearly invariably secondary to tuberculosis elsewhere, although often without symptoms except those of meningitis. It is most frequent in later infancy and early childhood. The clinical manifestations will be described under Meningitis (Vol. II, p. 615). In infancy involvement of the peritoneum is usually only one of the manifestations of a more general tuberculosis. After this period tuberculosis may be limited to the peritoneum, or the involvement of this be at least the chief cause of the symptoms observed. It may be apparently primary, the lesion at the portal of entry not being discovered; or much oftener secondary to tuberculosis elsewhere, as in the mesenteric glands or, less often, the lungs or other regions. (See Peritonitis, Vol. II, p. 235.) Tuberculous pleurisy with serous or purulent exudate is seen in later childhood; less often before this. Without effusion it is of common occurrence as one of the symptoms of general tuberculosis, or as an attendant upon tuberculosis of the lungs.

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[1] Arch. gén. de méd., 1893, CLXXI, 1, 270.
[2] Amer. Jour. med. Sci., 1907, CXXXIV, 228.
[3] Arch. Int. Med., 1921, XXVII, 631.
[4] Arch. of Pediat., 1917, XXXIV, 186.

Tuberculosis of the bones and joints and of the skin will be discussed under the disorders of those regions.

Prognosis.—Tuberculosis is frequently stated to be the cause of about 1/7 of all deaths. The census of the United States published in 1900 (Wilbur)[1] gives tuberculosis as the cause of death in 11.22 per cent. of the total mortality, and unquestionably the actual proportion was decidedly larger than this. On the other hand, a large number of cases which show tuberculosis at autopsy have died of other affections. The general mortality from tuberculosis has without doubt diminished in recent years. This is not so much because the patients recover more readily, although this has certainly resulted from improved methods of treatment, as because preventive measures have diminished the number of cases developing. It is noteworthy, however, as demonstrated by Behla[2] for Prussia, that the diminution in the number of deaths from tuberculosis in children has not kept pace with that for later periods of life; and the same was shown by Hoffmann[3] to be true to some extent for the Registration Area of the United States. The mortality from the disease was largely increased in parts of Europe during and after the World War. (See p. 627.)

The prognosis of tuberculosis in children varies with the age and with the form of the disease. In the 1st year there appears to be very little power of resistance, the disorder assumes an active form from the beginning, and the great majority of those infected die. Yet certain investigations, as by Pollak,[4] Osswald[5] and others, indicate that the prognosis is not so absolutely bad as has been generally believed. In proportion as age advances the power of resistance becomes greater and there is less likelihood of the affection producing clinical manifestations, and more chance of an arrest, permanent or temporary, of developed lesions. In the 2nd year the prognosis for tuberculous infection in general is much better, and still better after this period.

With regard to the disease already clinically manifested, the prognosis depends, too, in a large degree, upon the form of this. The subject is discussed more fully elsewhere under the proper headings. The primary lesion of Ghon tends to recover without further extension, except in the 1st year. Its presence is, however, always a menace, and there may at any time occur a dissemination of bacilli and the development of acute clinical manifestations. Miliary tuberculosis is probably always rapidly fatal. Tuberculous bronchopneumonia is practically always fatal either in the first attack or in the relapses which are likely to occur. If it assumes the chronic form it may last sometimes for several years, usually only to end fatally at last through an exacerbation of an acute bronchopneumonia, or through the development of tuberculosis elsewhere. Tuberculous bronchitis may terminate in a fatal bronchopneumonia; but, on the other hand, it may last for months and then disappear with the healing of the tuberculous focus on which it depended. Hilus-tuberculosis runs a chronic course and frequently ends in recovery. Only isolated cases of recovery from tuberculous meningitis have been reported, and these may have been but temporary arrests of the disease. (See Tuberculous Meningitis, Vol. II, p. 334.) On the other hand, tuberculous peritonitis frequently terminates favorably, with or without operation; tuberculous pleurisy usually recovers; osseous tuberculosis is often cured; that involving the bronchial lymphatic glands is in itself seldom a cause of death, and tuberculosis of the cervical glands is usually not a condition which menaces life. The prognostic indications of the tuberculin-reaction will be referred to later (p. 653).

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[1] New York Med. Jour., 1908, LXXXVIII, 798.
[2] Berl. klin. Wochenschr., 1913, L, 1951.
[3] Jour. of the Outdoor Life, 1913, X, 361.
[4] Beitr. z. klin. d. Tuberc., 1911, XIX, 373.
[5] Monatsschr. f. Kinderheilk., 1922, XXIII, 168.

Diagnosis.—Apart from the manifestations pertaining to, and the characteristics of, special localizations of tuberculosis, there are certain features which are suggestive from a diagnostic point of view. Careful attention should be given to the history of tuberculosis in the family as bearing upon the possibility of the inheritance of a diminished resistance to the disease, or still more of peculiar opportunity for the acquiring of the affection by association with tuberculous relatives. The existence of unfavorable hygienic conditions is to be investigated, such as render the subjects more susceptible or occasion unusually free contact with the germs. The earlier occurrence of other diseases which may predispose is also to be considered. Especially, in the case of pulmonary tuberculosis, are to be suspected previous attacks of pertussis, measles, pneumonia, bronchitis, and grippe. A generally defective state of health of the child, and the existence of the physique which is known to be associated with tuberculosis, are also of moment.

In the case of active tuberculosis of an obscure nature, the occurence of fever is important. In the large majority of cases some elevation of temperature is present at times. It is generally of an irregular type and often only slight; and is, of course, not conclusive, since so many other conditions are capable of producing it, especially in early life. In spite of this fact, the continual repetition of frequent rises of temperature, other causes having been sought for and excluded as far as possible, is an extremely suspicious circumstance. It is a matter of common occurrence, as I know from my own experience, to consider a general tuberculosis to be typhoid fever, until some distinct localizing process finally develops; and I recall 1 instance in which two eminent physicians had respectively made the diagnosis, the one of endocarditis and the other of chronic intestinal indigestion and toxemia, and which finally eventuated in a tuberculous peritonitis. Many analogous cases could easily be detailed.

A persistent increasing impairment of the general health also arouses suspicion, although not necessarily present in many forms of tuberculosis; nor of certain indication, since it may depend upon so many other factors. Chronic cough is of diagnostic value to a limited extent. It may depend upon many different causes, but tuberculosis of the lungs or pleura is always to be thought of. When paroxysmal and stenotic in character and associated with dyspnea, it may indicate tuberculosis of the tracheobronchial glands. Hemoptysis is so uncommon in early life that it need scarcely be considered as a possible diagnostic sign. Pleurisy with a serous effusion is always suggestive of tuberculosis when occurring in childhood. The discovery of nodular masses in the abdominal cavity points, in the case of children, more to tuberculosis than to any other condition, and ascites is very probably tuberculous, if cardiac and renal diseases can be excluded.

The examination of the blood is of little diagnostic value. Slight increase in the number of leucocytes is sometimes present, the degree varying with the form of the disease. The only exception seems to be tuberculous meningitis, in which there is not infrequently a decided leucocytosis. In acute pulmonary cases the absence of a high polymorphonuclear leucocytosis tends to exclude pneumonia of other than tuberculous or influenzal origin. The calcium in the blood is said by some investigators to be low during the active stage of the disease, but this is denied by others.

The *x*-ray examination is of very great value in some forms of tuberculosis, notably when it affects the bones and joints, tracheo-bronchial glands, lungs, and pleura. It must be admitted, however, that the pictures are sometimes misleading, and that the results of the examination must be regarded as confirmatory and the diagnosis must be in accord with other physical signs and symptoms. Recent published experience from various sources emphasizes the caution necessary in determining that shadows in the region of the hilus are of a tuberculous nature. The general opinion appears to be that the existence of complement-fixation in tuberculosis is not sufficiently certain to be depended upon, and that the same is true of the agglutinative and precipitin reactions often present. Cooke,[1] however, who has performed about 2000 complement-fixation tests in children, believes that the test is of value. The Wilbolz[2] auto-urine reaction for active tuberculosis has been widely tested. The opinions of investigators are divided; but the preponderance of evidence appears to be that the reaction is caused by the urinary salts rather than by antigen, and that it is unreliable, giving sometimes a positive reaction in entirely healthy children, or in those with an inactive tuberculous process, while those certainly tuberculous may at times respond negatively. The test is performed as follows: The patient's urine is evaporated over a water bath to $\frac{1}{10}$ of its volume and filtered through paper saturated with a 2 per cent. phenol solution. Minute amounts are then injected intradermally. Infiltration and redness and occasionally necrosis develop in 48 hours at the site of the injection in positive reactions.

When the disease is of a nature which permits of the discovery of tubercle-bacilli, the diagnosis is, of course, made certain. It is important, however, to be absolutely sure that the bacilli really are those of tuberculosis. They may be found in the large majority of instances of tuberculous meningitis and in many cases of tuberculosis of the lungs. Inasmuch as young children do not expectorate, special measures must be employed to obtain the sputum. For this purpose a swab of absorbent cotton or of muslin, firmly attached to a curved applicator, may be applied to the region of the glottis, and the sputum which the consequent coughing brings up can be caught upon this. Bacilli may also not infrequently be found in the stools, either in cases of intestinal tuberculosis or when the sputum has been swallowed in pulmonary disease.

The Tuberculin-reaction.—This may be obtained in various ways: (1) Tuberculin may be injected *subcutaneously* in an initial dosage of not over 0.1 milligram (.0015 grain) and the occurrence of a constitutional reaction with rise of temperature noted. This test is given the preference by some investigators because it furnishes a greatly increased percentage of positive reactions as compared with other methods. For example, where Pirquet with the cutaneous method found an incidence of tuberculosis of only 56 per cent. in children from 11 to 14 years of age,

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[1] Amer. Jour. Dis. Child., 1921, XXI, 78.

[2] Correspondbl. f. Schweiz. Aerzte, 1919, XLIX, 793.

Hamburger[1] found 95 per cent. by using the puncture method. The procedure is valueless when fever is already present, and is not devoid of the danger of rendering active a condition which is latent. Hamburger[2] details a method of employment of the "puncture" (Stich) reaction in a way to avoid the production of general symptoms. The patient is first tested by the cutaneous or percutaneous method. In cases where this gives a negative result, a subcutaneous injection of 0.01 mg. (.00015 grain) is administered not more than 48 hours later. With still further negative results repeated injections with increasing doses are given at 24 to 48 hour intervals until a dosage of 1.0 mg. (.015 grain) is reached. A positive reaction consists in the development of definite redness and tenderness at the seat of the puncture. (2) The *intracutaneous* test (Mantoux)[3] is obtained by injecting 0.1 c.c. (1.6 minim) of a 1:1000 solution of old-tuberculin, equivalent 0.1 mg, into the superficial layers of the skin. If this dose produces no local reaction it may be increased even up to 0.5 mg. (.0075 gr.). Active tuberculosis will almost certainly give a reaction to a dose of 0.1 mg. (.0015 gr.) and usually of 0.01 mg. (.0015 gr.). Mensi[4] after testing different diagnostic methods in 3889 children, concluded that the intracutaneous is that to be preferred. The resulting appearances in positive cases are very similar to those seen in the cutaneous test. (3) The *cutaneous* reaction of Pirquet[5] presently to be described. (4) The *percutaneous* test, devised by Moro,[6] consists in vigorous rubbing, lasting 1 minute, into the sound skin of a 50 per cent. mixture of tuberculin and lanolin. In 24 to 48 hours red papules appear varying in number with the intensity of the reaction. The test is rendered much more accurate if the cutaneous surface-fat is first removed by rubbing with ether. (5) The *ophthalmo*-reaction of Wolff-Eisner[7] and Calmette[8] is obtained by dropping into the eye 1 drop (0.062) of a 0.5 per cent. aqueous solution of old-tuberculin. The reaction varies from a slight reddening up to decided conjunctivitis. Owing to the damage to the eye sometimes produced, the test has with reason lost favor.

The greater convenience and the effectiveness of the cutaneous test makes it easily the choice of methods, at least for the first trial, although the intracutaneous method will give a larger number of positive reactions. It is performed by washing the skin of the upper arm with ether or alcohol, applying a drop of undiluted old-tuberculin, and then scarifying slightly through this with a sterilized needle or the special drill-shaped scarificator made for the purpose. A control scarification should be made a couple of inches distant for the purpose of comparison. A positive reaction develops within 24 hours, sometimes longer, and consists of a red, slightly indurated maculo-papule 5 mm. (0.2 inch) or more in diameter (Fig. 188). It reaches its height usually upon the 2d day and fades slowly, often leaving slight scaliness. A papule of smaller size than 5 mm. (0.2 in.) is not to be considered certainly positive (Pirquet).[9] If the reaction obtained is doubtful or negative, a second trial may be made in a few days or, still better, the puncture test or the intracutaneous test may be used.

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[1] Amer. Jour. Dis. Child., 1922, XXIII, 481.
[2] *Loc. cit.*
[3] Compt. rend. de l'acad. d. scien., 1908, CXLVII, 355.
[4] Arch. de méd. des enf., 1923, XXVI, 65.
[5] Wien. med. Wochenschr., 1907, LVII, 1370.
[6] Wien. klin. Wochenschr., 1907, XX, 933.
[7] Berl. klin. Wochenschr., 1907, XLIV, 7
[8] Comp. rend. acad. sci., 1907, CXLIV, 1324.
[9] Feer's Lehrb. der Kinderheilk., 1914, 677.

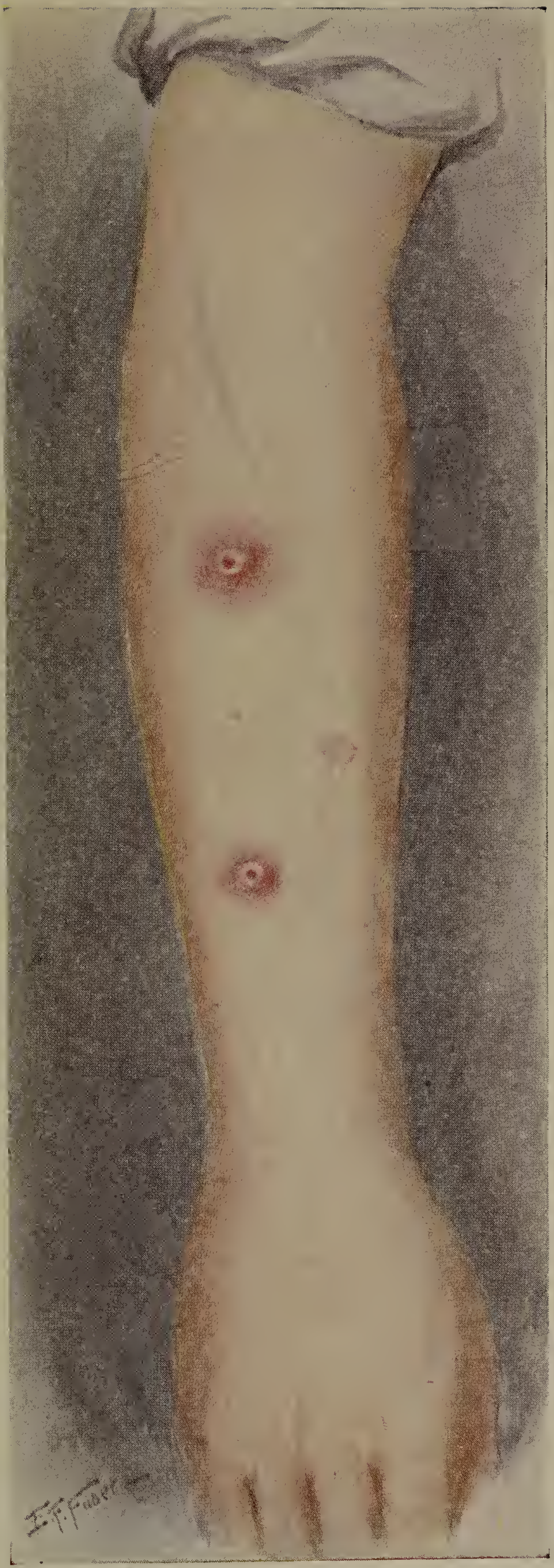

Fig. 188.—The Pirquet Cutaneous Tuberculin Reaction.

Child with incipient pulmonary tuberculosis; a + reaction. The control scarification is barely to be seen, and is midway between the tuberculin-reactions. (*Kolmer, A Practical Text-book of Infection, Immunity and Specific Therapy*, 1917.)

Value of the Tuberculin Reaction.—Under ordinary circumstances it is fair to assume that a negative tuberculin-reaction, especially if it is still so on the second trial, excludes the presence of tuberculous infection in the patient. In patients gravely ill in an advanced stage of the disease, the organism may be so unable to produce antibodies that no reaction takes place. Cases of general miliary tuberculosis and of tuberculous meningitis may also fail to respond to the test. There is often, too, observed an inhibitory action of some of the acute infectious diseases, notably measles, and sometimes pertussis, grippe, pneumonia and scarlet fever, the tuberculin-reaction failing to develop during their course, although positive before or after this.

A positive reaction indicates that the patient has a tuberculous infection, and that antibodies exist in the blood. The lesion may be small, old, healed, and entirely unimportant, and this should always be borne in mind in forming a diagnosis of the nature of the symptoms present. The bacilli in the lesion may be encapsulated and remain alive, but the reaction occurs even if they are dead. The lack of value for diagnostic and prognostic purposes increases in proportion as the patient is older. In infancy the finding of a positive tuberculin-reaction generally indicates an active process, and that very probably the symptoms observed are closely connected with this. This is especially true in the 1st year of life. It is then of serious import, although not a certain proof that the disease is advancing and that the issue will be fatal. The great frequency of the positive reaction in older children, to which reference has already been made (p. 626), shows how guarded one must be in attributing too much importance to it, so far as association with the existing symptoms is concerned. One of the most markedly positive tuberculin-reactions we have seen was in a 10 year old child, at whose autopsy there was no macroscopic or microscopic evidence of tuberculosis discovered.

In this connection it is interesting to consider the susceptibility to the tuberculin-reaction of those who exhibit the human or the bovine type of bacillus respectively. Although a small percentage of individuals will react with the tuberculin of only one of the two types of the bacillus, the large majority of those who give a positive reaction with one type will do so with the other also. (See contributions by Ramsey,[1] Cattaneo,[2] and DeLange.[3]) Downing and Higgins[4] believe that in the first stages of the disease the patient may react to but one type, presumably the infecting one.

In closing the subject of the diagnosis of tuberculosis in early life, it is important again to urge caution, especially in the case of children of the school-age. The numbers of statistical studies on large series of school-children show so great a range in the percentage-results, that the accuracy of the diagnosis based on the physical signs at this time of life becomes a matter of question. Reference is made here especially to the cases of children in good or very fair health, in whom slight physical signs, oftenest apical, are found. It is probable, as has been pointed out by Zimmermann,[5] Grosser,[6] Czerny,[7] Hamburger[8] and others, that the

[1] Amer. Jour. Dis. Child., 1915, X, 201.

[2] Zeitschr. f. Kinderheilk., Orig., 1913, VI, 506.

[3] Nederl. Tijdschr. v. Geneesk., 1914, LVIII, 2, 438

[4] Amer. Jour. Dis. Child., 1926, XXXI, 178.

[5] Zeitschr. f. Kinderheilk., 1922, XXXII, 15.

[6] Deutsch. med. Wochenschr., 1920, XLVI, 367.

[7] Zeitschr. f. aertzliche Fortbild., 1919, XVI, 545.

[8] Pfaundler and Schlossmann's Handb. d. Kinderheilk., 1923, II, 638

diagnosis of tuberculosis based upon such conditions is usually incorrect. An actual tuberculous lesion sufficient to produce the physical signs is almost always in childhood attended by very positive symptoms. It has already been pointed out that the recognition of tuberculosis of the hilus by *x*-ray examination is to be made very cautiously, and that even apparent lesions in other parts of the lungs, found by this method, may not be a positive proof of the existence of the disease; and, further, that a positive tuberculin-reaction is no guarantee that any symptoms or signs present are dependent upon tuberculosis.

Treatment. Prophylaxis.—The great need is that the infant be guarded against every possible contact with tuberculous subjects, even though these be the parents. No nurse with tuberculosis, healed or otherwise, should be employed. Dwellings should be thoroughly disinfected if the slightest suspicion exists of the presence of bacilli in them. The ingestion of milk from cows with tuberculosis is to be avoided, and when it cannot be made certain by veterinary inspection that the milk is above suspicion, it should invariably be pasteurized. Even this must be done thoroughly and at home, since Austin[1] found 7 instances of the presence of bovine bacilli in commercially pasteurized milk. The putting of toys and the like into the mouth of the infant must be prevented as far as possible, and the greatest cleanliness exercised in the care of its fingernails, and of the spoons, cups, rubber nipples, and other articles used in the giving of food. Promiscuous kissing of the baby by visitors is unwise, since virulent tubercle-bacilli are frequently present in the mouths of tuberculous individuals (Gloyne).[2] Association of an infant in its 1st year with an individual suffering from cough may be dangerous. In fact, during this period the baby is better from every point of view if kept from such contact with other individuals. When for any reason, as among the poor, it is impossible entirely to prevent contact of the infant with members of the household suffering from pulmonary tuberculosis, at least the greatest care in the collection and destruction of the sputum can be urged. The resisting power of the child must be maintained by abundant, suitable nourishment, life in the open air, plenty of sunshine, gymnastic exercises, cool bathing, suitable clothing and other hygienic measures. In this line is a temporary sojourn at the seashore or mountains if signs of debility become manifest. There is also great need in the case of delicate children for the prompt treatment of bronchitis, the removal of hypertrophied tonsillar tissue, and the avoidance of contact with pertussis, grippe and measles. In the production of immunity by tuberculin nothing of real value has as yet been accomplished. The employment of vaccines may perhaps later become of service both as a preventive and as a curative measure. von Ruck and Flack[3] have vaccinated 3000 children against tuberculosis since 1903, using von Ruck's extractive of tubercle-bacilli. None of the subjects had developed active tuberculosis up to the time of the report. Calmette and his co-workers[4] summarize their work on prevention by means of the ingestion of attenuated bovine bacilli. Apparently many infants so treated remained clinically free from the disease and often tuberculin-negative, even after definite exposure to their tuberculous parents. Selter[5] injected the attenuated bacilli subcutaneously. Raw[1] treated over 400 children with injections of dead attenuated cultures of bovine bacilli. None of the children had later developed tuberculosis although exposed to it. Other encouraging studies are found in the literature. It must be said, however, that the possibility of preventing tuberculosis by the employment of dead bacilli has been denied, and that the ingestion or injection of living attenuated germs is not yet past the experimental stage. In fact no measures of prevention by any specific method have as yet won general acceptance.

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[1] Amer. Jour. Dis. Child., 1919, XVII, 264.

[2] Tubercle, 1922, III, 497.

[3] Med. Rec., 1921, XCIX, 1048.

[4] Presse méd., 1925, XXXIII, 8251; Bull. de l'acad. de méd. Paris, 1926, XCV, 175.

[5] Deutsch. med. Wochenschr., 1925, LI, 1181.

Treatment of the Attack.—This depends so greatly upon the localization and on other factors, that it can be considered, for the most part, only in the separate sections treating of the various forms of the disease. During the acute febrile stage the patient should be much of the time at rest, but this should be, if possible, in the open air. Here may be mentioned also the benefit in all forms of tuberculosis to be derived from abundant nourishment, especially milk in some form, and from change of climate, particularly to elevated, dry regions, although the seacoast is of value in many cases. Treatment in sanatoria for children, situated in a favorable climate, is often efficacious. Even where change of climate cannot be obtained, efforts can be made to ensure a life largely in the open air, as by the making use in chronic cases of open-air schools, and the like, or of similar methods employed at home (Knopf;[2] Michael[3]). The employment of tuberculin of various sorts as a curative agent, of which so much was once hoped, has not obtained any settled place in the treatment of the disease, especially in children. It is but fair to state that it is highly recommended by physicians of experience; as by Wittich,[4] Schlossmann,[5] Beck[6] Solis-Cohen[7] Much[8] and many others. There would appear to be always danger of increasing the activity of the process, or of awakening it if it has been dormant. The initial dose recommended should be very small; not over 0.001 milligram (0.00015 grain) or even less. It should not be used except by those of experience. Among drugs probably the most useful is cod-liver oil, alone or combined with iodide of iron, creosote, or sometimes arsenic. If it is not well tolerated and diminishes the appetite, it should be abandoned. There is no evidence that cod liver oil exerts a specific action in tuberculosis. That the administration of calcium causes healing of tuberculous lesions has no experimental foundation (Maver and Wells,[9] Smith[10]) Czerny and Eliasberg[11] have employed parenteral protein-therapy in the form of hypodermic injections of horse-serum and think the results excellent. The treatment of tuberculosis by injection of gold salts (Sanocrysin) as recommended by Mollgaard[12] has yet to prove its value and lack of danger.

Heliotherapy has a very definite place in the treatment of tuberculosis, and may be carried out in the home or in any hospital, special equipment and location not being necessary. It is particularly valuable in cutaneous, glandular, osseous and abdominal tuberculosis, but has also beneficial effect in the pulmonary form. Heliotherapy has been extensively employed by Rollier[1] in Europe, and apparently first introduced in the United States at Perrysburg, N. Y., (Hyde and Lo Grasso.)[2] Certain precautions should be observed in its employment. The patient is first accustomed to the open air. Exposure to the sun should stop ½ hour before meals, and should not be begun within 2 hours after. If high fever, rapid pulse or nausea are caused, the treatment should be stopped or diminished. During the hot part of the day in summer treatment should be suspended. The head and eyes should be protected. The method of gradual exposure is outlined elsewhere. (Vol. I, p. 281.) Other forms of radiotherapy such as the *x*-ray, radium and the quartz lamp have yielded good results especially in glandular tuberculosis.

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[1] Brit. Med. Jour., 1924, II, 102.
[2] Med. Rec., 1913, LXXXIV, 875.
[3] Review in Amer. Jour. Dis. Child., 1916, XI, 162.
[4] Jahrb. f. Kinderheilk., 1912, LXXV, 166.
[5] Deutsch. med. Wochenschr., 1909, XXXV, 289.
[6] Zeitschr. f. Kinderheilk., Orig., 1913, VI, 439.
[7] Arch. of Pediat., 1920, XXXVII, 641.
[8] Tuberculosis of Children, Macmillan Co., 1921.
[9] Amer. Rev. Tub., 1923, VII, 1.
[10] Amer. Rev. Tub., 1923, VII, 33.
[11] Monatsschr. f. Kinderheilk., 1920, XVIII, 1.
[12] Chemotherapy of Tuberculosis, 1924; Ref. Jour. Amer. Med. Assoc., 1925, LXXXIV, 516.

CHAPTER XXII

SYPHILIS

This affection, the origin of which is unknown, but which in all probability existed in ancient times, became pandemic in Europe toward the end of the 15th century. In early life it is represented by 2 forms, (*A*) Acquired Syphilis; (*B*) Hereditary Syphilis.

Frequency.—The incidence of the disease is very great. It is true that Fruhinsholz[3] recorded it in but 186 (1.07 per cent.) of 17,282 children under the age of 12 years, only 12 of these being proven to be acquired syphilis; and Still[4] found hereditary syphilis in but 29 (0.6 per cent.) of 4830 children under 10 years of age. The statistics of Neumann and Oberwarth[5] on 69,221 children and of Cassel[6] upon 17,448 infants give a percentage very close to that of Fruhinsholz. If the diagnosis based solely upon the existence of a positive Wassermann reaction be included with those determined by clinical examination, the proportion of instances of hereditary syphilis is increased decidedly, ranging from 2 to 14 per cent. according to the various statistics collected by Churchill and Austin.[7] Studies made in the United States are interesting. Royster[8] found a positive Wassermann reaction in 15.47 per cent. of 659 colored children and in 7.04 per cent. of 341 white children. Jeans and Cooke,[9] basing this upon positive Wassermann reactions, estimated that in St. Louis 1.8 per cent. of the children of the poor whites, less than 1 per cent. of those of the well-to-do, and 15 per cent. of those of the colored population had hereditary syphilis. Williams[10] in a study of 4000 pregnant women, about 50 per cent. of whom were white and the other half colored,

[1] Die Heliotherapie der Tuberculose.
[2] New York Med. Jour., 1917, CV, 11.
[3] Rev. d'hyg. et de méd. inf., 1903, II, 1.
[4] Pediatrics, 1904, XVI, 577.
[5] Arch. f. Kinderheilk., 1905, XLII, 64.
[6] Arch. f. Kinderheilk., 1909, L, 154.
[7] Amer. Jour. Dis. Child., 1916, XII, 355.
[8] Amer. Jour. Syph., 1921, V, 131.
[9] Amer. Jour. Dis. Child., 1921, XXII, 402.
[10] Johns Hopk. Hosp. Bull., 1920, XXXI, 335.

obtained positive Wassermann reactions in 2.48 per cent. of the white women and in 16.29 per cent. of the colored. In addition 105 women with negative reactions showed clinical evidence of syphilis. Of 392 fetal deaths occurring in this series (99 white and 203 colored infants), 104 were due to syphilis. Yet these figures by no means represent the actual number of cases, since they take no account of the numerous syphilitic infants who are born dead, prematurely or at term, or who die soon after birth, to say nothing of the very large number of abortions due to this disease.

Germ.—Of both forms the cause is the same: evidently a germ of some sort, since the disease is eminently infectious. Various germs have been described, prominent among them being the bacillus of Lustgarten,[1] and the *cytoryctes luis* described by Siegel.[2] The researches of Schaudinn and Hoffmann[3] confirmed by many others, showed that the cause is a spirillum, the *spirochœta pallida*, or *treponema pallidum*, which is found in the primary lesions and mucous patches, and in hereditary cases in the various tissues and secretions of the body also, especially the liver.

The etiology in general, the pathological anatomy, and the symptoms of the two varieties of the disease will be considered separately.

(*A*) ACQUIRED SYPHILIS

Etiology.—This form is much more uncommon than the hereditary variety in early life, and yet not infrequently encountered. Infection takes place as in adults, except that it is usually by other paths than the genital tract. Kissing by a syphilitic mother or wet-nurse; nursing from a breast which is the seat of a syphilitic lesion; contact with infected clothing, drinking cups, nursing bottles, and the like, are the most frequent methods. Whether the disease can be transmitted by the milk itself of a syphilitic wet-nurse is doubtful. It has been communicated by vaccination in the days when human virus was employed, and from the mouth or instruments of a syphilitic operator performing ritual circumcision. Infection from the genitals of the mother during birth is possible but very infrequent. Profeta's law—that an apparently healthy child certainly will not acquire syphilis through kisses given by its syphilitic mother, or through nursing from or other contact with her—indicates only that the infant has acquired a certain degree of apparent immunity, because it is, in fact, already really syphilitic. Yet in view of recent observations the constancy of the law is in question. It seems quite possible that in some instances infants may escape infection, even though their mothers are in the active stage of syphilis during pregnancy, (Carle;[4] Cooke and Jeans.[5]). Fischl and Steinert[6] report on 17 healthy infants born of syphilitic mothers. These infants never developed signs of syphilis and their Wassermann reactions were uniformly negative, although taken from 6 to 12 times.

Pathological Anatomy and Symptoms.—These do not differ materially from those characteristic of adult life, age appearing to have no influence. There is an initial lesion at the point of infection, followed in due course by the usual secondary and finally tertiary manifestations.

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[1] Wien. med. Wochenschr., 1884, XXXIV, 1389.
[2] Münch. med. Wochenschr., 1905, LII, 1321.
[3] Arbeiten aus d. kais. Gesundheitsamte, 1904–5, XXII, 527.
[4] Presse méd., 1920, XXVIII, 244.
[5] Amer. Jour. Syph., 1922, V, 569.
[6] Arch. f. Kinderheilk., 1921, LXIX, 399.

The attack is usually milder than in adults, and less severe than hereditary syphilis; wide-spread cutaneous eruption is not uncommon, but is more macular than papular; and there is a marked disposition in children to the development of moist condylomata.

(*B*) HEREDITARY SYPHILIS

Etiology.—Of all infectious diseases this is the most frequently inherited. It may be transmitted by either parent or by both, but in the large majority of cases the father is the original source of the disease. The mother, already syphilitic at the time of conception, may infect the fetus, the father being healthy. Much less often the mother may acquire syphilis at some period during pregnancy, and then transmit it to the fetus (post-conceptional syphilis). Whether the syphilitic father can communicate it to the fetus without infection of the mother has been disputed. It would seem to be of common occurrence, if only clinical evidence of maternal syphilis is considered; and this is doubtless because the mother is herself a subject of the disease in a latent form, either at the time of parturition or later in life. Yet Colles[1] law, that the mother of a syphilitic infant will not become infected by contact with it, even though it have lesions in the mouth, shows that she has in some way become immune; although she has been without symptoms. The investigations of Knöpfelmacher and Lehndorff[2] with Wassermann's complement-fixation test, confirmed by many others, show that such mothers do, indeed, give the characteristic reaction for syphilis in a large proportion of cases; but the fact that they may have a negative Wassermann reaction does not mean that they are free from syphilis. If this point of view be accepted, then there is no such thing as the infection of the ovum by spore-carrying semen. On the contrary, the virus infects the mother, the placenta becomes syphilitic, and the organisms are thus allowed to pass through it to the blood of the fetus. Transmission of the disease to the fetus is, therefore, always from the mother.

Whether or not the infant of distinctly syphilitic parents will be born syphilitic depends upon various factors. When both parents are syphilitic the offspring will almost always be infected. The stage of the disease is important. If secondary symptoms are present in either parent, transmission is almost certain, but to this there are exceptions. If the symptoms in the parents have been overcome by treatment before the time of conception, or if they are tertiary, transmission will probably not occur, but to this also there are numerous exceptions. In general, the more recent the disease in either parent, the more certain is it likely to appear in the child and the more severe are the symptoms. As regards post-conceptional syphilis, the shorter the time between infection and parturition, the less liable is the fetus to be syphilitic. In early post-conceptional infection of the mother active treatment may prevent the transmission of syphilis to the fetus. There is no positive proof of the transmission of the disease to the third generation, and examination of the published reports of cases does not satisfactorily exclude the possibility of the disease having been acquired in the second generation.

It has been denied that hereditary syphilis is contagious, and it seems certain that the transmission of the disease by infants with this form of the affection is uncommon. Nevertheless, it is equally certain that such transmission can at times occur and syphilis be acquired by other children of the family, through kissing the affected infant, or through contact with contaminated spoons, cups, toys and the like. The spirochete can, indeed, be found in the lesions. Werther[1] records 10 cases in which the nurses were infected by the infant.

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[1] Pract. Observ. on Vener. Dis. and on the Use of Mercury, London, 1837, 304.

[2] Monatsschr. f. Kinderheilk., 1909, VIII, 34.

Pathological Anatomy.—In cases of early abortion there are often no determinate microscopical anatomical changes whatever found in the fetus. In 62 such cases examined by Hecker[2] gross lesions were discovered in only 15; *i.e.* 24 per cent., and lesions shown only by the microscope in an equal number. On the other hand, in the fetus approaching full term, and in infants dying a few days after birth, the changes are very characteristic. In children not showing symptoms of syphilis at birth, but dying of it after a few weeks or months, it often happens that few, if any, pathological changes of the internal organs are discovered. The longer the time after birth before symptoms appear the less liable are the internal organs to be found diseased. The basis of the early alterations is a diffuse cellular proliferation arising from the perivascular connective tissue with involvement of the vessels. With this there is an interference with the development of the organs. Only later is there a tendency to the production of isolated gummata.

The most constantly present and definite pathological changes are those of the osseous system, especially the long tubular bones and the ribs. In the fetus and in young infants the lesion is usually an osteochondritis. Acquired syphilis does not exhibit this alteration. It occurs oftenest in the epiphyses of the long bones of the legs and arms (epiphysitis). There is a widening of the cartilaginous layer through multiplication of the cells, with hardening due to abnormal calcification and, later, a softening from cellular infiltration, exuberant granulation-tissue, and necrosis with consecutive separation of the epiphysis. Sometimes suppuration occurs as a result of a mixed infection. Dactylitis is a lesion sometimes observed, especially in early childhood. In later childhood osteochondritis is not often seen, but there is found an osteoperiostitis, particularly of the skull and of the bones of the forearms and legs. This may be diffuse and hyperplastic, or it may be represented by isolated gummata, especially on the cranium. Involvement of the joints also occurs in the form of synovitis or chondroarthritis.

Enlargement of the spleen is nearly always present in the fetus or the new-born. It exhibits merely hyperplasia but nothing characteristic. At a later period is observed an enlargement dependent upon interstitial changes or, less frequently, gummata. Changes in the liver are seen in the fetus and the new-born about as frequently as those of the spleen. The organ is enlarged, firm, hard and irregular, with the capsule thickened. On section it is of yellowish color, sometimes with numerous whitish, miliary nodules consisting of minute gummata. There is also decided increase of the interstitial connective tissue, with consequent atrophy of many of the hepatic cells, and narrowing of the branches of the blood-vessels and small biliary passages. The process may be diffuse, or limited to certain areas. After early childhood the liver less often shows alterations, although gummata of considerable size are sometimes found. The alimentary canal may exhibit condylomata of the tongue, and the same lesion or chronic inflammation or ulceration of the pharynx and tonsils. Cellular infiltration and interference with parenchymatous development may occur in the stomach and intestines.

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[1] Münch. med. Wochenschr., 1918, LXV, 71.

[2] Jahrb. f. Kinderheilk., 1901, LIV, 685.

A catarrhal inflammation of the nose is very frequent, and ulcerative lesions, superficial or deep, are often observed in the later stages of the disease. Perichondritis or ulceration of the larynx is sometimes seen, and exceptionally gummata may form in the trachea or bronchi and terminate in stenosis. In the fetus and in infants dying soon after birth there is often present the so-called "white hepatization" of the lung. In this a considerable portion of the pulmonary tissue appears whitish-grey, airless, and smooth on section, due to filling of the alveoli with degenerated epithelium, thickening of their walls, and increase of the interstitial connective tissue. Pulmonary changes at later periods of the disease are less common, and consist of gummatous deposits or extensive interstitial inflammation. Lesions of the heart are not uncommon, and gummata and interstitial myocarditis have been seen even in infancy. Aortitis may occur. The nervous system is not frequently involved. Gummata are only rarely observed in the brain and spinal cord, and chronic meningitis, chiefly basilar, is sometimes met with; but the most frequent lesion, especially in infancy is, hydrocephalus. (See Vol. II, p. 647.)

The genito-urinary system exhibits chronic interstitial nephritis, occasionally even in infancy. In the fetus and new-born enlargement of the kidney and parenchymatous changes are common. Gummata of the kidney are exceptional in early life. The testicle may be enlarged as the result of interstitial inflammation, and gummata are sometimes found. Chronic suppurative otitis is a frequent result of syphilitic pharyngitis. The eyes are less often involved in early life; choroiditis; retinitis and optic neuritis sometimes occurring; and iritis developing even during intrauterine life. Keratitis is common among the later lesions. Multiple enlargement of the lymphatic glands is seen, especially in the groin, axilla, neck and about the elbow. The pancreas, thymus gland, peritoneum, suprarenal bodies and thyroid gland occasionally exhibit gummata, interstitial inflammation, or other syphilitic manifestations. Small cystic formations are frequently found in the thymus gland. Lesions of the skin are described under symptoms.

Symptoms.—It is convenient to divide the symptoms of hereditary syphilis into I. The Early Manifestations; and II. Later Manifestations.

I. The Early Manifestations of Hereditary Syphilis.—Mothers with recent syphilitic infection, contracted at or near the time of conception, very commonly abort and the accident is likely to be repeated in later pregnancies. The lesions found in the fetus, born dead, have already been described. (See Pathological Anatomy.) Other infants are still-born at term. Some are born alive with evidences of severe infection, and are usually capable of living only a few days or weeks. They are feeble, wasted, atrophic, and exhibit all the symptoms of extreme inanition. There may be present at birth coryza and a pemphigoid eruption of the skin, especially on the soles and palms, but sometimes elsewhere. Enlargement of the liver and spleen are demonstrable. The skin about the mouth shows fissures, and excoriations are present on the buttocks. Still other infants, although feeble and poorly developed and with enlargement of the liver and spleen, do not at first possess the group of symptoms characteristic of syphilis, but after a few days rapidly develop them, the atrophy and inanition being prominent features from the moment of birth. The large majority of syphilitic infants, however, appear healthy and well-developed at birth and symptoms appear only after an interval (*infantile syphilis*). A general estimate by Miller[1] based upon his own observations with those of Zeissl and Kassowitz would make from 50 to 65 per cent. of the cases first show symptoms in the 1st month, chiefly in the 3d and 4th weeks; from 20 to 30 per cent. in the 2d month, and nearly all of the remaining 10 to 15 per cent. in the 3d month. Generally the danger of syphilis appearing is over after 3 months but to this there are exceptions, and the first symptoms may occasionally not develop until the 4th month or even later.

The earliest symptom in the child apparently healthy at birth is usually a persistent coryza, producing the "snuffles" often described. This is soon followed by a hoarse, high-pitched and very persistent crying especially at night. In a short time cutaneous manifestations appear, frequently accompanied by slight fever; and then, or earlier, evidences of osseous lesions with consequent apparently paralytic conditions. Sometimes the symptoms mentioned are little, if at all, marked, and severe visceral affections are more prominent and often lead to a fatal ending.

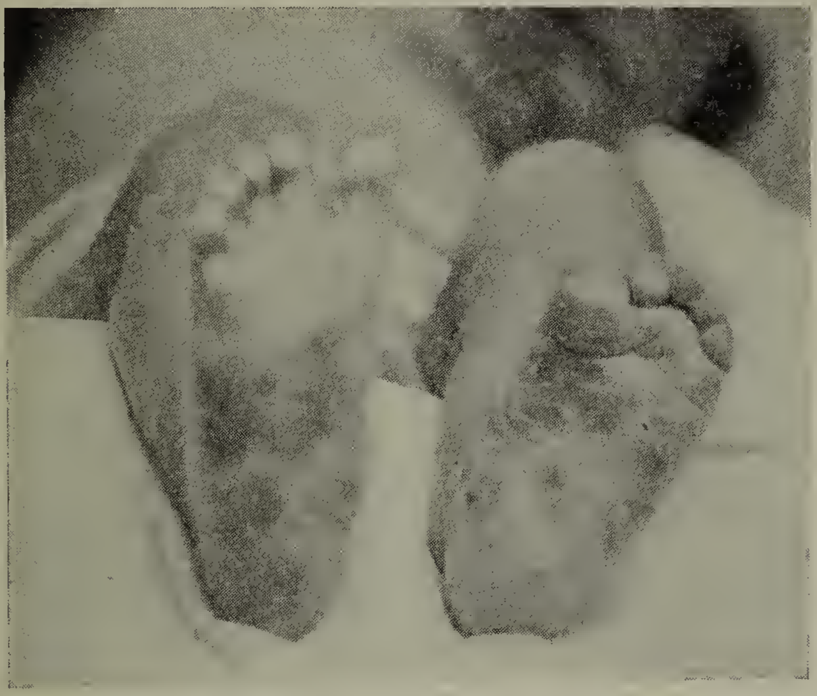

FIG. 189.—HEREDITARY SYPHILIS, BULLOUS ERUPTION ON SOLES OF FEET. *Courtesy of Dr. J. F. Schamberg.*

As a rule, however, decided visceral symptoms are characteristic of fetal syphilis, and the earlier they manifest themselves in the new-born the worse the general symptoms are liable to be. The majority of infants who are in good condition at birth and exhibit no syphilitic manifestations of any sort for some weeks, will present no visceral symptoms of any moment.

The promptness of development and the severity of the symptoms are, in general, proportionate to the intensity of the infection. In severe cases increasing debility and wasting begin early and advance as the disease progresses, and infants my finally exhibit all the evidences of extreme marasmus. In milder cases the general health and nutrition may remain unaffected.

The symptoms referred to, as well as others which develop, require more detailed description:

Cutaneous Symptoms.—The severest symptom of this nature, which has been designated *pemphigus syphiliticus neonatorum*, is a bullous eruption, with bloody or purulent contents, oftenest found on the palms

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[1] Jahrb. f. Kinderheilk., 1888, XXVII, 362.

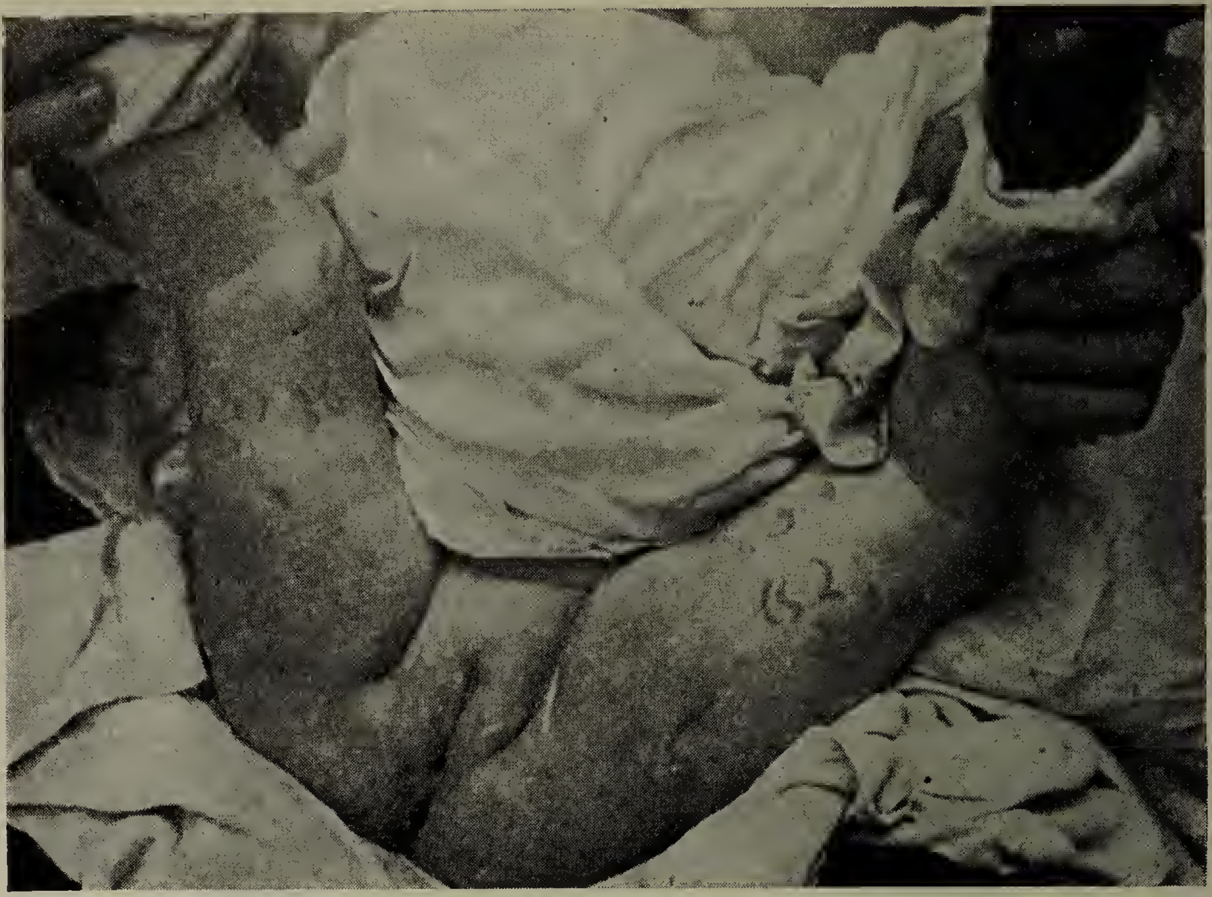

FIG. 190.—HEREDITARY SYPHILIS, MACULO-PAPULAR ERUPTION. Unusual degree of annular appearance. *Courtesy of Dr. J. F. Schamberg.*

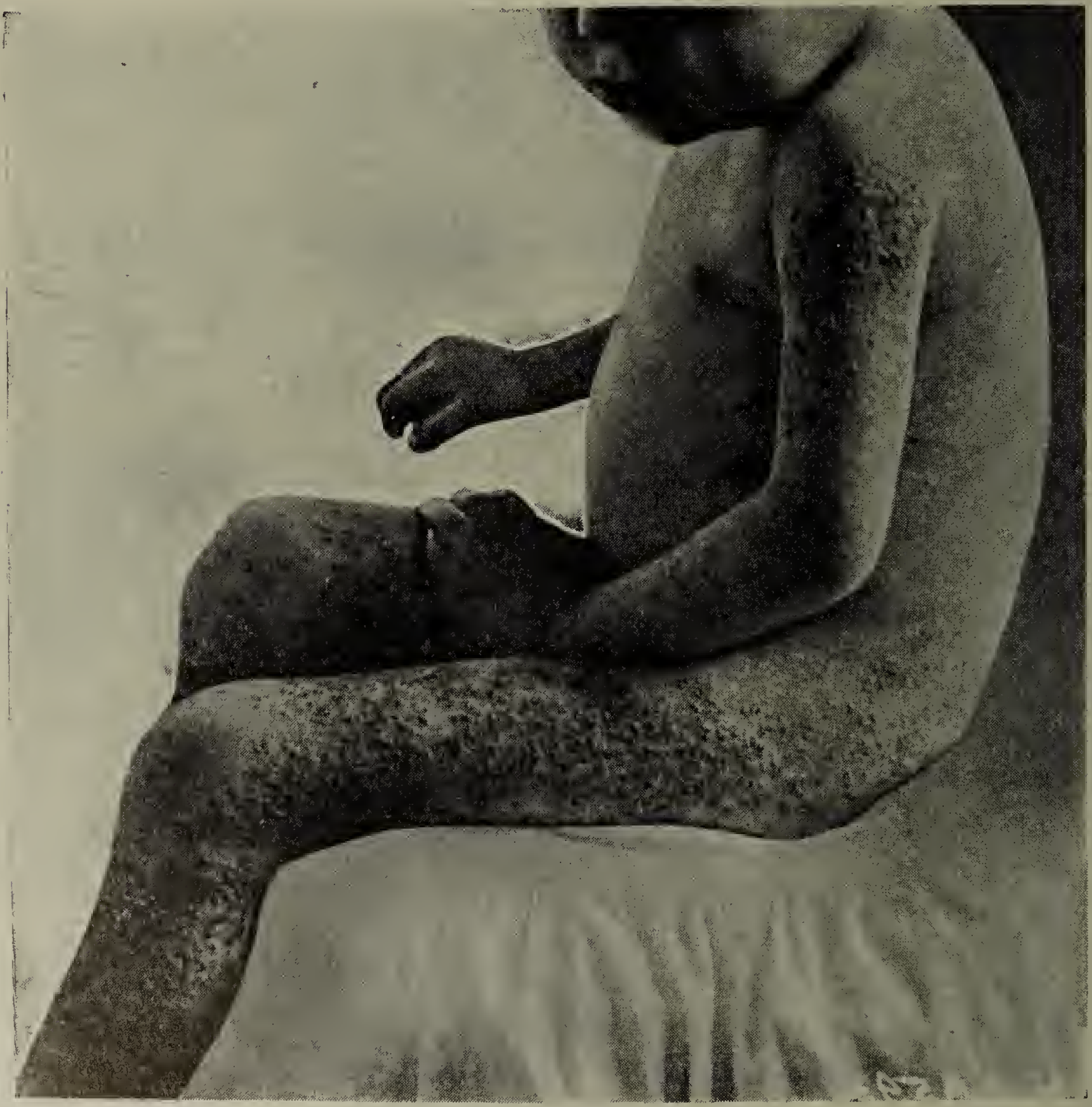

FIG. 191.—PAPULO-PUSTULAR SYPHILODERM. *Courtesy of Dr. M. B. Hartzell.*

and soles, but at times wide-spread and causing extensive exfoliation of the skin (Fig. 189). Most instances show the lesion at birth and the cases generally terminate fatally. In milder cases of syphilis there is no cutaneous eruption at first, and the earliest evidence of it in any form appears generally about a week after the development of the coryza. The eruption may be circumscribed or diffuse. The *circumscribed* form is much the same as that found in acquired syphilis. It is oftenest maculo-popular, and occurs in the shape of small, slightly elevated, pea-sized or larger macules associated with papules more or less numerous, situated especially upon the lower extremities, the face, scalp, neck, and flexor surfaces of the upper extremities, the palms and soles. Scaling may be present upon the surface. It is unattended by itching and varies in color from coppery-red to brown or yellowish according to the age of the lesion. Absorption may take place in the center, producing in some cases a well-marked annular appearance (Fig. 190). In certain regions there may develop flattened elevations with moist surfaces—the "moist condyloma" or mucous patch. These are commonest about the mouth, the anal region and the genitals, but may appear in any situation where the skin is thin and delicate, such as behind the ears, between the fingers and about the navel. The condyloma, however, is not the earliest eruption of hereditary syphilis, but is particularly characteristic of relapses. A more papular eruption of a brown-red color is seen oftenest upon the palms and soles and the forehead. Less often seen, and in severer cases, is a papulo-pustular eruption (Fig. 191) which may terminate in ulcerated or ecthymatous lesions. The roseola of acquired syphilis is rarely if ever witnessed in the hereditary form.

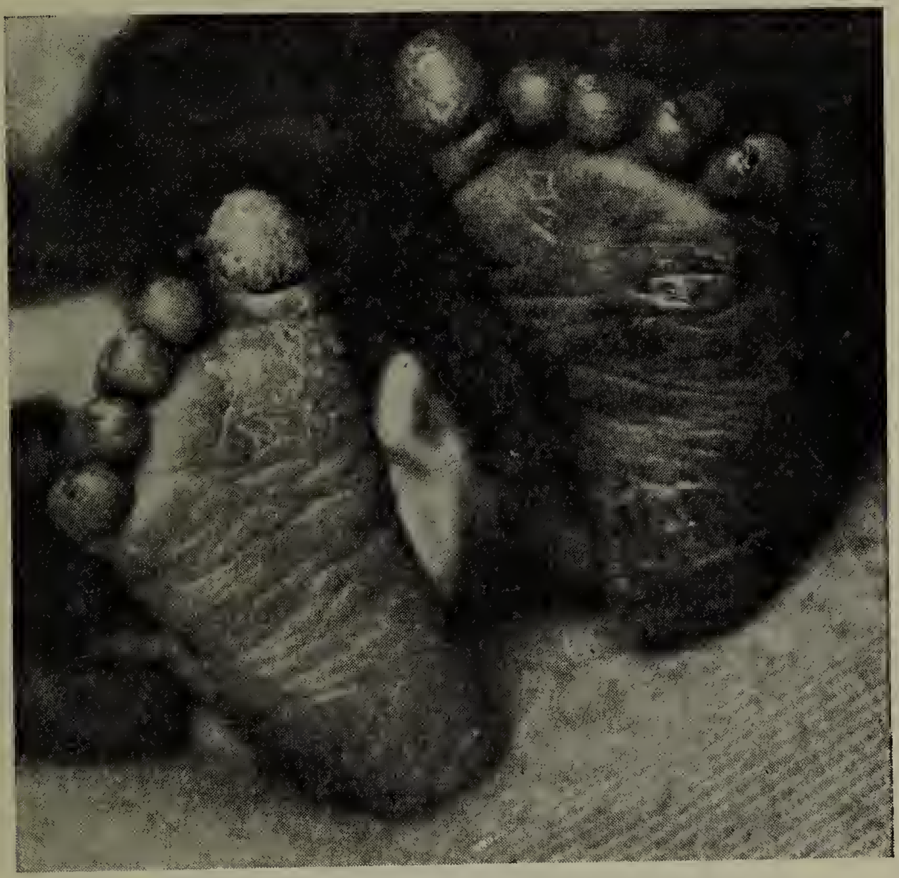

Fig. 192.—Syphilitic Scaling of Feet.

From an infant aged 6 weeks, in the Children's Medical Ward of the Hospital of the University of Pennsylvania. At the age of 2 weeks snuffles and cutaneous eruption developed; the latter seen as macules on the face and limbs and as a red diffuse infiltration on the soles of the feet. The illustration shows the typical scaly appearance following.

The *diffuse* eruption is common and very characteristic, and is not observed in acquired syphilis. It consists of a diffuse infiltration of the skin, situated usually on the palms and soles, face, scalp, genital and anal regions, and the flexor surfaces of the thighs. It presents a somewhat shining surface of a copper-red or brownish-yellow color and renders the skin distinctly stiffened. Slight desquamation is often present. In

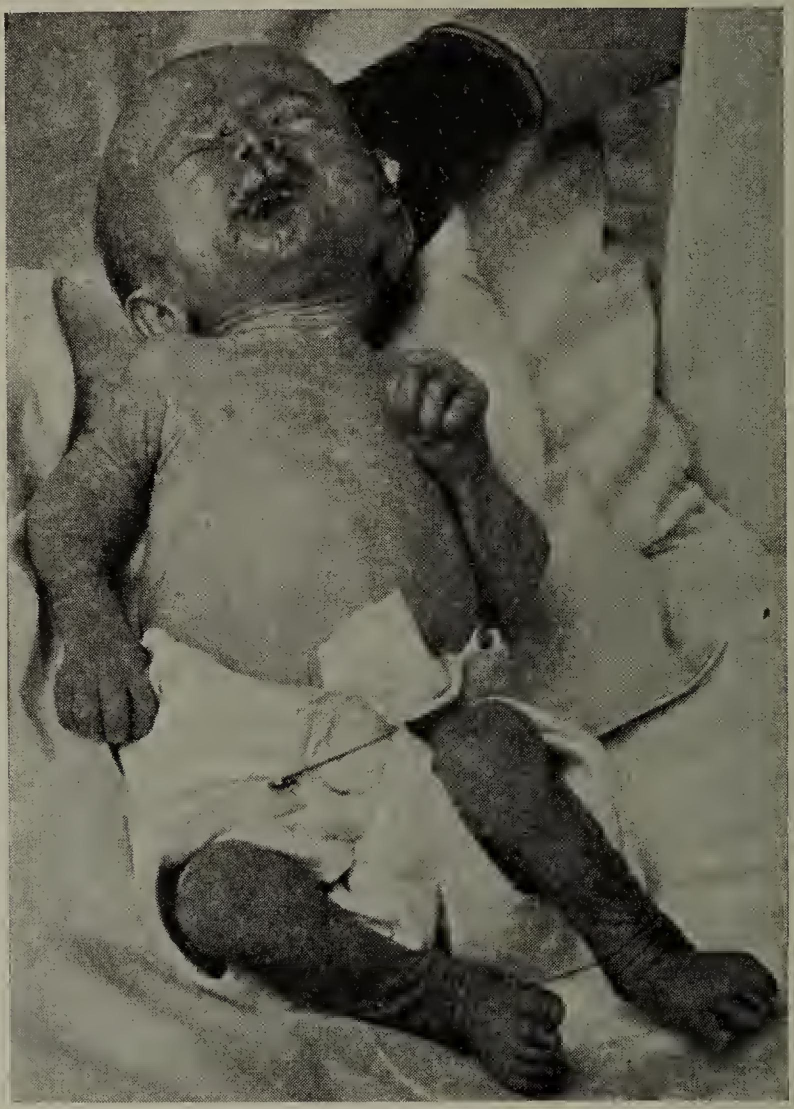

FIG. 193.—FISSURES ABOUT THE MOUTH IN HEREDITARY SYPHILIS.

Female child, aged 6 months, in the Children's Ward of the Hospital of the University of Pennsylvania. Previous history not discoverable, except that the child had been ill since its 1st month. Under treatment for a month with mercury and arsphenamine; no improvement; death.

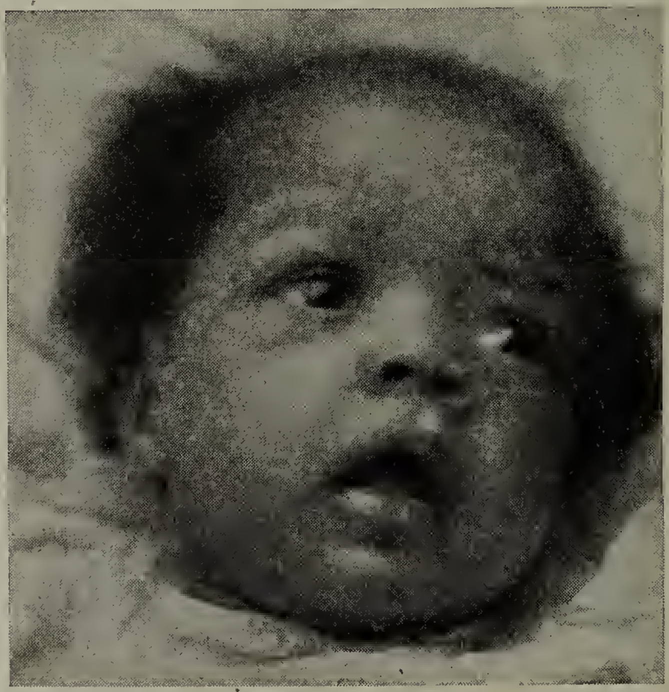

FIG. 194.—SYPHILITIC ALOPECIA.

Courtesy of Dr. J. F. Schamberg.

other cases it produces extensive desquamation in small scales or larger flakes (Fig. 192). Sometimes when inflamed it suggests the appearance of eczema with formation of crusts, but differs in the presence of stiffness of the skin and the absence of the intensely red color of the latter disease.

Fissures in the skin are very characteristic early symptoms. They occur especially about the mouth and eyelids, at the alæ of the nose, and at the anus. Those about the mouth are situated on the lips, and extend a short distance into the skin beyond. They are often arranged radially, especially at the labial angle (Fig. 193), are narrow, infiltrated, rather deep, painful, bleed readily and are often covered with crusts. When healed they may leave very characteristic linear scarring.

The nails are often dry, shriveled, narrowed and curved in a sharp transverse curve, exhibit transverse fissures, and may fall off. In other cases inflammation about the nail occurs (syphilitic paronychia). There may be loss of hair from the scalp (Fig. 194), eyelids and eyebrows. This is present at birth in fetal cases.

Mucous membranes.—Lesions of the mucous membranes, apart from the rhinitis to be described, are not frequently early symptoms in infancy and are usually not seen until the infant is some months old. Broad condylomata may develop on the lining of the mouth, the tongue and the vulva, but oftener upon the skin near the muco-cutaneous border (p. 663). Ulcers, general superficial, may occur upon the mucous membrane of the mouth, pharynx, and elsewhere. "Snuffling" is generally the first manifestation of the disease, and a very persistent one. It is nearly always present, and often at birth, and may be the only symptom. The nasal mucous membrane is swollen, and respiration may be much interfered with, rendering sucking difficult and sleep disturbed. Later there develops a serous, mucous or muco-purulent discharge, often tinged with blood, and thick crusts block the nostrils and interfere still further with breathing. The mouth may be dry from the constant mouth-breathing. Hoarsenes may attend the coryza, being sometimes one of the earliest symptoms. Hemorrhages occasionally occur from the mucous membrane and from some of the cutaneous lesions, or sometimes in the internal organs. Some of the cases of hemorrhagic disease in the new-born very probably owe their origin to syphilis.

The **lymphatic glands** may show moderate enlargement, especially in the axilla, elbow, groins and neck. The enlargement of the cubital glands is suggestive, but not proof of the existence of syphilis, since it may occur in other diseases also. From a study of 390 children Reichenecker[1] concludes that epitrochlear adenitis is most frequently due to syphilis, and Götzky[2] believed that in infancy it is almost certainly dependent upon this disease. Fabbris[3] found enlargement of the epitrochlear glands in 71 per cent. of 112 children with active manifestations of syphilis; and in 63 per cent. of 154 with latent syphilis, whereas it was present in only 21 per cent. of children with rickets and in only 26 per cent. of tuberculous children. He considers enlargement of the retromastoid glands next in importance to that of the epitrochlear. It should, however, be stated that these views do not meet universal acceptance, and the diagnostic value of enlargement of the lymphatic glands in congenital syphilis is probably over-estimated. Syphilitic glands

[1] Le nourrisson, 1915, III, 193. Ref. Brit. Jour. Child. Dis., 1916, XIII, 60.
[2] Zeitschr. f. Kinderheilk. Orig., 1913, VII, 113.
[3] La Pediat., 1922, XXX, 560.

appear as multiple, hard bodies, without tendency to suppurate. The **teeth** are often late in their eruption and decay early, but show nothing characteristic.

The **osseous system** (see also Vol. II, p. 752, Syphilis of the Bones) is frequently affected in early life, the most characteristic symptom being an osteochondritis, first described by Wegner.[1] According to Sutherland and Mitchell[2] the frequency with which this change is reported by various observers varies from 70 to 93.4 per cent. The lesion really

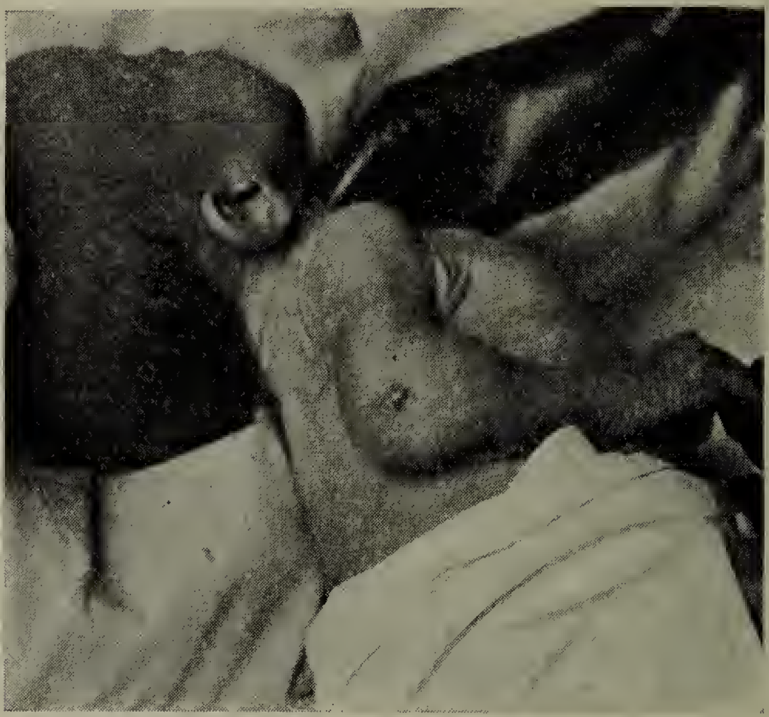

Fig. 195.—Syphilitic Epiphysitis with Secondary Suppuration.
Same case as Fig. 193.

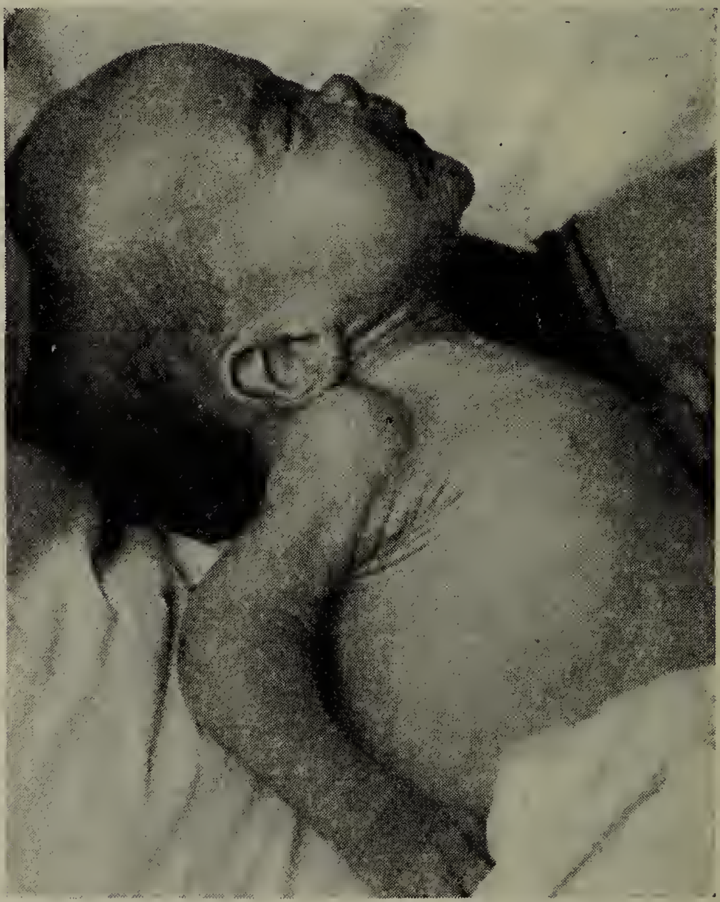

Fig. 196.—Saddle-nose in Early Syphilis.
Same case as Fig. 193.

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[1] Virchow's Arch. f. path. Anat., 1870, L, 305.
[2] Jour. Amer. Med. Assoc., 1923, LXXXI, 1752.

begins in fetal life and lasts a variable time, although the clinical evidences of it are usually not discoverable at birth. It appears first as a tender swelling at the junction of the shaft and epiphysis, oftenest in the extremities, and may advance to complete separation of the epiphysis with temporary loss of power and pain on passive movement, suggesting paralysis;—the syphilitic pseudo-paralysis described by Parrot.[1] This paralytic condition occurs oftenest in the earlier months of life, and usually in one arm, which hangs motionless with the forearm in full pronation and the palm of the hand turned outward. The inability to move the limb depends upon pain, not upon loss of power. The process may involve the joint itself with secondary suppuration (Fig. 195), or a large portion of the shaft of the bone may be attacked by the disease. Usually, however, there is a tendency toward spontaneous disappearance of the osteochondritis after the 3rd month, even when antisyphilitic treatment has not been instituted. It is undetermined whether all the cases of pseudo-paralysis depend in reality upon an osseous lesion. A form of osteitis of the bones of the fingers (dactylitis), and, less often, of the toes, has been described by Hochsinger[2] as observed during the early symptoms in infancy. It begins chiefly in the proximal phalanx, is bilateral, and never advances to suppuration. (See Syphilitic Dactylitis, Vol. II, p. 754, Fig. 410.) A more destructive dactylitis may develop at a later period in infancy. It is described under the later symptoms of syphilis.

Craniotabes is an occasional osseous lesion seen in infancy. It consists of thin, softened spots, particularly in the occipital region, but is not entirely pathognomonic of syphilis since it may occur in rachitis as well. (See p. 689; Vol. II, p. 728.) Flattening of the bridge of the nose producing the "saddle-nose" is sometimes an early symptom (Fig. 196). It may be only an apparent flattening at this stage, but often depends upon actual involvement of the cartilages and bones. The affections of the **eye** and **ear** among the early symptoms of syphilis, already referred to in discussing pathological anatomy, consist principally of iritis, choroiditis, optic neuritis and purulent otitis.

Visceral lesions are, as indicated, early symptoms more characteristic of syphilis of the fetus and the new-born than of the disease first showing itself in infants past this period. They date from intra-uterine life and are well-marked only in unfavorable cases. The commonest clinical manifestation is decided enlargement of the liver, the organ presenting a hard edge on palpation and projecting considerably farther downward than normal. Ascites and icterus occasionally result. (See Congenital Obliteration of the Bile Ducts, p. 307.) The spleen also may be much enlarged. Symptoms depending upon lesions of the nervous system are not very common early manifestations. Among them are those of meningitis and the consequent hydrocephalus. This may develop at the time of the early cutaneous eruption, or later during relapses. A functional disturbance is, however, very frequent; namely the great wakefulness with persistent crying, especially at night. This may very probably be due, at least to some extent, to the pains in the bones. Albuminuria may or may not depend upon the existence of syphilitic nephritis. According to Hintzelmann[3] evidences of nephritis were found in 14 out of 41 syphilitic infants.

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[1] Arch. de phys. norm. et path., 1871–2, IV, 319.

[2] Pfaundler and Schlossmann. Handb. d. Kinderheilk., 1906, I, 2, 916.

[3] Zeitschr. f. Kinderheilk., Orig., 1913, IX, 27.

The **general nutrition** suffers badly in severe cases. With the development of other symptoms there is a rapid emaciation with marked anemia, the blood showing diminution of the red cells and hemoglobin. The skin often exhibits, besides the pallor, a peculiar *cafe-au-lait* color, either throughout or in certain localities, especially the face. The veins of the scalp are sometimes much dilated. The debility is great, and a fatal ending may occur dependent upon a marantic state, and entirely independent of any discoverable affection of the digestive apparatus or fault with the food. In cases which recover, the anemia persists after the disappearance of all other symptoms. Sometimes the marasmus is the earliest or even the only symptom, and not infrequently it does not yield at all to antisyphilitic treatment.

The *frequency of the occurrence* of the different early symptoms, according to the observations of Still,[1] shows snuffling 70 per cent.; cutaneous eruptions 69 per cent.; splenic enlargement 45 per cent. and quite decided in 22 per cent.; affections of the eye 15 per cent., laryngitis 14 per cent.; epiphysitis 11 per cent.; orchitis 8 per cent. Hochsinger[2] found enlargement of the liver in 31 per cent. of the cases, always accompanied by splenic enlargement.

II. Later Manifestations of Hereditary Syphilis.—The symptoms just described would be classified as secondary ones in the acquired disease, there being, of course, no primary lesion in the inherited affection. They appear for the most part in the 1st and 2d months of life, and most or all of them disappear entirely and permanently in a few weeks under proper treatment. Unless, however, treatment is long continued, recurrence occurs in a few months or by the end of the 1st year or later, exhibiting some of the original cutaneous and other symptoms, but with a special tendency in early childhood to the development of condylomata. That this may take place in spite of medication is shown by the fact that in 208 cases of congenital syphilis observed by Hochsinger[3] over a period of more than 4 years, 131 (63 per cent.) showed recurrence, and of these 112 (54 per cent.) had undergone treatment carried out in a thorough manner. A lowering of the large number of recurrences has been effected since the intensive treatment by various arsenical preparations has been instituted.

In addition to those described, new symptoms may appear which would be classified, for the most part, as tertiary in acquired syphilis; prominent among them being gummata in various regions. These are seen usually after the period of early childhood. In other cases the later symptoms develop about the time of puberty or even after this, without any of the earlier secondary symptoms having been observed, or, if noticed, so slight that their nature was unrecognized. To this form of the disease is sometimes applied the title "*Syphilis hereditaria tarda.*" The existence of such a condition without previous symptoms has been much disputed, as is, indeed, the proper application of the name.

The principal later symptoms of syphilis are the affections of the permanent teeth and of the bones, eyes and ears, and various lesions of the skin, principally of a condylomatous or gummatous nature. Some of the later symptoms must be described more in detail:—

The **general nutrition** suffers, there being a decided retardation of growth, general impairment of vigor, delayed puberty, and anemia. The last may persist after other symptoms have disappeared.

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[1] Lancet, 1904, II, 1402.

[2] Pfaundler and Schlossmann, Handb. d. Kinderheilk., 1906, I, 2, 921.

[3] Ergebnisse. inn. Med. u. Kinderheilk., 1910, V, 84.

Osseous changes are present, usually represented by localized gummata or by a hyperplastic osteoperiostitis. This latter affects chiefly the long bones of the extremities and the cranium. It produces thickening and tenderness, and pain which is most marked at night. In the tibia, which is the bone oftenest involved, there is a thickening and curvature of the anterior edge, resulting in the well-known sabre-tibia (Vol. II, p. 755, Fig. 411). When the cranium is attacked there results a diffuse thickening with corresponding deformities (Fig. 411). A dactylitis affecting one or more fingers and often going on to necrosis, and in which the soft parts are also involved, is seen oftener in early childhood than at a later period. It attacks usually the proximal phalanx. (See Dactylitis, Vol. II, p. 754.) Gummata of the bones (Fig. 197) may develop in various regions and persist for a long time; finally breaking down and forming ulcers, or being absorbed and leaving depressions. On the skull, especially the parietal and frontal regions, they form large nodular masses. These or the osteoperiostitis referred to produce decided alterations in the shape of the head—the "keel-shaped" cranium, with central frontal deposit, and the "natiform" cranium, with bilateral parietal deposits. Hochsinger[1] considers these as more commonly early symptoms. A frequent site of gummata in childhood is the nasal septum and hard palate, in both of which localities breaking down is liable to occur. The loss of tissue is sometimes wide-spread and the palate may be perforated or much of the nose destroyed. The saddle-nose, described as an occasional early symptom, may develop in childhood also, as the result of destructive necrosis in the bone and cartilage (Fig. 198). A painless swelling of the knee-joints is a not uncommon distinctly later

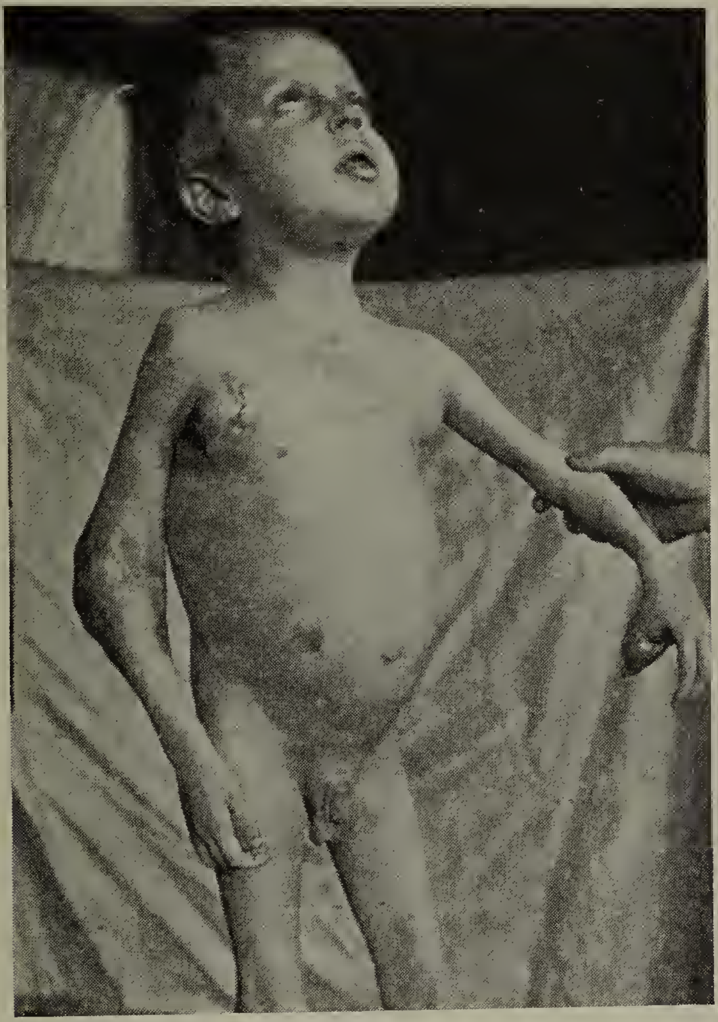

Fig. 197.—Gummata of Elbows and Chest in Late Hereditary Syphilis.
Courtesy of Dr. H. R. Wharton.

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[1] Pfaundler and Schlossmann; Handb. d. Kinderheilk., 1906, I, 2, 920, 931.

symptom. It is bilateral and may be either a hydrarthrosis (Fig. 199) or a hyperplastic synovitis, and results finally in ankylosis. Less often the ankle is attacked, but any joint in the body may be affected. Goff[1] has found pes cavus, generally bilateral, a rather constant occurrence in this disease.

The **skin** may exhibit syphilitic tubercles and gummata in various situations, but not so frequently as in acquired syphilis. These tend to break down and leave ulcers, often covered by thick crusts and finally replaced by scars. A papular eruption of the face may develop during relapses. Condylomata are, as stated, the cutaneous manifestations particularly liable to appear during relapses, and may, indeed, be the only cutaneous lesion after the 1st year and in early childhood. They are the commonest of all symptoms at these times but are not frequent after this period. In general, the longer the time elapsed since infancy, the less pronounced as symptoms do cutaneous eruptions become.

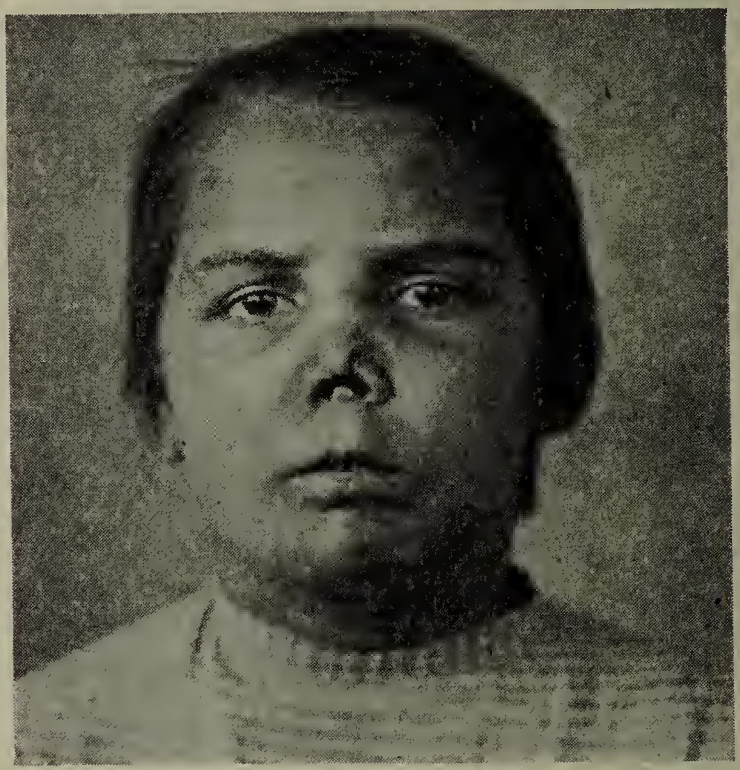

Fig. 198.—Saddle-nose in Later Hereditary Syphilis.
Courtesy of Dr. Harry Lowenburg.

The incisor **teeth** of the second dentition, especially the 2 upper central ones, show very characteristic changes, described by Hutchinson[2] (Fig. 200). They are far apart and small, with notching of the cutting surface. In other cases they are tapering and characteristically "peg-shaped." Unerupted Hutchinson's teeth may be demonstrated by the *x*-ray (Stokes and Gardner).[3] The so-called Fournier's or Moon's tooth is also common, but not entirely pathognomonic. It consists of a 6-year-old molar in which the hypoplastic and deformed crown is separated by a circular line of demarcation from the body of the tooth (Stoll).[4] Mucous placques and ulcers appear on the mucous membrane of the mouth, tongue and pharynx, and alterations of the larynx may develop.

Enlargement of the **lymphatic glands,** referred to as seen in infancy, becomes more common after the 1st year. The **nervous system** is involved in many diverse ways in the later symptoms of hereditary syphilis, although not as frequently as in adults. Jeans[1] found that ⅓ of the syphilitic children he examined had such involvement. Evidence corroborative of this diagnosis may be observed in the spinal fluid in the form of a positive Wassermann reaction, increased number of cells, positive globulin and albumin reactions and a characteristic colloidal gold curve. Chronic meningitis may develop (see Vol. II, p. 627). The pseudo-

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[1] Jour. Amer. Med. Assoc., 1926, LXXXVI, 392.
[2] Brit. Med. Jour., 1858, II, 822.
[3] Jour. Amer. Med. Assoc., 1923, LXXX, 28.
[4] Jour. Amer. Med. Assoc., 1921, LXXVII, 919.

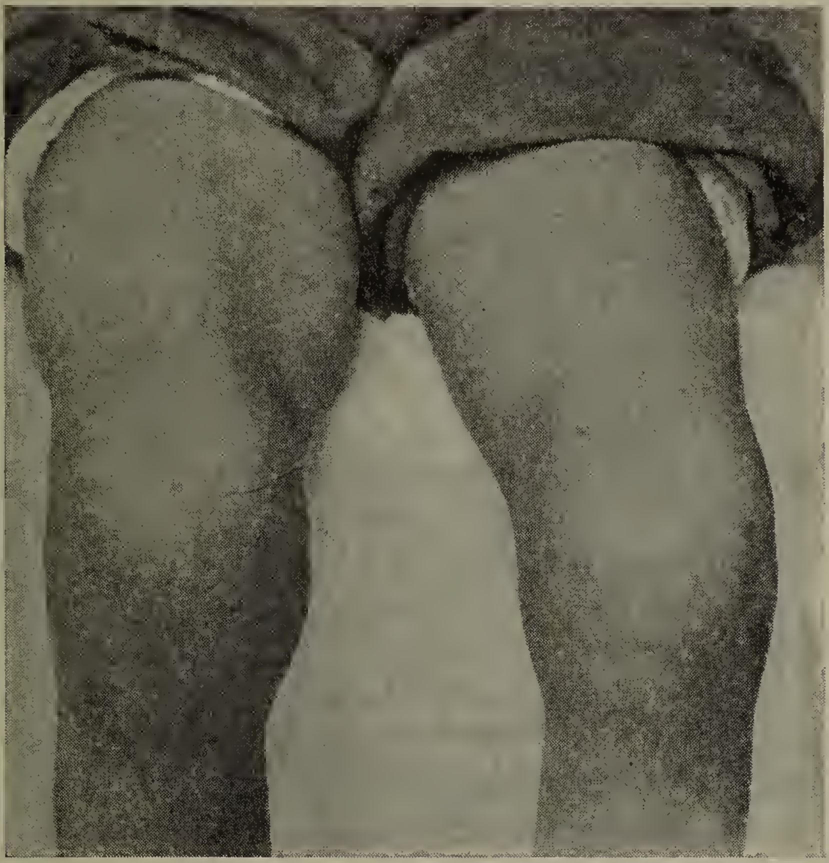

Fig. 199.—Hydrarthrosis in Later Hereditary Syphilis.
Courtesy of Dr. J. F. Schamberg.

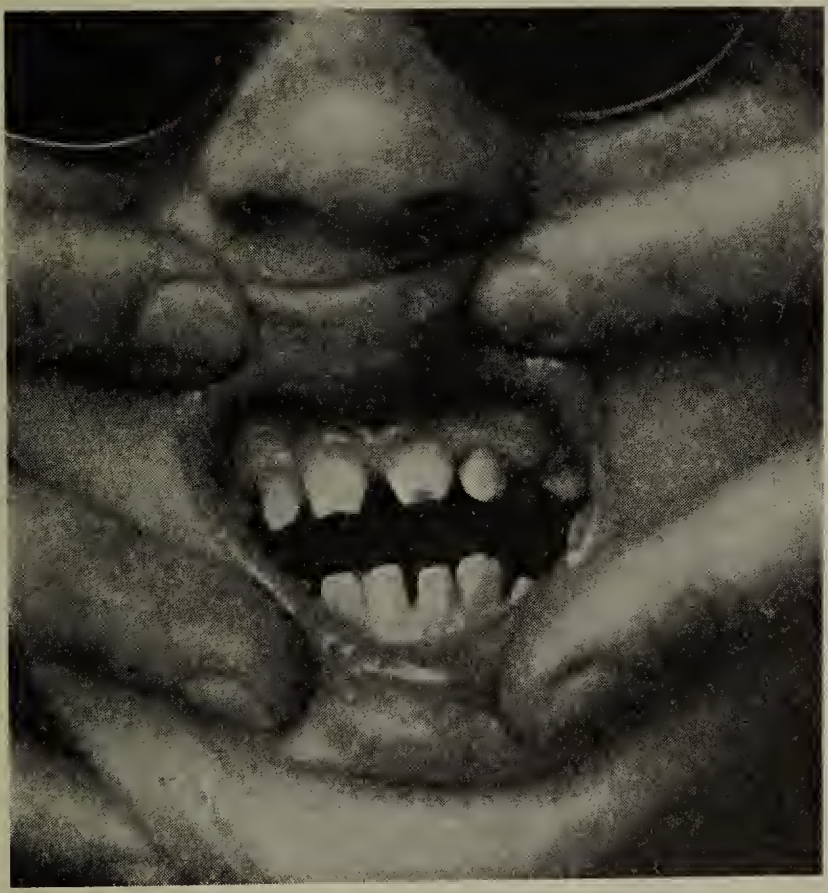

Fig. 200.—Hutchinson's Teeth in Later Hereditary Syphilis.
Courtesy of Dr. J. F. Schamberg.

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[1] Amer. Jour. Dis. Child., 1919, XVIII, 173.

paralysis described appears exceptionally to be of nervous origin. Paralysis of the cranial nerves may occur dependent upon meningitis, encephalitic processes the result of endarteritis, or gummatous growths. Retarded mental development perhaps not appearing until later childhood, idiocy, and epilepsy may be encountered. White and Veeder[1] observed defective mental condition in 15.4 per cent. of 308 cases of hereditary syphilis. It should be stated, however, that Weiss and Izgur[2] in the study of a large number of mental defectives could find no evidence that the syphilitics were more predisposed to mental disturbances than were others. Juvenile paresis and, less often, tabes are sometimes seen.

The **eye** likewise, exhibits late symptoms. Chronic interstitial keratitis is one of the most frequent of these. It occurs generally in later childhood, attacks both eyes, and produces slight cloudiness or even entire opacity of the cornea, which, however, usually disappears under treatment. Choroiditis, iritis, and retinitis may occur. **Hearing** may be affected by chronic otitis, or, in older children, by a disturbance which is of nervous origin, perhaps labyrinthine. The latter is very characteristic. It may result in deaf-mutism. Syphilis of the internal organs may also be among the later symptoms, dependent upon interstitial changes or the formation of gummata. Decided enlargement of the liver and spleen may be observed, and combined with this is alteration of the blood, great anemia developing with diminution in the number of red blood-cells and hemoglobin. A clinical picture may result suggesting Banti's disease (Osler).[3] Interstitial nephritis may develop, and the pancreas and testicles sometimes exhibit lesions. A form of chronic ascites resembling tuberculous peritonitis is occasionally seen. Syphilitic endarteritis may cause areas of gangrene, or the larger arteries may be affected and produce aneurysms even in childhood.

Reviewing the later symptoms described it will be noticed that some are characteristic of relapses in infancy and early childhood, while others, chiefly of true late hereditary syphilis, appear oftenest about the period of puberty. Among the former are condylomata of the skin and mucous membranes, debility, retardation of growth, deformities of the skull, dactylitis, and enlargement of the lymphatic glands. Among the latter are osteoperiostitis, gummata in various regions including the viscera, affections of the teeth, keratitis, labyrinthine deafness, affections of the knee-joints, and retarded mental development and other psychic disturbances. The combination of deformed teeth, deafness and keratitis has been described as "Hutchinson's triad." Gummata may, however, occur at an earlier period, and visceral involvement may take place in the early relapses or as a quite late symptom, as well as being seen in the new-born and the fetus. Indeed there is no sharply dividing line between the ages at which the various later symptoms may show themselves. It is rather the stage of the disease which is to be considered.

Prognosis of Syphilis.—The prognosis of **hereditary syphilis** varies with the severity of the infection, and with the promptness and thoroughness of treatment. A large proportion of syphilitic mothers abort, or are delivered of still-born children, and those infants born alive with decided evidences of fetal syphilis nearly always die soon. Taking all cases together the death-rate is high. Coutts[4] in 1102 syphilitic pregnancies reported 376 abortions and 396 early deaths, making a total death-rate of 70.05 per cent., without including the deaths occurring later in life. In a series of 414 pregnancies reported by Le Pileur[1] 154 aborted, and of 260 infants born alive 141 died in a very short time. The total mortality equalled 71.26 per cent. These two series occurred in hospital practice. Apart from this, however, the death-rate usually does not reach these figures. Thus, in a series of 1127 pregnancies in syphilitic families, reported by Fournier[2] occurring in the civic population of Paris in private practice the mortality in the offspring, including abortions, equalled but 42 per cent. The majority of infants apparently healthy at birth and showing no symptoms for some weeks, can be saved by energetic treatment. In general, the less recent the disease in the parents, the better the state of nutrition in the infant when the symptoms appear, the greater the delay in the appearance of these symptoms and the less their intensity, and especially the earlier antisyphilitic treatment is commenced after birth, or preferably before it, the greater will be the probability of the child living. White and Veeder's[3] figures show that in 308 treated cases 22 per cent. were cured; while 47 per cent. were either unimproved or died. Patients with involvement of the central nervous system usually exhibit little improvement. The importance of prolonged persistent treatment in reducing the mortality is demonstrated by the experiments of Müller and Singer[4] in 214 children observed over a period of years. From 7 to 9 courses of treatment were given during 3 to 4 years, with a resulting mortality of only 22.8 per cent.

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[1] Amer. Jour. Syph., 1922, VI, 353.
[2] Jour. Amer. Med. Assoc., 1924, LXXXII, 12.
[3] Clin. Jour., 1914, XLIII, 462.
[4] Lancet, 1896, 1, 971.

Very important, too, is the influence of a supply of suitable nourishment, especially breast-milk, children fed in this way having a far better chance of survival. It is noteworthy, also, that the later children of syphilitic parents oftener survive than do the earlier ones. Death is due not so much to the lesions themselves, unless the viscera are decidedly involved, as to the profound effect which the virus exerts upon the constitution, with the general asthenia which results and the ease with which complicating processes, such as sepsis, pneumonia, intestinal disorders, and the like, develop and determine a fatal ending. The degree of the marantic symptoms constitutes probably the most important prognostic guide. There is always, too, to be remembered the tendency to repeated relapses, these sometimes occurring in spite of thorough specific treatment. They constantly diminish in intensity, however, if treatment is persisted with.

There is a tendency for symptoms of the tertiary type to appear in later childhood, even in cases which seemed apparently entirely cured earlier, although still more so in those who have never received early treatment. Most of these cases yield to treatment unless severe visceral manifestations are present, especially of the kidneys and liver. The nervous manifestations, too, are often entirely resistant.

The *duration* of the disease is very uncertain. Under proper treatment in favorable breast-fed subjects the characteristic cutaneous eruptions generally disappear in 3 to 4 weeks; the rhagades, glandular swelling and snuffling lasting a longer time, the last mentioned being one of the most resisting symptoms. The asthenia and anemia, however, may remain after other symptoms have disappeared, and the child may die of causes not directly syphilitic. Only long persistence in treatment can prevent the return of symptoms or the development of new ones. Untreated infants lose the cutaneous eruption in a few weeks but soon relapse. The later symptoms of the tertiary type yield only slowly, but the prognosis as regards life is much better, Rabl[1] finding only 9 deaths in 93 cases. The chance of ultimate recovery under satisfactory continued treatment is well illustrated in the statistics of Hochsinger:[2] of 263 cases kept under observation for from 4 to 12 years 79 had died, 112 exhibited symptoms of some sort, not always syphilitic, and 72 were entirely free from symptoms of any kind.

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[1] Thèse de Paris, 1851. Ref., Fournier, L'hérédité syphilitique, 1891, 312.

[2] L'hérédité syphilitique, 1891, 309.

[3] Amer. Jour. Syph., 1922, VI, 371.

[4] Arch. f. Kinderheilk., 1919, LXVII, 161.

The prognosis of **acquired syphilis** in infancy is very much better than that of the hereditary disease, owing to the absence of the severe constitutional impression made by the latter. Fournier[3] saw only 1 death in 42 cases.

Diagnosis.—The diagnosis is easy in typical cases with the group of characteristic symptoms. In syphilitic still-born infants, as in those syphilitic infants born prematurely and dying soon after birth, spirochetes may be found in the tissues, there may be syphilitic disease of the placenta, and section of bones reveals typical changes. Jeans and Cooke[4] observed characteristic changes in the placenta in 27 per cent. of syphilitic cases, and only in such. Apart from these features, the clinical diagnosis in the new-born rests principally upon the enlargement of the spleen and liver, coryza, and osteochondritis. Syphilitic osseous lesions can be demonstrated by the *x*-ray. In early infancy the most representative symptoms are snuffling, hoarseness, cutaneous eruptions, fissures about the mouth and anus, and the debilitated condition present from birth or developing later. Relapses are characterized by return of symptoms or the development of condylomata and sometimes of visceral involvement. Among the much later diagnostic symptoms of hereditary syphilis the most important are the alteration of the teeth; the development of gummata in various regions, especially the cranium and the tibiæ; interstitial keratitis; deafness of central origin; depression of the bridge of the nose from destructive lesions: ulcerations of the interior of the nose or of the palate; enlargement of the lymphatic glands; thickening and curvature of the tibiæ, decided hyperplasia of the liver and spleen, and general retarded development.

In mild cases of syphilis where no characteristic group of symptoms is present, the diagnosis apart from serum-tests is difficult. The known presence of syphilis in the parents, or the occurrence of repeated earlier abortions may confirm the importance of a few suspicious symptoms in the infant. So, too, response to antisyphilitic treatment is important diagnostically.

Cutaneous symptoms may be very little marked in the syphilitic cases and, when present, are carefully to be distinguished from various non-specific affections of the skin. The maculo-papular cutaneous eruptions of syphilis differ from those not syphilitic chiefly in their situation and their color. In syphilis they are principally found on the face and the lower extremities, and exhibit always a tendency to a coppery or raw-ham tint. The diffuse syphilitic infiltration sometimes resembles eczema intertrigo, but is distinguished by the absence of the intense red color of the latter and the presence of a coppery or yellowish-brown tint and a tendency to scaling, combined with the evidence of infiltration of the skin which produces a decided hardness and stiffness. Syphilitic pemphigus is situated on an indurated base and generally occupies the soles and the palms, thus distinguishing it from other pemphigoid eruptions in the new-born. The pustular and ulcerated cutaneous lesions of syphilis may suggest those of tuberculosis. The latter, however, are without the dusky-red color of syphilitic lesions, show less crusting, and the edges are often undermined.

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[1] Lues hereditaria tarda, 1887. Ref., Gerhardt's Handb. d. Kinderkr.; Syphilis, 389.

[2] Ergebn. d. inn. Med. u. Kinderheilk., 1910, V, 125

[3] La syphilis héréditaire tardive, 1886, 600.

[4] Amer. Jour. Dis. Child., 1921, XXII, 402.

Coryza in the early weeks or months of infancy, without fever and not yielding to ordinary forms of treatment, is suspicious, even though no other symptoms are discoverable. Infantile scurvy may be mistaken for the osteo-chondritis of syphilis. In the latter, however, the lesion is nearer the epiphysis, and the process usually begins at an earlier age. Meningitis, hydrocephalus, and other manifestations of syphilitic involvement of the central nervous system can be distinguished from non-syphilitic disease by the history, the association with other syphilitic symptoms, and the Wassermann reaction on the blood and spinal fluid.

The value of the complement-fixation reaction in the diagnosis of syphilis, as applied by Wassermann, Neisser and Bruck[1] and as modified by others, has been supported by the testimony of numerous later investigators. It would appear that although a positive Wassermann reaction is of very great importance a negative result is of decidedly less value. Symmers and Darlington[2] after a long series of observations concluded that a negative reaction was present in from 31 to 56 per cent. of cases with characteristic post-mortem lesions of syphilis, and a positive reaction in at least 30 per cent. of cases without syphilitic lesions found at autopsy. (See review by Towle.)[3] There are several limitations to the Wassermann test in early life. It may be negative in the first weeks. This was true in 37 per cent. of the cases of Cooke and Jeans,[4] the reaction always becoming positive later if the infant was syphilitic. On the other hand, a positive reaction in the 1st month is not absolute proof that an infant is syphilitic, the antibodies or substances responsible for the reaction having passed from mother to fetus, but the spirochetes not being transmitted (Findlay).[5] When for any reason the test cannot be applied to the infant, it may be made upon the mother, a positive reaction on her blood usually indicating that a similar reaction would occur in her offspring. Yet this is not always the case, since a healthy infant is occasionally born to a syphilitic mother (See p. 657). In many institutions it is customary to take blood for the test from the placental end of the umbilical cord at birth. This is not a dependable procedure according to some (Kilduffe)[6] but Jeans and Cooke[7] found that the cord-blood responded positively in 63.6 per cent. of cases later proved to be syphilitic, and that it was never positive in non-syphilitic cases. While positive reactions may be obtained at times on breast-milk and urine and other fluids (Klauder and Kolmer;[8] Cotellessa[9]), these are not to be relied upon. When the Wassermann test was first used it was found to be positive at times in other conditions than syphilis; as, for instance, in tuberculosis, scarlet fever, malaria, leprosy, cachectic states and certain cutaneous disorders. Since improvement in the technique the number of these positive results has decreased, although consideration must still be given to the possibility. In other words, the Wassermann reaction must be looked upon as corroborative evidence, and it must be considered in conjunction with the clinical symptoms. In the presence of symptoms, for example, treatment should be instituted in the face of a negative reaction.

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[1] Deutsch. med. Wochenschr., 1906, XXXII, 745.
[2] Jour. Amer. Med. Assoc., 1918, LXX, 279.
[3] Amer. Jour. Dis. Child., 1912, IV, 180; 1914, VII, 318.
[4] Amer. Jour. Syph., 1922, VI, 569.
[5] Amer. Jour. Dis. Child., 1924, XXVIII, 133.
[6] Amer. Jour. Med. Sci., 1922, CLXIV, 677.
[7] Amer. Jour. Dis. Child., 1921, XXII, 402.
[8] Jour. Amer. Med. Assoc., 1921, LXXVI, 1635.
[9] La Pediat., 1923, XXXI, 860.

The exact value of the Noguchi[1] luetin test is still under dispute, and further study is required. Luetin is a suspension of dead spirochaetae pallidæ. It is injected intradermally in amounts of 0.07 c.c. (.003 fl. oz.). A positive reaction is indicated by the appearance at the site of injection in 24 to 48 hours of a raised papule 5 to 10 mm. broad, (0.20 to 0.39 in.), surrounded by a red areola and occasionally with a pustular top. The papule becomes indurated, this lasting for about a week. Control tests are necessary especially in view of the fact that in syphilis non-specific cutaneous sensitiveness is increased (Kolmer and Greenbaum).[2] There is good evidence that the test is helpful. Meyers[3] thinks it is of more value than the Wassermann in hereditary syphilis, and that it is positive in 65 per cent. of cases. DeVilla and Ronchi[4] performed the test on 50 non-syphilitic children, all of whom gave no reaction. In 79 syphilitic children it was positive in 48.1 per cent.; and while the Wassermann reaction was present in 81 per cent. it was found that under 1 year of age the luetin reaction was more likely to be positive. Kolmer[5] thinks a positive luetin reaction may be accepted even though the Wassermann is negative.

Among other tests for syphilis should be mentioned especially the Kahn[6] precipitation-test, the specificity of which is confirmed by many workers, some of whom prefer it to the Wassermann reaction.

Use may be made of the Lange[7] colloidal gold test on the spinal fluid. In syphilis there may be a reaction in the lower dilutions (1 : 40 and 1 : 80). To this there are exceptions, and there is no regular agreement with the blood-Wassermann (Grulee).[8]

The differentiation of the hereditary from the acquired form depends principally upon the early occurrence of visceral lesions and the existence of epiphysitis, pseudoparalysis, fissures, coryza, pemphigus, diffuse infiltration of the skin, or other symptoms characteristic of the inherited disease but not seen in acquired syphilis. In acquired syphilis, on the other hand, there occur a primary lesion, visceral involvement later in the attack and less frequently, and roseola.

Treatment. **Prophylaxis.**—According to the advice given by Fournier[9] a syphilitic adult should undergo energetic treatment more or less continuously for from 3 to 4 years after the onset of the disease, and for 2 years there should have been no symptom whatever discoverable. Marriage may now take place, although there is even then no absolute certainty that the disease will not be transmitted to the offspring. If a woman becomes pregnant and her husband is syphilitic, she should at once undergo continuous treatment throughout pregnancy, whether or not she herself exhibits symptoms. If she has formerly shown evidences of syphilis, but appears to have recovered, she should still receive treatment. It is probable that by these means the infant will escape the disease. If the mother's Wassermann reaction can be made negative, there is a good chance that her infant may be born free from the disease. Pregnancy renders a woman more amenable to antisyphilitic treatment, and better results follow, even with imperfect methods, than in the case of syphilitic women who are not pregnant, and of men (Williams).[1]

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[1] Jour. Exper. Med., 1911, XIV, 557.

[2] Jour. Amer. Med. Assoc., 1922, LXXIX, 2063.

[3] Arch. of Pediat., 1920, XXXVII, 223.

[4] La Pediat., 1923, XXXI, 938.

[5] Amer. Jour. Dis. Child., 1920, XIX, 344.

[6] Serum Diagnosis of Syphilis by Precipitation, 1925.

[7] Zeitschr. f. Chemotherap. u. verwandte Gebiete, 1913, Orig., I, 44.

[8] Amer. Jour. Dis. Child., 1924, XXVIII, 147.

[9] Syphilis et mariage, 1880, 108; 115.

All healthy infants should be guarded with care against the acquiring of postnatal syphilis especially from a syphilitic wet-nurse, and a thorough examination of the nurse should be insisted upon, including an examination for the Wassermann reaction. It is probable that an apparently healthy infant, born of a syphilitic mother may be suckled by her without danger of infection (Profeta's Law). At the same time, in view of the difference of opinion still existing regarding this, the immunity of the infant should not be trusted absolutely, and the mother should receive treatment and should avoid caressing the infant or other unnecessary contact with it. If the reaction of the infant is negative, it is better that it be not nursed by the positively syphilitic mother. Promiscuous kissing of the baby by friends or strangers is also to be avoided.

Treatment of the Disease.—Immediately after birth in the case of infants whose parents are known to be syphilitic; or, in other cases, on the first evidence of the disease in the infant; treatment with mercury should be commenced. The drug may be administered in various ways. A popular method is that of rubbing into the skin daily 5 to 10 grains (0.32 to 0.65) of mercurial ointment, selecting different situations upon different days, as the axilla, abdomen, groin, thigh, etc., in order to avoid the production of cutaneous irritation. A favorite method is to place the ointment upon the binder, and thus keep it in contact with the abdomen. The skin should be washed with soap and warm water before every inunction, and the ointment gently rubbed in for 10 or 15 minutes. In order also to avoid irritation, if the skin is the seat of any extensive cutaneous eruption, the ointment may be replaced by the sublimate bath, using 5 to 15 grains (0.32 to 0.97) of the bichloride of mercury to each bath of 5 gallons (19 liters) given daily. The water should be heated to from 100° to 105°F. (37.8° to 40.6°C.) and the child should remain in it for from 5 to 15 minutes.

Instead of these methods, or in addition to them, mercury may be given internally, either as calomel, $\frac{1}{10}$ of a grain (0.0065); the protiodide, $\frac{1}{20}$ to $\frac{1}{10}$ of a grain (0.0033 to 0.0065); hydrargyrum cum creta, 1 to 2 grains (0.065 to 0.13) or corrosive sublimate, $\frac{1}{100}$ to $\frac{1}{50}$ of a grain, (0.0006 to 0.0013) 3 or 4 times a day. If the mercury produces diarrhea, its administration should be stopped for a few days or a very small amount of opium given. Corrosive sublimate is rather more prone than the other preparations to occasion digestive disturbance. Salivation rarely occurs in early infancy, and less frequently in children than in adults. Mercury may also be administered hypodermically in the form of the chloride or salicylate. These salts have the advantage over calomel or grey oil in that they are much more rapidly absorbed. Hypodermic medication may be combined with oral administration, or may be given alone. In the first method an injection is given once a week, and in the second about 2 or 3 times a week. The usual dose of mercuric chloride is 1/10 to 1/8 of a grain (0.006 to 0.008) for small infants and larger for older ones.

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[1] Johns Hopk. Hosp. Bull., 1922, XXXIII, 383.

Mercurial treatment to be effective and continue so must be persisted with for 1 or 2 years, if it does not produce stomatitis or anemia and debility. If any such results develop it should be stopped for a time. In any event it is often better to allow brief periods of intermission to occur, and it is not necessary to employ the treatment so energetically during the whole of the period. Treatment should be given at intervals during the succeeding years of infancy and early childhood, and again at puberty, in order to insure a continued absence of symptoms. In young syphilitic infants mercury usually seems to have a tonic effect and, in addition to its specific action, causes an improvement in the nutritional state. In older children there is sometimes a loss of weight during mercurial treatment.

Arsenic in some of its forms (salvarsan, neosalvarsan, arsphenamine, sulpharsphenamine, neoarsphenamine, silver arsphenamine, arsnobenzol, sulpharsenol, etc.) is a recognized adjunct in the treatment of syphilis at all ages. Although its action is often more rapid, it has not supplanted mercury, and the best results are obtained when both drugs are used. Death may occur within 24 hours after arsenical injections. This has been attributed to the toxic action of arsenic, and also to the death of large numbers of spirochetes in the tissues. It is especially liable to happen when too large doses of arsenic are given to a patient in an active stage of syphilis. Arsenic has little effect when given by mouth or rectum, and in most of its forms is very irritating to the tissues if administered subcutaneously or intramuscularly. Boone and Weech,[1] however, report good results with intramuscular injections of sulpharsphenamine in doses as large as 0.02 gram per kilogram of body-weight (0.14 grain per pound) given weekly. Neoarsphenamine also may be given intramuscularly, and has even been introduced by the intraperitoneal route (Rosenberg).[2] Its strength is about 2/3 that of arsphenamine. As a rule neoarsphenamine seems to be best fitted for general use in infancy. In older children the intravenous administration is usually preferred and the superficial veins selected should be those of the scalp, the arm, the leg or the external jugular. The longitudinal sinus is a dangerous place to introduce the drug. We have seen 3 infants in whom localized necrosis of the brain followed such a procedure. There is a wide difference of opinion regarding the size of the dose. The method of Jeans[3] is a satisfactory one. Every 2 months a course is given of 3 intravenous injections of arsphenamine at weekly intervals. The dose employed is 0.01 gram per kilogram of body-weight (0.069 grain per pound). During this period treatment by mercury is not stopped. Other combinations of mercurial and arsenical treatments may be used. Thus a course of 6 injections of an arsenical preparation at weekly intervals may be alternated with 6 weeks of treatment by mercury; or an intramuscular injection of mercuric chloride may alternate with an intramuscular or intravenous injection of an arsenical, a period of 3 to 5 days intervening between each treatment.

If there is evidence of neurosyphilis and medication, such as has been outlined, does not produce results, intraspinal treatment after the method of Swift and Ellis[1] may be employed. This consists in withdrawing blood from the patient 1 hour after the intravenous injection of arsphenamine, inactivating the serum at 56°C. (132.8°F.) for ½ hour and then injecting from 5 to 15 c.c. (0.169 to 0.507 fl.oz.) of the serum intraspinally. The result sought is the production of a negative Wassermann reaction, and the absence of globulin or any other evidence of pathological condition in the spinal fluid. Two or 3 courses of intraspinal treatment given at the time of intravenous arsphenamine injections may be needed. In this form of the disease Wile and Wieder[2] recommnd an arsenical preparation known as tryparsamide which has a distinctly trypanocidal effect. It can be administered at weekly intervals intramuscularly or intravenously in doses of 0.04 to 0.05 gm. or less per kilogram of body weight (0.28 to 0.36 grain per pound) and may be combined with mercurial treatment. The drug is contraindicated in disease of the optic nerve.

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[1] Amer. Jour. Dis. Child., 1924, XXVII, 39.

[2] Jour. Amer. Med. Assoc., 1924, LXXXII, 682.

[3] Jour. Amer. Med. Assoc., 1921, LXXVI, 167.

Bismuth has an antispirochetal action of which advantage may be taken when there is intolerance to arsenic. It was first employed extensively for clinical purposes by Fournier and Guénot,[3] after experiments by Sazerac and Levaditi.[4] It has been used as the tartrobismuthate of sodium and potassium in aqueous solution or in olive oil, potassium tartrobismuthate in aqueous solution, or bismuth trioxide in olive oil. Intramuscular injections are administered at intervals of 2 to 4 days, 10 to 12 injections constituting a course. The drug has been little used in children. A young infant should not receive more than 0.01 gm. (0.15 grain) of bismuth at a dose. Bismuth may also be given by inunction, using a 50 per cent. ointment in the same manner and dose as with mercury. Symptoms of overdose consist of foul breath, stomatitis, polyuria and albuminuria. A number of other drugs such as flumerin and mercurosal are at present under investigation, all of them having a spirocheticidal value.

Iodide of potassium or of sodium is of service only for the distinctly later manifestations of syphilis, such as occur in later relapses; and at any period when the viscera or the bones are involved. The earlier relapses, with condylomata, require the use of mercury. The employment of the iodides depends upon the nature of the symptoms, irrespective of whether they occur in infancy or after it. They may be given to infants in doses of from 2 to 10 grains (0.13 to 0.65) 3 times daily; alone or, often better, combined with the external use of mercury. The combination with mercury is especially indicated in visceral syphilis.

With whatever specific therapy is employed, it is important that it be continued for at least a year in infants, and for 2 years in older children, after the clinical symptoms have disappeared and a negative Wassermann reaction has been obtained. It is, however, often very difficult to accomplish the desired negative response, especially in late hereditary syphilis. In these so-called "Wassermann-fast" cases some clinicians have combined with other treatment intramuscular injections of such foreign proteins as milk (Greenbaum and Wright)[5] or gonococcus-vaccine (Herrold).[6] The results are said to be favorable.

Very important at all periods is treatment of a general tonic nature. In early infancy the general nutrition must be supported or increased

[1] N. Y. Med. Jour., 1912, XCVI, 53.
[2] Jour. Amer. Med. Assoc., 1924, LXXXIII, 1824.
[3] Ann. instit. Pasteur, 1922, XXXVI, 14.
[4] Ann. instit. Pasteur, 1922, XXXVI, 1.
[5] Arch. Dermatol. and Syph., 1924, X, 551.
[6] Jour. Amer. Med. Assoc., 1926, LXXXVI, 413.

by suitable nourishment, especially by the use of human milk, and by the employment of all hygienic measures calculated to increase the vigor, which so often falls below normal in syphilitic infants. For the anemia and debility which commonly develop among the later symptoms iron and cod-liver oil are of value, and may with benefit replace for a time the administration of mercury. For such cases, too, exposure to fresh air and sunshine and the employment of similar hygienic measures, with abundant nourishment, are especially important.

Local Treatment.—Many of the lesions require local measures to hasten their cure. Condylomata and cutaneous ulcerations and fissures may be dusted with calomel, covered with mercurial ointment, or painted with a solution of nitrate of silver. Ulcers on the mucous membranes require careful cleansing followed by the application of calomel or, occasionally, nitrate of silver. Gummatous ulcerations are well treated with iodoform or mercurial applications. The coryza is often benefited by removing crusts from the nose, followed by the employment of a diluted yellow oxide of mercury ointment or by the insufflation of calomel well-diluted with sugar (1:20).

SECTION III

GENERAL NUTRITIONAL AND MISCELLANEOUS DISEASES

Here may be classified a number of disorders the cause and nature of which is not clearly understood, or which seem to belong properly to no other category. Of these rheumatism is now very frequently placed among the acute infectious disorders; yet, inasmuch as its position here is not yet positively proven, and on account of its relationship to certain other affections, it has been thought best to place it provisionally in the class now to be considered. Rachitis, scorbutus and some forms of malnutrition and infantile atrophy are best classified as nutritional disorders, and may be placed here; as may also be diabetes, certain of the diatheses, acidosis and, probably, pellagra. Obesity, which could properly be considered in this connection, will be discussed under Diseases of the Internal Secretions, since many cases depend upon these.

CHAPTER I

RACHITIS

(Rickets)

Evidences of rickets are found in the skeletons of children of the stone age, according to Kjerrulk,[1] and rickets also existed in ancient Greece and was frequent among the Romans. It is uncertain whether it was present in ancient Egypt (Findlay).[2] The disease was first named Rachitis and clearly described by Glisson.[3] It is very widely spread in civilized countries, although its prevalence varies greatly with the locality. A large proportion of infants brought to the out-patient departments of hospitals in many of the larger cities of Europe and America exhibit the disease. Morse[4] found it in 79.5 per cent. of 400 infants in Boston, and Schmorl,[5] in Dresden, evidences of the disease at autopsy in 89.4 per cent. of 345 children from 2 months to 4 years of age. Practically all (96.6 per cent.) of those from 4 to 18 months exhibited the disease. It was estimated to be present in 95 per cent. of the children in Hamburg (Volland);[6] 74.9 per cent. of those of the poorer classes in Dundee (Foggie);[7] 86 per cent. of infants in Riga (Mey),[8] but in only 13.5 per cent. in Christiania (Quisling).[1] Without quoting the numerous available statistics it may be said that 50 per cent. or more of the children of the poor of large cities in the United States suffer from rickets. The percentage is larger in northern than in southern cities. It is essentially a chronic disorder of nutrition, affecting the bones most strikingly; but general in its nature and involving as well the ligaments, muscles, mucous membranes, nervous system and other parts of the body.

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[1] Hygiea, 1923, LXXXIV, 530.

[2] Glasgow Med. Jour., 1919, XCI, 147.

[3] Tractatus de rachitide, etc., Lond., 1650. Ref., Rehn. Gerhardt's Handb. d. Kinderk., III, 1, 43.

[4] Jour. Amer. Med. Assoc., 1900, XXXIV, 724.

[5] Ergebn. d. inn. Med. u. Kinderheilk, 1909, IV, 437.

[6] Jahrb. f. Kinderheilh, 1885, XXII, 118.

[7] Scottish Med. and Surg. Journ., 1905, XVI, 231.

[8] Jahrb. f. Kinderheilk, 1896, XLII, 273.

Etiology.—This is not known with certainty, but there are factors quite well understood which are closely connected with etiology and pathogenesis, and a knowledge of which can be utilized in prevention and cure. To mention[2] Hess and Unger, who have had associated with them at various times Pappenheimer, McCann, Weinstock, Lundagen, Gutman, Steiner, Tolstoi, Calvin, Wang, and Felcher; McCollum, Park, Powers, Shipley and Simmonds, working together or separately;[3] Howland and Kramer,[4] Sherman and Pappenheimer;[5] Pappenheimer, McCann and Zuckner[6] is to give only a partial list of investigators to whose work some degree of acknowledgment is made in this connection. Excellent reviews have been published by Hess,[7] Howland[8] and Park.[9]

Many influences appear to possess some etiological relationship, and all of these are intimately connected with 3 factors: (1) light, (2) proper balance of calcium and phosphorus in the diet, and (2) certain agencies present in cod liver oil and other substances which have to do with the calcification of bone. The significance and the possible mode of action of these factors will be discussed under Pathogenesis.

Diet has a decided influence. Clinical evidence of the disease is much less frequent and much less severe in breast-fed children, unless nursing is unduly prolonged. Maternal feeding, however, by no means confers immunity, milder degrees of rickets being often easily recognized in apparently thriving breast-fed infants; and in Eliot's[10] studies the *x*-ray has shown evidence of it with great regularity. It is generally admitted, too, that infants fed upon condensed milk or other proprietary food having an excess of carbohydrate and an insufficient amount of fat and protein are especially prone to rickets. Yet rickets may follow overfeeding as well as under-feeding. A very great deficiency in the amount of food leads to wasting without the development of rachitis; and, on the other hand, it is not infrequent that babies become rachitic who have been fed upon breast-milk apparently of the best quality as far as fat, carbohydrate and protein are concerned. This is especially true of the Italians and Negroes in the northern cities of the United States. It will be shown in the section on Pathogenesis that the dietetic deficiency does not lie so much in the amounts of the elements mentioned as it does in the improper balance of salts. That diet is the only factor, or that it often acts alone, is not the case, because animals and infants can be prevented from acquiring rickets or even cured without change in the rickets-producing diet, when they are exposed to ultra-violet rays or when cod liver oil is administered. Yet the increase of late rickets in the World War is very suggestive of the influence of improper diet.

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[1] Arch. f. Kinderheilk, 1888, IX, 293.

[2] Jour. Amer. Med. Assoc., 1921, LXXVII, 39; 1922, LXXVIII, 29; 1579; 1596; 1922, LXXIX, 2210; 1923, LXXX, 687; 1923, LXXXI, 15; Amer. Jour. Dis. Child., 1923, XXVI, 271; 1924, XXVII, 1; Jour. Biol. Chem., 1921, XLVII, 395; 1923, LVII, 731; Jour. Exper. Med., 1922, XXXVI, 427; 447; Proc. Soc. Exp. Biol. and Med., 1921, XIX, 8.

[3] Jour. Biol. Chem., 1921, XLV, 333; 343; 1921, XLVII, 507; 1922, L, 5; Amer. Jour. Dis. Child., 1922, XXIII, 91; Johns Hopk. Hosp. Bull., 1922, XXXIII, 31; 125.

[4] Amer. Jour. Dis. Child., 1921, XXII, 105.

[5] Jour. Exp. Med., 1921, XXXIV, 189.

[6] Jour. Exp. Med., 1922, XXXV, 421.

[7] Jour. Amer. Med. Assoc., 1922, LXXVIII, 1177.

[8] Medicine, 1923, II, 349.

[9] Phys. Rev., 1923, III, 106.

[10] Jour. Amer. Med. Assoc., 1925, LXXXV, 656.

Hygienic conditions in general have been known for many years to be important, and such matters as over-crowding and lack of exercise and sunlight have been grouped together as causes in determining the preponderance of the disease in cities as compared with rural districts. Poverty and its attendant circumstances certainly have an effect; but, on the other hand, rickets is common, although generally less severe, among the well-to-do, where at first sight the diet, and apparently the hygienic surroundings, have been carefully supervised. Analysis will show that it is lack of sunlight that seems to be the prominent and real factor in so-called poor hygiene. Proper exposure to the sun will largely eliminate the effect of poor diet and uncleanliness. The action of sunlight is diminished by the smoke of cities; this helping to explain in part the incidence of rickets there. Findlay[1] has insisted that confinement and lack of exercise have an influence independent of lack of sunlight.

Season is a factor, active manifestations of the disorder being less frequent during the summer, and sufferers from it improving at this time. This common clinical observation has been confirmed by the autopsy statistics of Schmorl[2] upon 281 cases of rickets. The microscopic study of the bones in his cases showed evidences of a beginning rachitic process in comparatively few instances during the summer, while the proportion of those with evidences of healing was at its maximum at this time.

Geographical distribution and *climate* are likewise factors. The disease is especially frequent in northern temperate climates, less common in subtropical countries, and rare in the tropics and far north. In England, Austria, Northern Italy, the Netherlands and Germany it is very common, while it is much less so in Japan, Greenland, Turkey, Denmark, Norway, Greece, Southern Italy and Southern Spain (Palm).[3] It has been considered to be rare in China, but recent reports (Tso;[4] Suzuki[5]) do not indicate that this is the case. It is less common at high altitudes than at low elevations in the same country. The element of light enters into the influence of season, geographical distribution and climate. In the winter-time in the temperate zone there is the natural tendency to increase the amount of clothing and, for the sake of warmth, to spend more time indoors; the hours of sunlight are less than in summer; and, not only that, but the short ultra-violet rays, which are the ones concerned in the prevention and cure of rickets, are less numerous. In other words, in winter a less efficient sun has less chance to do its work. In warmer climates there is increased efficiency of the sun and a greater opportunity for its action because of lighter clothing and increased exposure. The infrequency of rickets in countries of higher latitudes must be explained on some other grounds than protection by ultra-violet light, and illustrates the interdependence of the several factors concerned in the causation of the disease. It seems reasonable to suppose that, where there is the lack of the beneficial action of the sun's rays, it is the diet which is protective—a diet which contains large amounts of fat of a type rich in the antirachitic factor. Breast-feeding, too, has its determining influence in certain countries. The greater number of ultra-violet rays present in the sunlight of high altitudes as well as the lack of the filtering action of moisture on these rays will explain the effect of mountains in prevention and cure.

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[1] Lancet, 1922, I, 825.

[2] *Loc. cit.* 439.

[3] Practitioner, 1890, XLV, 270.

[4] China Med. Jour., 1924, XXXVIII, 112.

[5] Jour. Orient. Med., 1924, II, 142, Ref. Jour. Amer. Med. Assoc., 1924, LXXXIII, 1035.

Apart from locality, *race* seems to exert a powerful influence under certain conditions. Rickets is particularly prevalent in races from warm regions transported to colder ones. Thus among the Italians and Negroes of the northern cities of the United States nearly every infant exhibits more or less rickets, while in the Southern United States it is far less common among the Negroes, and rare in these in the tropics, and it is not frequent in Southern Italy. This racial prevalence in northern cities cannot be the result of improper dietetic conditions, for these are in no way inferior to those active among the poor immigrants from other countries, and may be due to the fact that in their new and colder environment they are less exposed to sunlight. It is probable, too, that the darker-skinned races of the south need more sunlight to protect them than do those of lighter skins. It has been shown that pigmented skin filters out the ultra-violet ray, and that the darker the skin the more the rays are lost in this way (Sambon;[1] Hess[2]). It has been thought that *heredity* plays a part, and that the tendency to become rachitic, once having developed, is readily perpetuated and increased by inheritance. Siegert[3] especially has emphasized this fact, and we have seen instances which seem to be corroborative; and Zimmern[4] and others have reported cases showing remarkable family tendency. It seems likely, however, that an apparent hereditary influence can be explained on other grounds, such as diet and, particularly, environment. Hess and Weinstock[5] have advanced a strong argument against hereditary influence, namely: that the races that are particularly liable to develop rickets when transported from warmer to colder climates have not suffered from the disease in their native environment.

Age is a factor is important. Rickets is observed especially in the first 2 years of life. It rarely shows itself clinically before the 3d month and generally not until after the 6th month. Baginsky,[6] in 620 cases found symptoms present in 43 from 3 to 6 months of age; 173 from 6 to 12 months; 220 from 12 to 18 months; and 113 from 18 to 24 months. It rarely begins after the 2d year of life. Its height is usually about the end of the 1st or the beginning of the 2d year. Even cases of *fetal rickets* have repeatedly been reported, but doubt attaches to most of these. (See Fetal Rickets, p. 702.) So, too, *late rickets* has been described, the symptoms not appearing until later childhood or puberty. The condition is, however, certainly not common and does not differ essentially from osteomalacia. (See Late Rickets, p. 702.) Prematurity especially predisposes to rickets. Two factors enter into this; the first that the premature infant has not the benefit of the large amount of calcium which should have been deposited in the body during the last few weeks of intrauterine life; the second, that the infant seldom receives the protection afforded by sunlight.

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[1] Jour. Trop. Med., 1907, X, 67.

[2] Lancet, 1922, II, 367.

[3] Jahrb. f. Kinderheilk, 1904, LIX, 237.

[4] Nouv. iconog. de la Saltpetrière, 1901, XIV, 299.

[5] Jour. Amer. Med. Assoc., 1924, LXXXIII, 1558.

[6] Prakt. Beiträge z. Kinderheilk., Tübingen, 1882. Ref. Vierordt, Nothnagel's Spec. Path. u. Therap.; Rachitis, VII, 2, 1.

The condition of the parents, apart from the existence of rickets in them already referred to, predisposes to the development of the disease only by the general debilitating effect upon the offspring or through the production of poor breast-milk by the mother. *Sex* exerts no influence of moment. *Previous disease of the infant* has a definite influence. Thus prolonged catarrhal states of the digestive apparatus may be followed by the disease, perhaps through rendering the child unable to profit by food which, in itself, is of good quality; perhaps through favoring the production of some toxic substance in the intestine. Should the digestive disturbances be severe, however, wasting of the child is liable to occur rather than rickets, and, on the whole, digestive disorders are not a very prominent etiological factor. Acute severe disease of any nature appears sometimes to be the starting point for rickets, or to make the symptoms worse if already present.

Pathogenesis.—In the consideration of this subject, facts which have been ascertained by a study of rickets in animals may be applied in certain measure to the solution of the problem in infants. That human rickets and the rickets of rats are the same is the belief of most investigators, although some would deny this (Mouriquand).[1] That the intermediary metabolism in the rat and the human is the same is less certain.

We know that in some way in rickets there occurs a failure of lime-salts to be deposited, and that there is a cessation in the transformation of osteoid tissue into true bone; and it is evident that the agencies, such as cod liver oil, ultra-violet rays, and the like, already mentioned (see Etiology) must be operative in some manner in regulating the mineral metabolism. The demonstration by Stöltzner[2] and by Brubacher[3] that in rickets tissues other than bones possess a normal amount of calcium gave rise to the belief that increased elimination of calcium had little to do with the causation of the disease. The work of Schabad,[4] confirmed by others (Schloss;[5] Jones;[6] Orr, Holt, Wilkins and Boone[7]), showed that a disturbed calcium and phosphorus balance certainly existed, there being an increase of excretion of these minerals in the active stage and a decrease during convalescence. Other studies have taught that the calcium of the blood in uncomplicated rickets is usually normal or only slightly diminished, whereas the amount of phosphorus is constantly lower than normal in the active stages (2.25 to 3.75 mg. per 100 c.c.). The conclusion to be drawn is that, since the blood-calcium is normal but that of the bones decreased, the latter condition is in a sense the result of the disease rather than the cause. That is to say, there exists in rickets an inability of the bones to retain a proper amount of calcium even when sufficient is supplied.

The question to be considered regards the action of the respective diverse factors, such as food, sunlight and cod liver oil, upon the mineral metabolism. First to be studied is the influence of *diet*, and to this there are several confusing angles. Whether or not the breast-milk, received by those nursing infants who develop rickets, is impoverished in salt-content is not certain. This is claimed to be the case by some authors, but denied by others (von Meysenbug).[1] It has been maintained that the breast-milk in colored mothers, whose babies so often suffer from rickets, contains less calcium and phosphorus than that of white mothers (Burhans and Smith);[2] and it is conceivable that the diminished salt-content of breast-milk in long-continued nursing may have an effect (See p. 102.) The influences of rapid growth may also be present in the case of the well-nourished breast-fed infant. Yet the obvious fact, that artificially fed infants are more apt to be the victims of rickets than are the breast-fed, cannot well be explained upon the basis of intake of mineral matter alone, since many artificial foods exceed human milk in the mineral content. Moll[3] has shown that breast-fed infants excrete less phosphorus in the urine than do the bottle-fed. Jones, James and Smith[4] suggest that rickets may develop from too high a degree of alkalinity in the intestinal tract, this favoring phosphorus-elimination. Cow's milk, in contradistinction to breast-milk, would produce such an intestinal alkalinity, since its high buffer-content would neutralize an excess of hydrochloric acid in the stomach. Orr[5] and his associates point out that increased acidity of the intestine favors solution of calcium phosphate and its consequent absorption, and there are other studies, such as those of Telfer[6] and Findlay,[7] which indicate that defective intestinal absorption of mineral matter may be the cause of rickets. A greatly diminished intake of calcium has a distinct influence, but it would appear chiefly or only when there is a disturbed ratio between this and the phosphorus in the diet. In experimental rickets-producing diets in animals two alterations in mineral content are employed; in one the calcium is practically normal but the phosphorus low; in the other the phosphorus normal but the calcium low. Such abnormalities in mineral intake result in poor retention, an increase in one of these minerals in the diet causing a diminished retention of the other. It would be a mistake, however, to disregard here the influence of ingredients of the diet other than minerals, since it is known that other faults in the food have an effect. Thus the low-fat-high-carbohydrate diet seems especially productive of rickets, and this is because of the lack of retention of minerals which such improper feeding causes.

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[1] Paris méd., 1923, XIII, 406.

[2] Jahrb. f. Kinderheilk., 1899, L, 268.

[3] Zeitschr. f. Biol., 1890, XXVII, 517.

[4] Arch. f. Kinderheilk, 1910, LIII, 380; LIV, 83.

[5] Monatsschr. f. Kinderheilk., Orig., 1914, XIII, 271.

[6] Jour. Amer. Med. Assoc., 1924, LXXXII, 429.

[7] Amer. Jour. Dis. Child., 1924, XXVIII, 574.

That athreptic infants do not develop rickets has been explained on the ground that the starving body is given a chance to rectify its abnormal mineral metabolism, when the burden of carrying on the metabolism due to food ingestion is removed.

The effect of overfeeding in producing rickets is not so clear. Jundell[8] offers the explanation, that the overstrain of general nutritive functions leads to diminution of special cell-functions, particularly those of internal secretion, which he believes to be concerned in the causation of rickets; and he notes that cod liver oil combined with overfeeding does not hinder the development of rickets, whereas if combined with relative inanition it does. Overfeeding may also have an effect in causing rapid growth, a condition which predisposes to rickets in animals and probably also in humans. The body's demand for calcium under such conditions may divert this from the osseous system.

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[1] Amer. Jour. Dis. Child., 1922, XXIV, 200.
[2] Amer. Jour. Dis. Child., 1923, XXVI, 303.
[3] Jahrb. f. Kinderheilk, 1909, LXIX, 450.
[4] Proc. Soc. Exp. Biol. and Med., 1923, XXI, 199.
[5] *Loc. cit.*
[6] Quart. Jour. Med., 1922, XVI, 63.
[7] Jour. Amer. Med. Assoc., 1924, LXXXIII, 1473.
[8] Hygiea, 1921, LXXXIII, 753.

The statements quoted above are some of the various explanations regarding the influence of diet. They are to an extent conflicting and certainly confusing. It would appear that the most that can safely be said, in the present state of our knowledge, is that it is not so much the intake of mineral matter as its retention in the bones which is at fault; that a lack of balance between the calcium and the phosphorus in the food has much to do with this failure of retention, but that other dietetic faults than that of the mineral matter are of importance, although the manner of their action is far from being understood. It should always be remembered, in considering the influence of diet in the human infant, that other factors are involved with it, and that it usually does not operate by itself. This is proved by the observation, already made in discussing Etiology, that rickets may develop on a perfectly satisfactory diet properly balanced in fat, carbohydrate, protein and salts, and that on an improper diet, of a nature that is ideal for the production of rickets, the disease may be prevented by exposure to the ultra-violet rays or by the administration of cod liver oil.

The relationship of *sunlight* to rickets is of extreme interest, as previously pointed out. It may be seen in the influence of season, race, climate, altitude, poor hygiene, and probably also of age. It is the short violet rays of the sun under 313 millimicrons in length that possess the antirachitic power, and such rays can also be produced artificially by the mercury-vapor quartz lamp, or by the carbon arc-light. Their effect is distinctly observed both in prevention and in cure. In the latter it is demonstrated not only by clinical improvement but also by the return of the inorganic phosphate of the blood to normal (4.5 to 5 mg. per 100 c.c.), while the local healing (deposition of calcium) can be shown by *x*-ray pictures of the epiphyses. The seasonal tide of blood-phosphates, in which the ebb is in the winter, has to do with lack of sunlight and not with food.

Exactly the same results that are accomplished by ultra-violet radiation; namely, clinical improvement, return of blood-phosphate to normal and deposition of calcium in the epiphyses, can be brought about by the administration of *cod liver oil*, and to a less extent by other substances such as egg-yolk and butter. Cod liver oil will likewise prevent a faulty diet from producing a rachitic condition. It is curious that two such different things as sunlight and cod liver oil should act in the same manner, and it leads to the thought that the dissimularity may not be so great as it appears. It has, in fact, been claimed (Kugelmass and McQuarrie)[1] that cod liver oil emits ultra-violet radiation. This has been denied by others (Schlutz and Morse);[2] but its confirmation would explain its effectiveness, and would be in accord with the fact that rations, which would be provocative of rickets in rats, can be exposed to the ultra-violet radiation of the mercury-vapor quartz lamp and rendered antirachitic (Steenbock and Black).[3] Contrary to the opinion of Mellanby,[4] it is probable that the antirachitic property in cod liver oil

[1] Science, 1924, LX, 272.

[2] Amer. Jour. Dis. Child., 1925, XXX, 199.

[3] Jour. Biol. Chem., 1924, LXI, 405.

[4] Lancet, 1920, I, 856.

does not bear any relation to the fat-soluble vitamin. Zucker[1] and Dubin[2] found the active principle only in the non-saponifiable portion. Later studies have shown that lack of the vitamin leads to osteoporosis rather than to true rachitic changes. It has also been demonstrated that prolonged heat and oxidation,—processes which destroy the growth-promoting properties (vitamin) of cod liver oil—do not render it incapable of protection against rickets (McCollum, Simmonds and Baker;[3] Wagner and Wimberger[4]).

While the effects of radiant energy and of cod liver oil are well appreciated, it is not certain how these are produced. Park, Guy and Powers[5] do not believe that these agents cause the body to bring into action new or unusual properties, but rather that they restore to the organism normal and ordinary powers which have been in abeyance. There are some investigators, as Stöltzner,[6] Lehnerdt and Weinberg,[7] Sweet,[8] Jundell,[9] Aschenheim,[10] and Vollmer[11] who uphold the view that the thymus, adrenals or other organs of internal secretion influence the pathogenesis of rickets. It may be that the external factors of diet, light and cod liver oil act upon the endocrine system in such a way as to influence mineral metabolism. The parathyroids must be thought of in this connection, and it is at least true that they are large in rickets, due to multiplication of cells (Pappenheimer and Minor).[12] Lukacs[13] claims that there is increased secretion of trypsin in rickets and that this may possibly bear a relationship to some of the symptoms.

Other theories regarding the etiology of rickets have been held from time to time. One of these is the belief in the influence of infection, but all the older experiments which would support such a theory did not allow sufficiently for the factors of diet and light. Pritchard[14] has insisted that the central feature of rickets is decalcification of bone dependent upon withdrawal of alkalies to neutralize acid-bodies in the blood. This theory is opposed to that of the great majority of students of the disease; viz: that the fault lies rather in a failure of minerals to be deposited than in a demineralization after deposition. Hesse, Weinstock and Helman[15] have shown that the administration of cholesterol or phytosterol to animals protects against rickets but only if irradiated; and Bosanyi[16] claims that cystin, adrenaline, pilocarpin and the globin of hemoglobin have antirachitic properties.

Pathological Anatomy. Lesions of the Bones.—The principal anatomical lesions are in the osseous system. Macroscopically the bones are softer and more flexible than normal, the degree to which they may be bent being remarkable in severe cases. All the bones of the body are affected but the changes are especially marked in the long bones. There is enlargement at all the epiphyseal junctions, but most decided at the wrists and ankles and at the costochondral articulations (Figs. 201, 202 and 203), the thickening in the latter position being always much more evident on the pleural surface. Moderate thickening of the shaft of the long bones also takes place and all the distinctions of outline marked by angles, ridges and the like, are less marked than in normal bones. There is also thickening of the flat bones in certain regions, with the production, especially upon the skull, of large bosses; while in other portions of the cranium spots of very thin, parchment-like bone may be found (craniotabes). (See Vol. II, p. 728.) These alterations produce deformities of many sorts, and fractures are common (Fig. 204). The degree of deformity varies greatly, depending upon the severity of the case. Arrested growth in the length of the bones may occur as a result of the changes in the epiphyses.

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[1] Proc. Soc. Exper. Biol. and Med., 1922–23, XX, 136.

[2] Amer. Jour. Dis. Child., 1925, XXX, 72.

[3] Jour. Biol. Chem., 1922, LIII, 293.

[4] Lancet, 1924, II, 55.

[5] Amer. Jour. Dis. Child., 1923, XXVI, 103.

[6] Münch. med. Wochenschr., 1921, LXVIII, 1481.

[7] Münch. med. Wochenschr., 1921, LXVIII, 1482.

[8] Brit. Med. Jour., 1921, II, 1067.

[9] *Loc. cit.*

[10] Deutsch. med. Wochenschr., 1923, XLIX, 85.

[11] Jahrb. f. Kinderheilk, 1922, XCIX, 133.

[12] Jour. Med. Research, 1921, XLII, 391.

[13] Amer. Jour. Dis. Child., 1926, XXXI, 235.

[14] Brit. Med. Jour., 1919, II, 627.

[15] Jour. Biol. Chem., 1925, LXIII, 305; Jour. Amer. Med. Assoc., 1925, LXXXIV, 1910.

[16] Johns Hopk. Hosp. Bull., 1926, XXXVIII, 72.

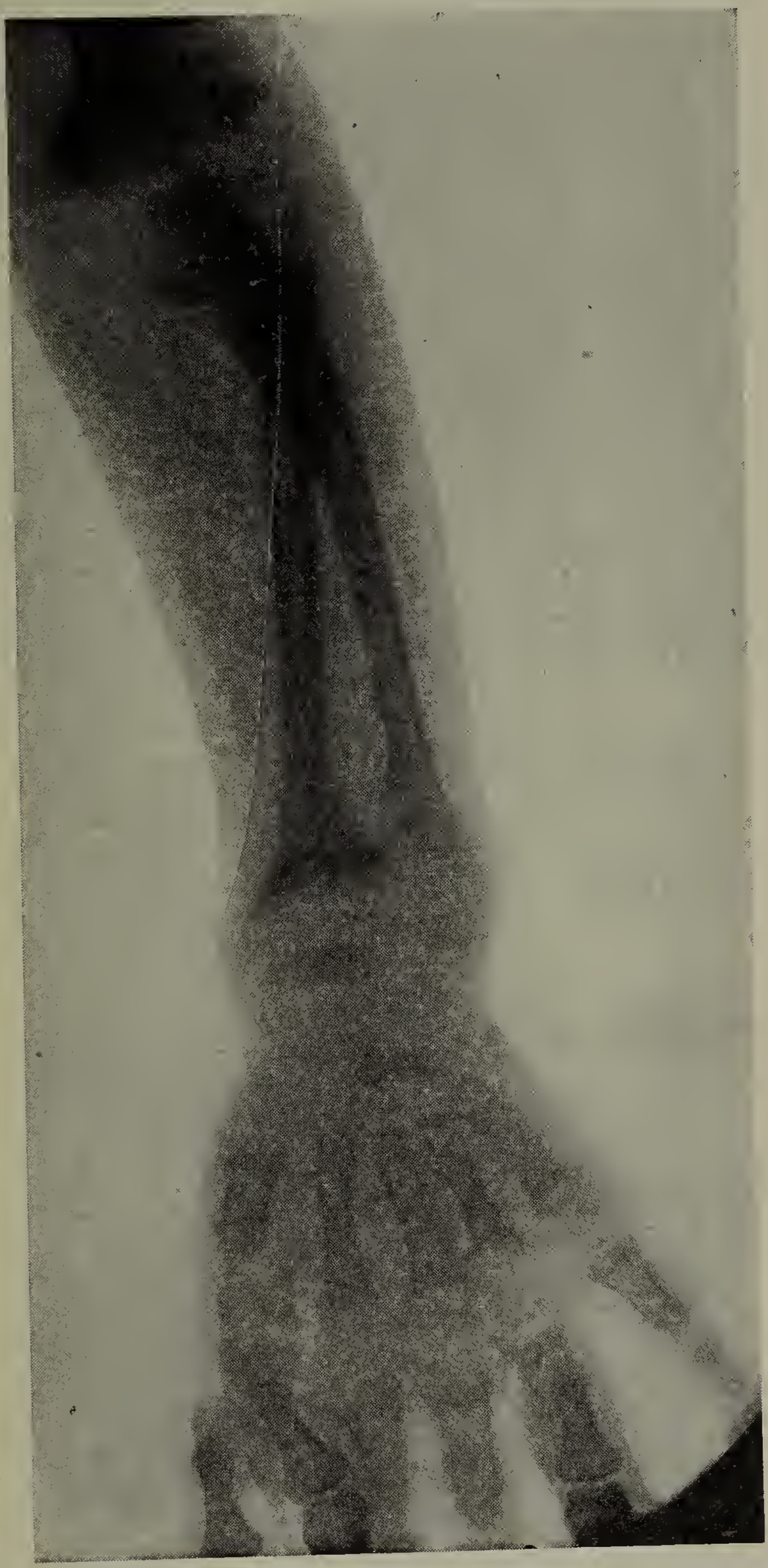

Fig. 201.—Radiograph of the Wrist in Rickets.

Shows the enlargement at the wrist-joint. From a colored child, aged 2 years and 9 months, in the Children's Hospital of Philadelphia.

Longitudinal section shows the junction of the shaft and the epiphyses much wider, thicker and softer than normal (Fig. 207) with a broadening of the bluish, cartilaginous layer. The centres of ossification are hyperemic, spongy and enlarged. It is to this and to the general widening of the cartilaginous layer that the enlargement of the epiphysis is due.

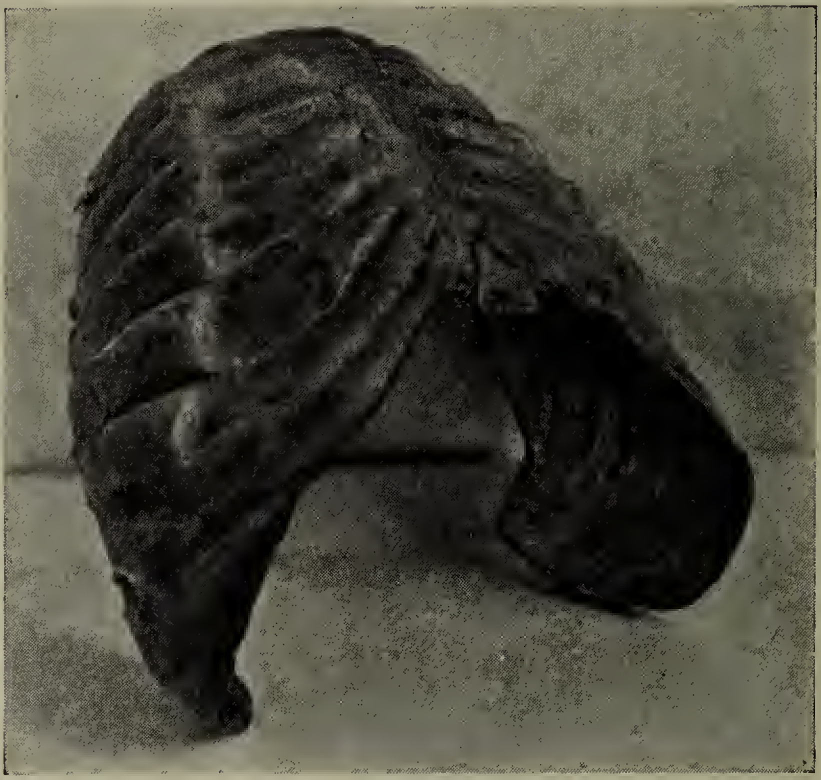

Fig. 202.—Anatomical Specimen of Rachitic Chest.
Shows the rachitic rosary produced by enlargement at the costo-chondral articulations.

The medulla of the shaft is redder and more jelly-like than normal and the medullary cavity broadened and its bony divisions rarefied. The periosteum of both the flat and long bones is hyperemic, and beneath it are several layers of friable, spongy, vascular tissue, with large intertrabecular spaces.

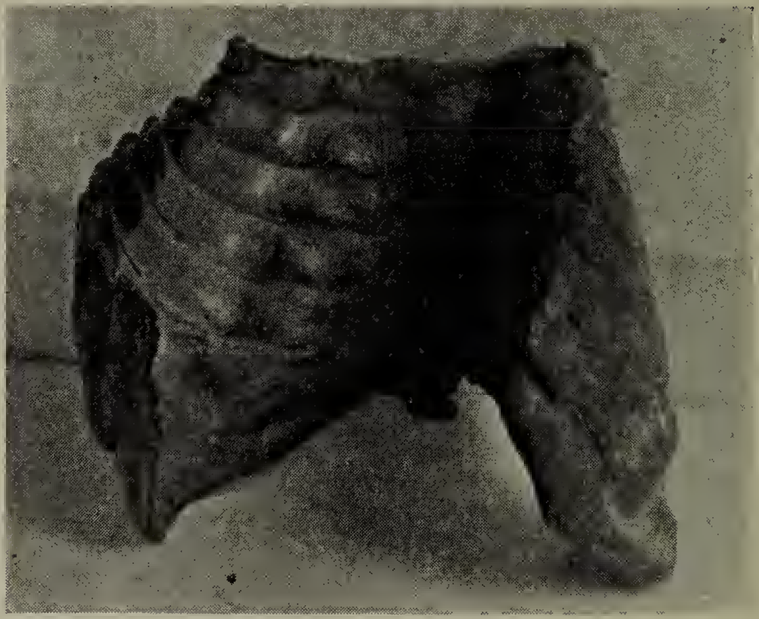

Fig. 203.—Rachitic Chest Viewed from the Inside.
Same case as in Fig. 202.

Microscopically there are very characteristic changes seen. The proliferating layer of the epiphyseal cartilage, situated next to the hyaline cartilage, is hyperemic and the cartilage cells more numerous than normal and irregularly arranged. The columnar zone next to it is still more affected in the same way, being abnormally wide and exhibiting an unusual length of columns of cells and increase of vascularity. This is not due to an abnormal production of cartilage but to a failure of it to be transformed into bone in the normal manner (Schmorl).[1] The layers of cartilage are transversed irregularly by canals containing much-dilated blood-vessels and soft, vascular, imperfectly formed "osteoid" tissue, deficient in lime-salts. The zones of calcification and, last, of ossification, are broader than in healthy bone and have lost their normal, sharply-defined outlines, exhibiting instead calcified areas irregularly mingled with others still cartilaginous, and with scattered, unusually large medullary spaces containing osteoid tissue. This disappearance of the sharply-defined junctions of the zones, as seen in the normal epiphysis, with the irregular inroads of one into the other, is one of the most striking characteristics of rickets. The spongy portion of the shaft exhibits increased vascularity, with erosion of the trabeculæ, resulting in large, medullary spaces.

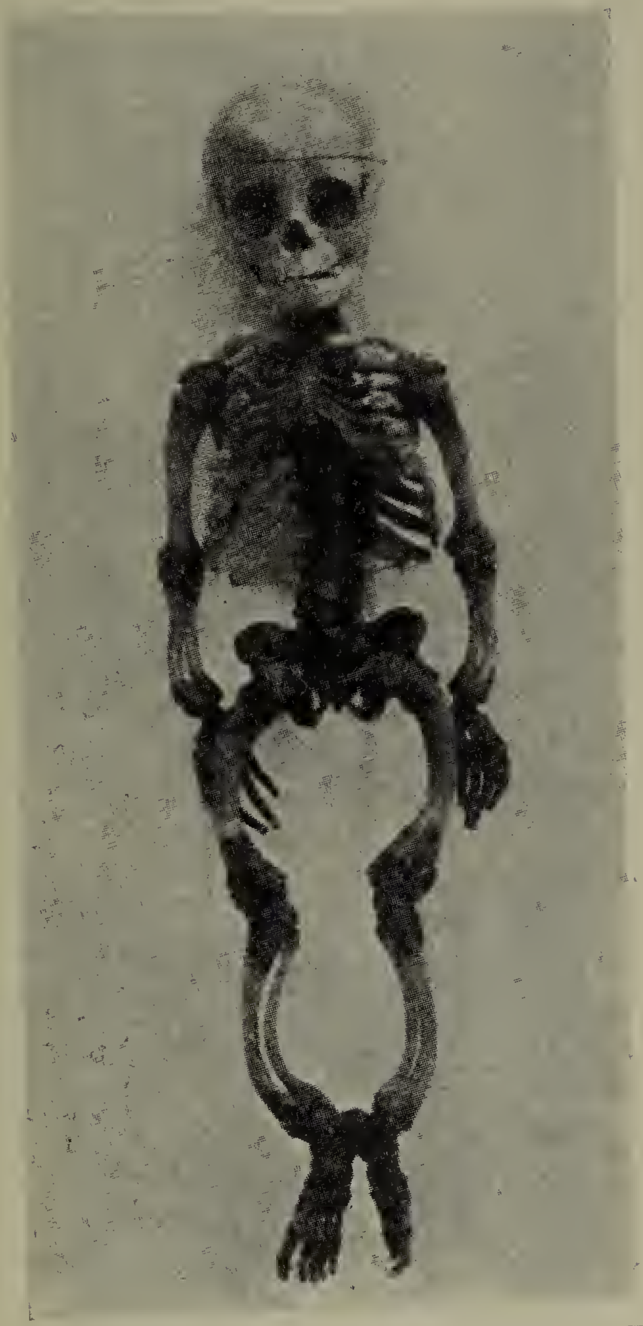

Fig. 204.—Skeleton of a Severe Case of Rickets.

From a Negro child of 6 years, in the Pennsylvania Hospital of Philadelphia at the time of his death. (*Donhauser, Bull. of Ayer Clinical Laboratory*, 1907, *No.* 4, 13.)

Beneath the periosteum of both long and flat bones an analogous process is seen, with cell-proliferation, vascularity, and imperfect calcification, resulting in thickening and in the production of osteoid tissue.

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[1] *Loc. cit.*, 424

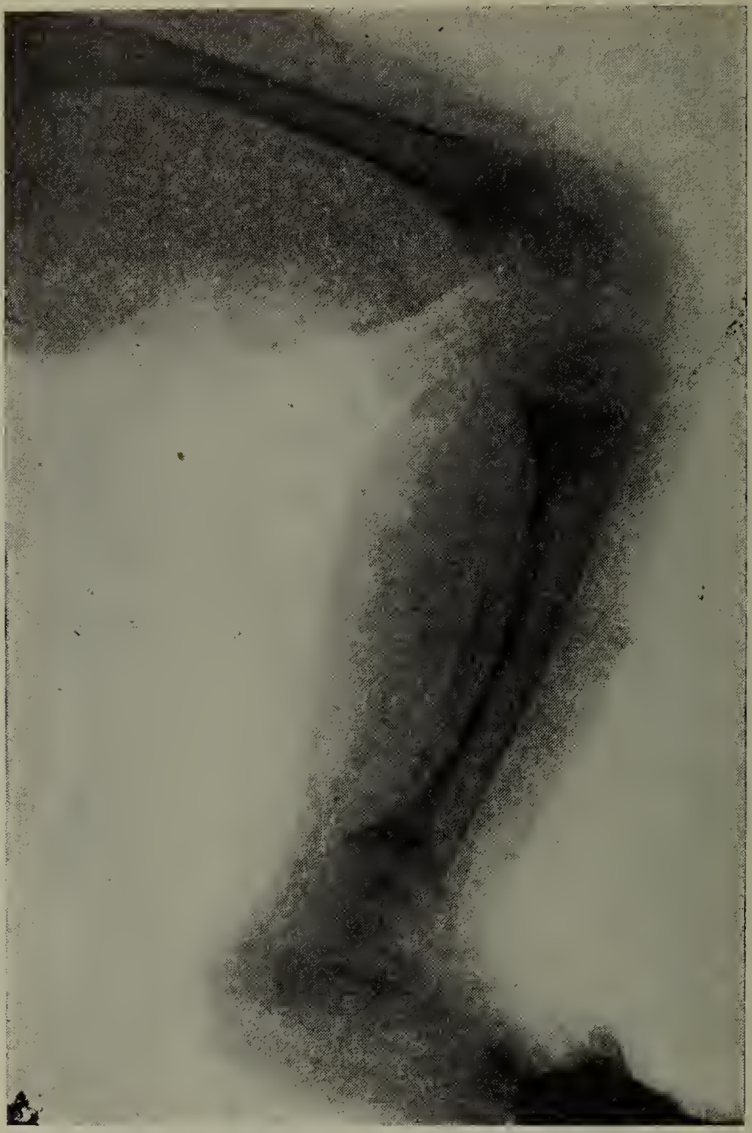

Fig. 205.—Radiograph of Left Leg in Rickets. Moderately Advanced Stage.
Child aged 1 year. Shows the hazy outline of the epiphysis and of the ends of the diaphysis of the long bones.

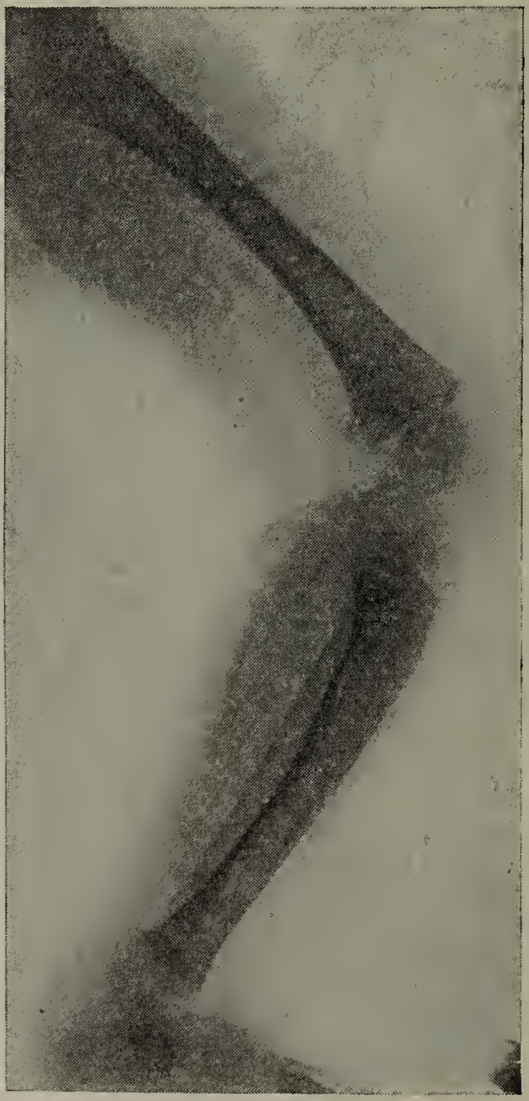

Fig. 206.—Radiograph of Leg of Normal Child of 8 Months.
Contract with the condition in rickets Fig. 205 well illustrated.

Chemically the rachitic osseous tissue is very decidedly altered in the amount of lime and of phosphorus contained, these being sometimes diminished to 50 per cent. or even 25 per cent. of the normal. The percentage of organic matter and of water is increased. These characteristics account for the unusual degree of softness and flexibility of the bones, which suffer in consequence from green-stick fractures, with the production of a large amount of callus.

The *x*-ray appearance of the bones is characteristic. In the early stages there is a loss or diminution of the clear-cut line of demarcation between the epiphysis and the diaphysis in the long bones. In a later stage there is a general diminution in the density of the shadow, and an indistinct shading of the epiphysis and diaphysis into each other is present (Fig. 205). The end of the diaphysis is broadened and often somewhat cup-shaped.

During recovery from the disease the bony changes disappear through resorption and condensation of the imperfect bone taking place, and lime-salts are again deposited. The development of very hard, compact, dense, bony tissue (eburnation) may result.

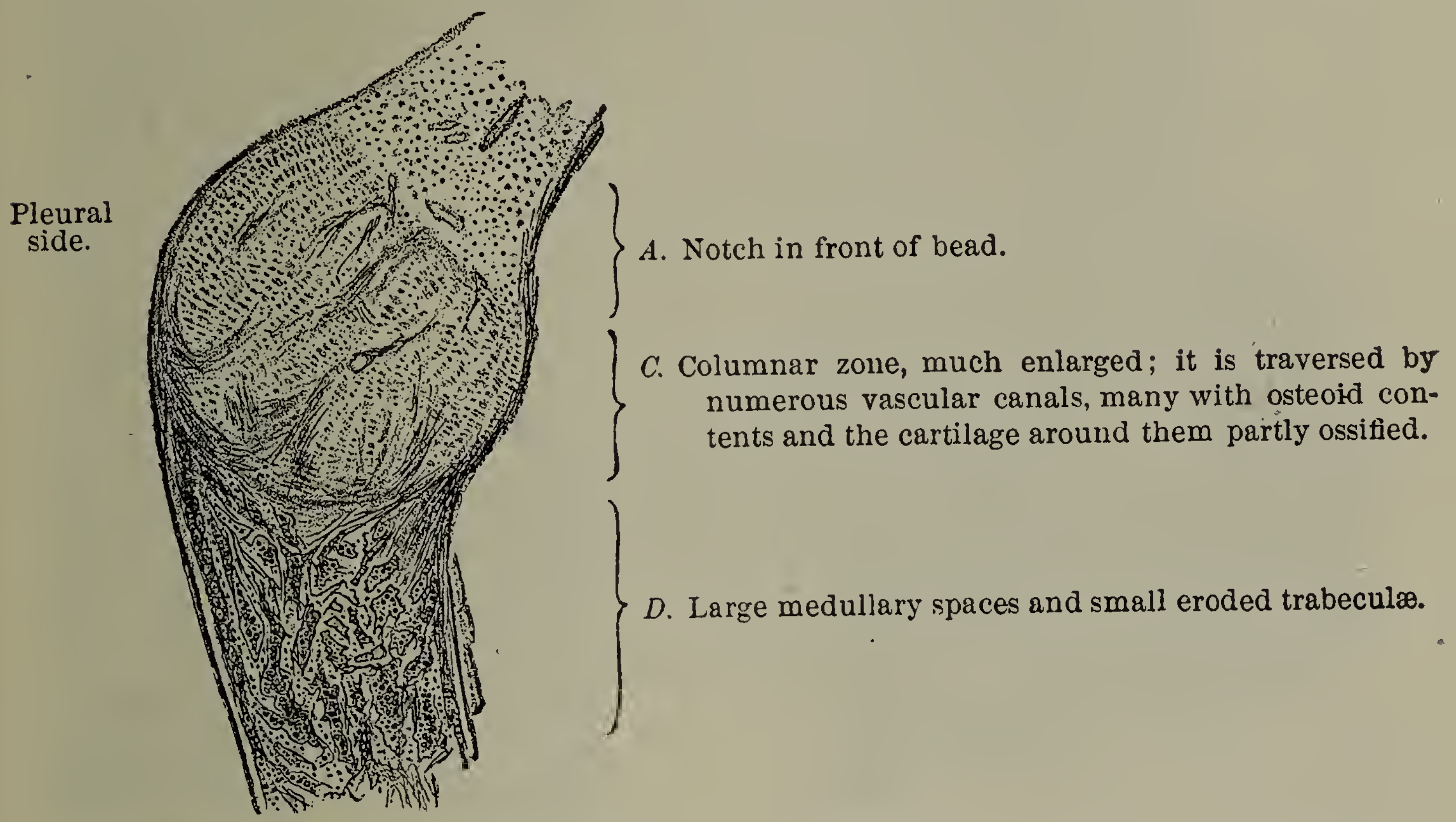
Pleural side.
A. Notch in front of bead.
C. Columnar zone, much enlarged; it is traversed by numerous vascular canals, many with osteoid contents and the cartilage around them partly ossified.
D. Large medullary spaces and small eroded trabeculæ.

Fig. 207.—Longitudinal Section of Rib of Rachitic Child Aged 15 Months. About 3 times natural size. (*Barlow and Bury, Keating's Cyclopædia of the Diseases of Children*, 1889, *II*, 241.)

In brief, the osseous changes in rickets consist in unusual hyperemia; excessive proliferation of cartilage; irregularity in the transformation of cartilage into bone, with disturbance of the normal division into zones; deficient deposit of lime-salts, with consequent over-development of osteoid tissue which fails to change into true bone; thickening of the periosteal layer through undue cellular production, and the formation of masses of osteoid tissue here; absorption of trabeculæ in the spongy bone, with consequent increase of the medullary spaces; and hyperemia and widening of the central marrow-cavity.

Lesions of the Soft Parts and Organs.—The spleen is often enlarged, yet not necessarily so. Catarrhal conditions of the lungs are frequent, with areas of atelectasis and others showing emphysema. Moderate enlargement and fatty infiltration of the liver may occur. The gastroenteric tract often exhibits catarrhal inflammation with unusual distention of the stomach and intestines. The lymphatic glands are frequently enlarged. Thinness and weakness of the muscles are evident; well seen in the abdominal walls. The ventricles of the brain are often moderately dilated.

Symptoms.—Although the skeletal changes are the most characteristic, the constitutional symptoms are the first to become manifest. These seldom are noticeable before the age of 3 months, although the microscopical changes in the bones appear even in the 2d month. Among the earliest manifestations are head-sweating, restlessness, and rocking of the head on the pillows. These early symptoms develop gradually, and usually attract little attention. In a short time beading of the ribs is noticed in nearly all cases. Other bony enlargements now develop, especially of the skull, ribs, wrists, and ankles, while characteristic alterations of the shape of the chest appear and the abdomen becomes pot-bellied. The general health may remain unaffected at first, but in well-marked cases the child finally grows anemic and suffers readily from respiratory and gastrointestinal catarrhal processes and sometimes convulsions. The teeth are late in appearing and decay early; the fontanelle fails to close at the proper time; growth of the body is interfered with; and when sitting and walking begin, at a period much later than normal, curvature of the long bones and of the spine is produced.

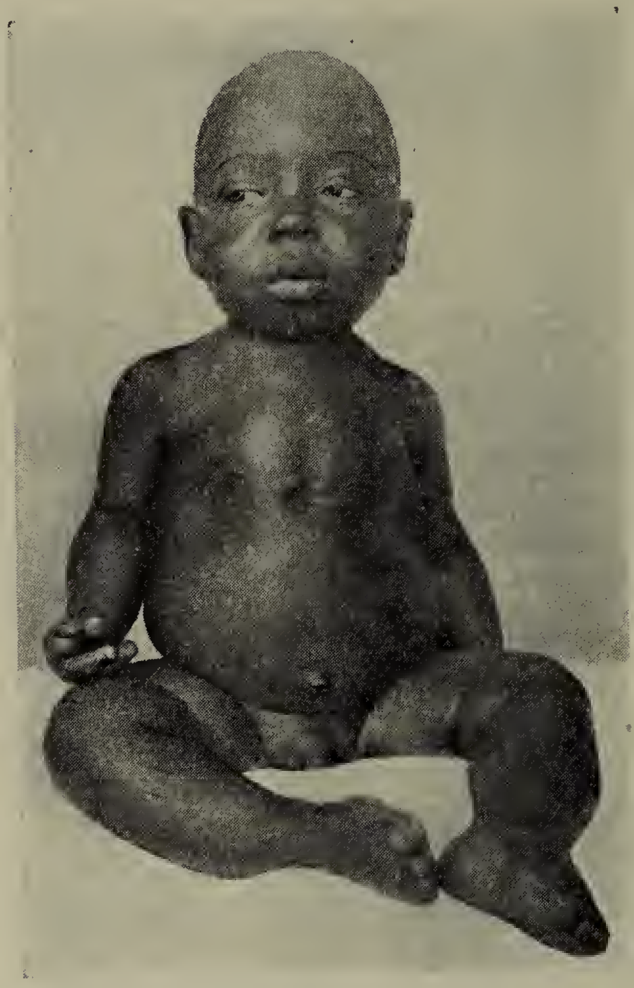

Fig. 208.

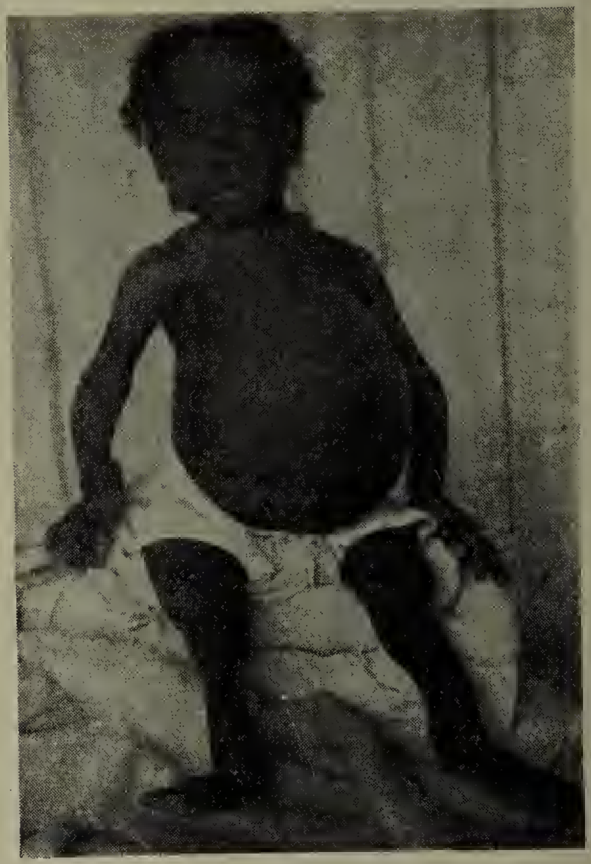

Fig. 209.

Fig. 208.—Rachitis, Showing Especially the Lateral Depression of the Thorax and Curvature of the Tibiæ.

From a child in the Children's Hospital of Philadelphia.

Fig. 209.—Rachitis, Showing the Pot-belly and the Distortion of the Extremities.

From a child of 4 years, in the Children's Medical Ward of the Hospital of the University of Pennsylvania.

The complex of symptoms and the general appearance of the patient are very characteristic (Figs. 208 and 209) and the diagnosis easy. A more detailed description is required:

Head.—The head appears, and often is, larger than normal in its horizontal circumference and is usually brachycephalic. The forehead is prominent, and the occiput and vault flattened. This produces a peculiarly box-like form (Fig. 210). The shape, which develops oftenest about the end of the 1st year, depends largely upon the deposit of rachitic bone which takes place especially upon the frontal and parietal eminences, along the sutures, and about the fontanelles. The flattening of the occipital region is due to pressure upon the pillow; and should the child while in bed lie very constantly on one parietooccipital region, great asymmetry of the dead may result, one oblique diameter being much shorter than the other, one ear nearer the front, and even one cheek more prominent. The anterior fontanelle in rickets is larger than normal, and its time of closing is delayed, this occurring sometimes not until after the 2d year. A systolic murmur may often be heard over it. In severe cases the posterior fontanelle and the sutures remain open until the end of the 1st year. In the posterior portion of the skull thin, soft membranous spots are often found in the 1st year (craniotabes, see Vol. II, p. 728). Schwarz[1] found that of 4944 infants, 734 showed craniotabes during the 1st year and 301 of these during the first few months, before the period of life when rickets is most frequent. The condition is not indicative of rickets in the absence of other signs. The veins of the scalp are distended, especially over the temporal regions. The hair is quite commonly worn away from the back of the head by the frequent rocking movements. The face seems small, the upper jaw being narrower than normal and the lower somewhat square. These various deformities usually disappear as recovery takes place, but the relative increase in size may persist in severe cases to

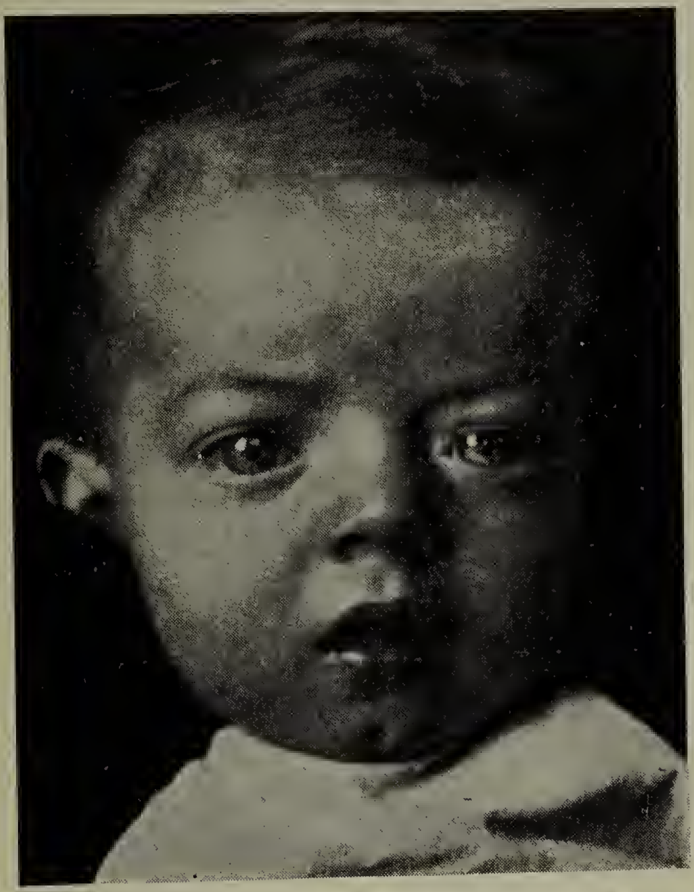

Fig. 210.—Moderate Rachitic Alteration of the Head.

Shows the prominent frontal bosses, in a patient, aged 9 months, in the Children's Hospital of Philadelphia.

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[1] Amer. Jour. Dis. Child., 1920, XIX, 384.

some extent throughout life. The teeth usually appear late and out of the normal order, not infrequently none having erupted during the 1st year. They are generally of good form, although they may decay early. On the other hand, the teeth of the permanent set have often been damaged by the previous rachitic condition, and have a decided tendency to malformation, erosion and caries.

Thorax.—Beading of the ribs at the costochondral articulations is the most frequent and the earliest osseous symptom. It is quite visible to the eye unless the infant is well covered with fat. Morse[1] found it in all of 318 cases of rickets, and the only symptom in 41 per cent. of these. It is very doubtful whether slight enlargement of the costochondral junctions can be considered sufficient evidence in itself to warrant a diagnosis of rickets. It may be present in the new-born (Schiff).[2] Schwarz[3] found it in 13 per cent. of infants during the early months of life. In all well-marked cases there is in addition a depression of the cartilages

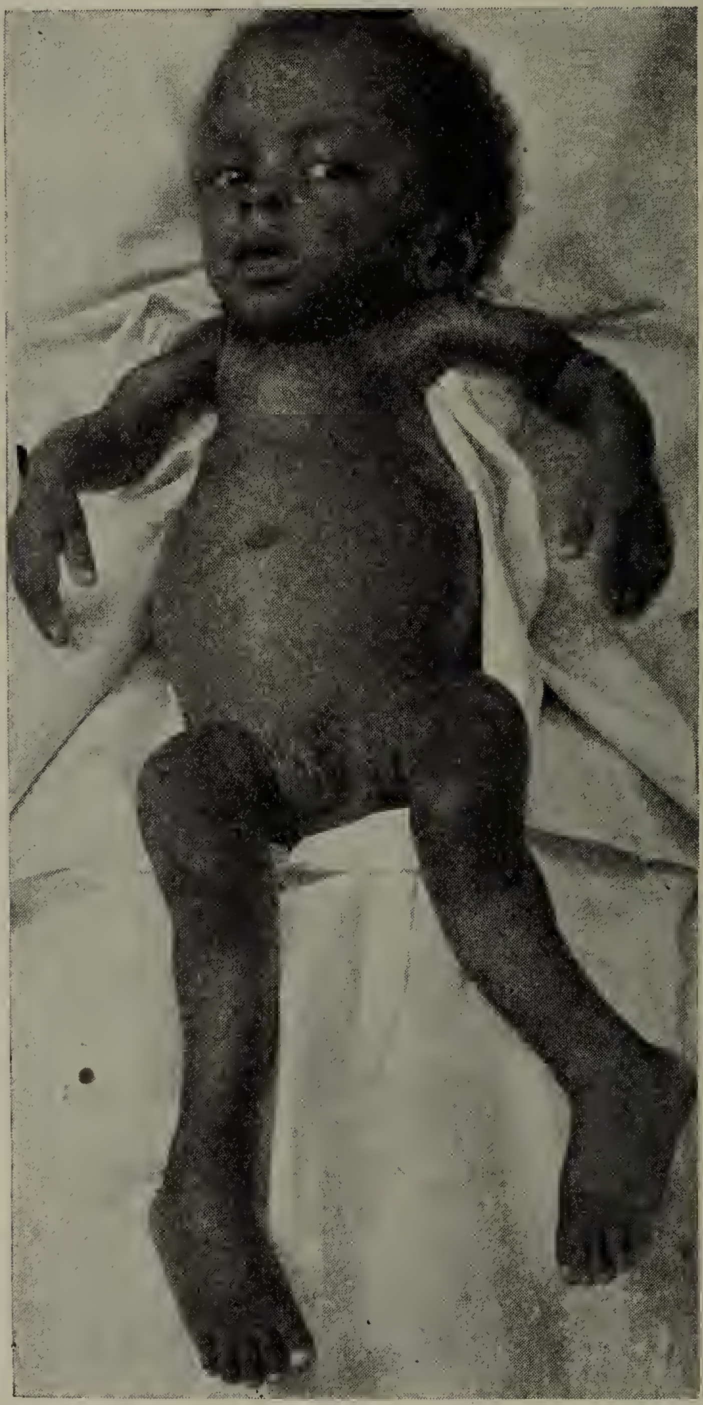

Fig. 211.—Multiple Deformities in a Severe Case of Rickets.

From a child in the Children's Ward in the University Hospital, Philadelphia. Shows especially the great contraction of the thorax with the costal flare.

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[1] Jour. Amer. Med. Assoc., 1900, XXXIV, 724

[2] Monatsschr. f. Kinderheilk., 1921, XXII, 46.

[3] *Loc. cit.*

anterior to the epiphyseal enlargements. A flattening or even a concavity of the sides of the thorax in a vertical direction develops, extending backward to the posterior axillary line and downward about as far as the 7th rib. As a result of this alteration the sternum is unusually prominent, producing a condition suggesting "pigeon breast," but usually differing somewhat from the typical instances of this seen in certain other conditions, in that the whole sternum is pushed forward in a straight line, without any anterior curvature of it. The rachitic thorax also exhibits bulging at the angle of the ribs posteriorly, accompanied by a flattening of the back. These changes produce in well-marked cases a deformity known as the "violin-shaped" chest.

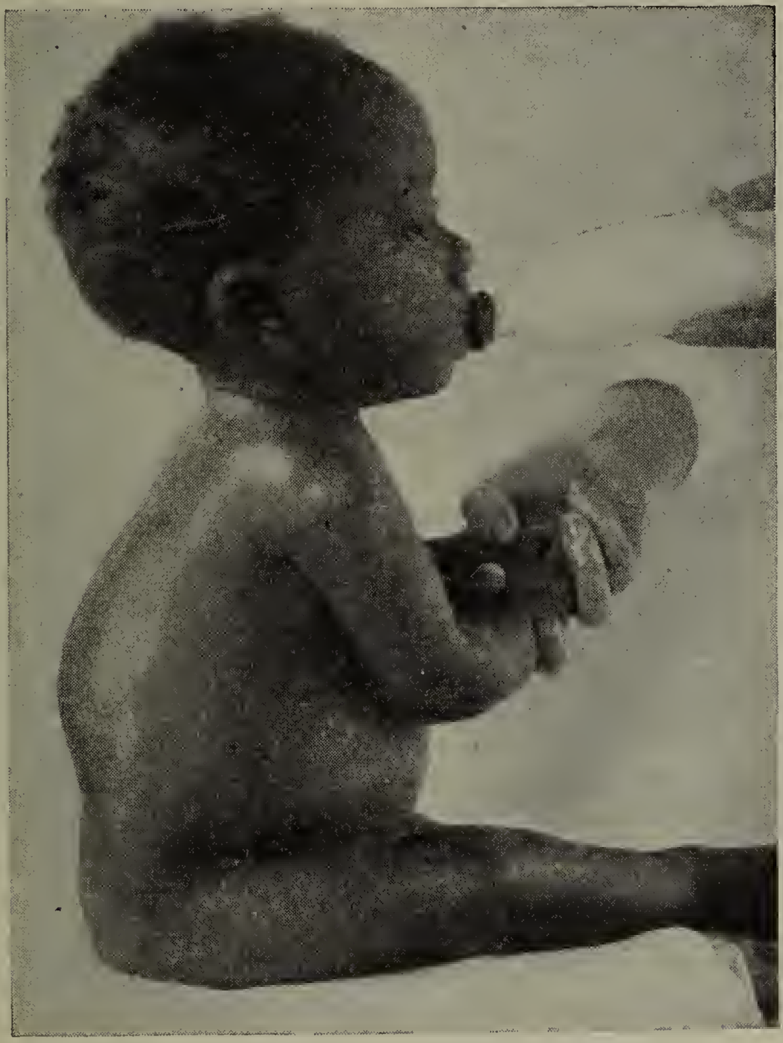

Fig. 212.—Rachitic Spinal Curvature.

Very well-marked when child is sitting. *Courtesy of Dr. H. R. Wharton.*

In addition to the vertical lateral depression there is a horizontal one (Harrison's groove) caused by the decided flare of the costal border. This is at about the level of the xiphoid cartilage and corresponds to the insertion of the diaphragm. Occasionally a funnel-shaped depression of the lower part of the sternum occurs. In some cases the clavicles are thickened at the extremities, shorter, and more curved than normal, and may show green-stick fractures.

The thorax as a whole is lengthened in an anteroposterior direction, and is small and narrow, except for the sudden widening of the lower portion (Fig. 211). The deformities are produced by the action of the muscles of respiration, including the diaphragm, upon the very soft bones; and by the pressure of the back against the bed, and of the distended stomach and intestines and the enlarged liver against the costal border.

Spinal Column.—This may be normal, but a certain degree of lateral curvature is common in infants, and may be very persistent. Still more frequent, and always seen in well-marked cases, is a long posterior

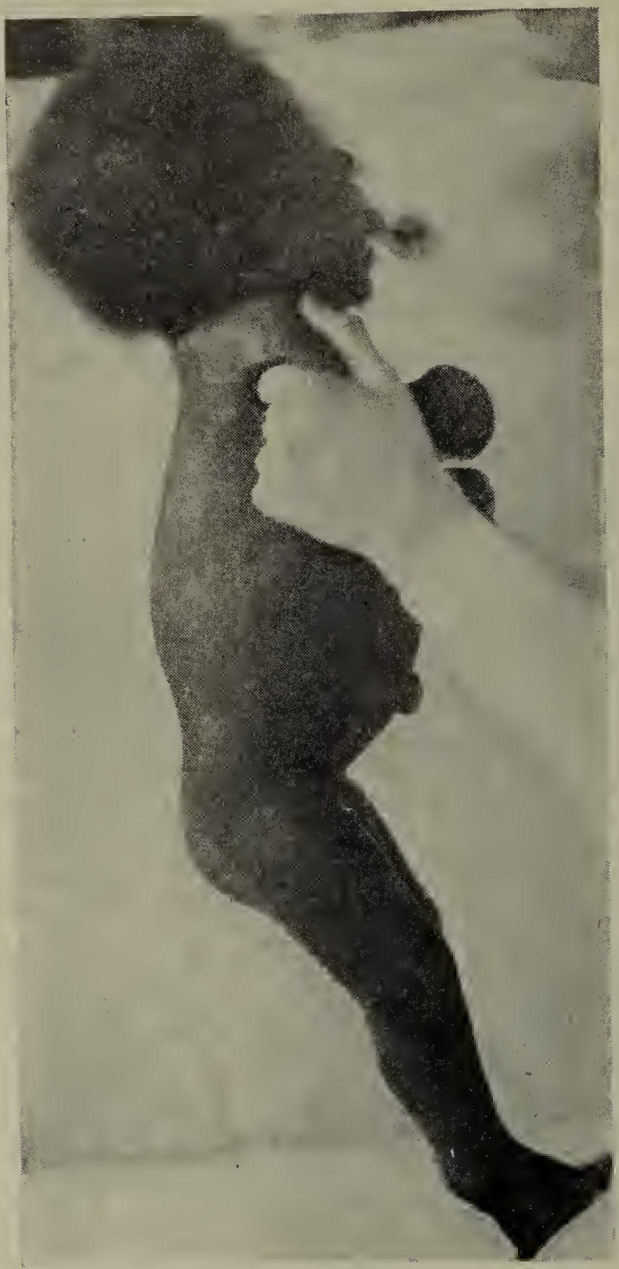

Fig. 213.

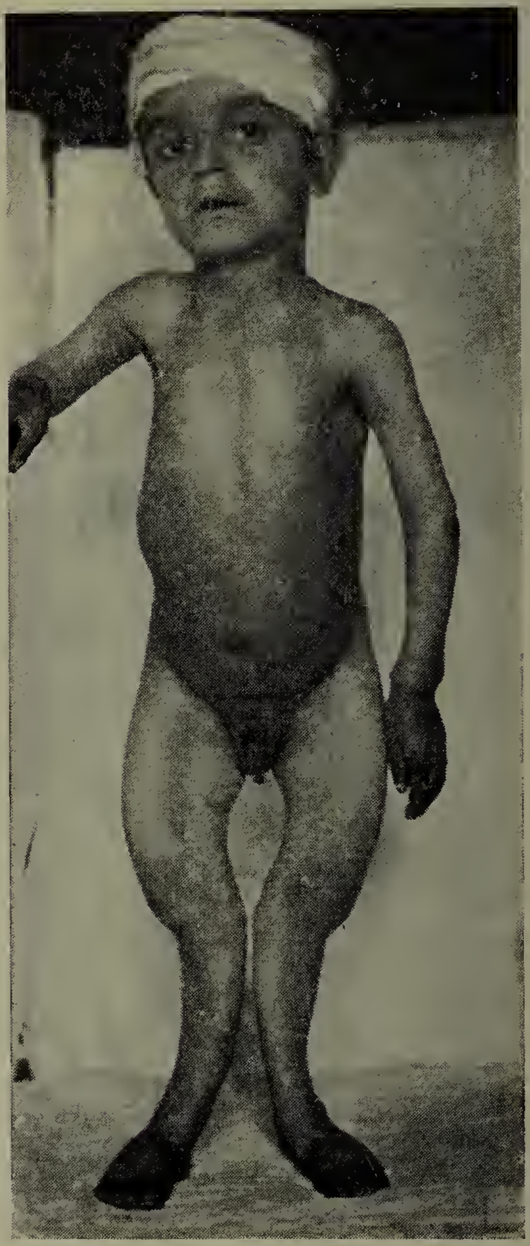

Fig. 214.

Fig. 213.—Rachitic Spinal Curvature.

Same case as in Fig. 212. Very decided diminution in the degree of curvature when the child is suspended. *Courtesy of Dr. H. R. Wharton.*

Fig. 214.—Severe Case of Rickets.

Illustrates the enlargement of the wrists and the curvature of the legs. *Courtesy of Dr. H. R. Wharton.*

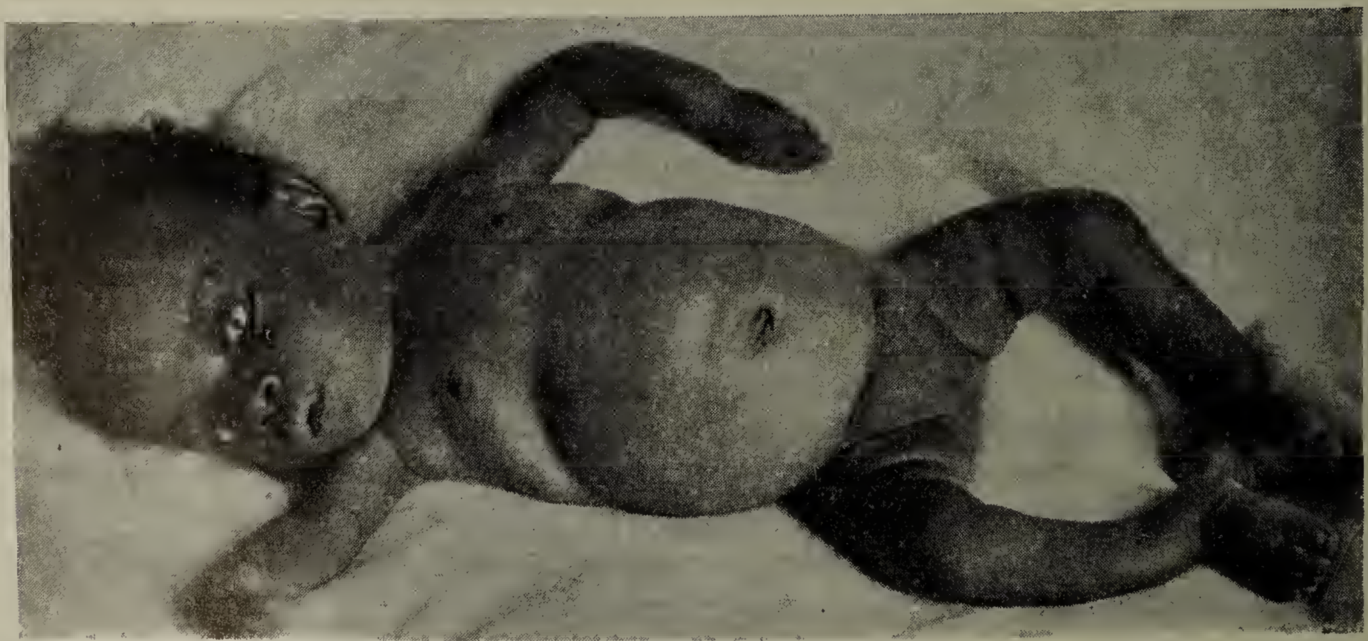

Fig. 215.—Rachitic Deformity, Showing the Curvature of the Limbs and the Well-marked Pot-belly.

Patient in the Children's Ward of the University Hospital, Philadelphia.

curvature dependent upon the weakness of the ligaments and of the muscles of the trunk. It usually disappears entirely or to a considerable extent when the child is suspended from the arm-pits (Figs. 212 and 213).

Pelvis.—In severe cases the pelvis may become permanently deformed, the anteroposterior diameter being shorter and the outlet narrower, with thickening of the crests of the ilia.

Extremities.—Epiphyseal enlargement at the wrist (Fig. 209) is an early symptom nearly always present, and that of the ankle is also very frequent. Similar enlargement is often discoverable at the lower end of the humerus and femur, less frequently at the upper end of these bones and of the tibia and fibula. Bending of the shafts of the long bones occurs in the lower extremities, the commonest deformities being knock-knees and bow-legs (Fig. 214). (See Vol. II, pp. 724, 725.) The tibia may be bent outward or forward (Fig. 215). The femur is curved only in the severe cases, and generally in a forward and outward direction. Coxa vara is another rachitic deformity. Sometimes a striking distortion results from the child sitting cross-legged, the femora being partly rotated outward and the tibiæ and fibulæ of one side fitting into those of the other where pressure one upon the other has been constantly exercised. Bending of the humerus is not common but the bones of the forearm often curve outward. Greenstick fractures on the concave side of the bone are not infrequent, especially in the forearm and the tibia, and add to the deformity. Bending is produced largely by the action of position and of muscular pull upon the soft bones, although partly by asymmetrical growth of the epiphysis. It occurs in the legs even before walking is commenced, but is much increased by it. The deformity of the extremities becomes a marked symptom toward the end of the 1st year.

Growth of the long bones in length is very commonly interfered with, especially in the lower extremities, and in the worst cases permanent dwarfism may be a final result. Occasionally thickening of the fingers occurs. Fat-foot is a common rachitic deformity.

Ligaments.—Relaxation of these aids in producing deformity. It is seen especially in the spine and in the larger joints, aiding in the production of knock-knee, over-extension of the knee-joints, weak ankles, and scoliosis.

Muscles.—Lack of tone of the muscles of the body is a symptom commonly present. They are poorly developed, pale, and small. As a result, cases of well-marked rickets are very late in standing and walking, this being occasionally deferred even until early childhood is well under way. In one instance a child of 4 years was unable to walk, although without any serious deformity. The lack of muscular power is often, indeed, so great that the diagnosis of paralysis may be made. I have repeatedly seen the mistake occur. The condition of "pot-belly," a nearly constant symptom of rickets, depends to a large extent upon weakness of the abdominal muscles, although the weakness of the gastric and intestinal walls, combined with the tendency to flatulent dtioistenn, aids in its production. The abdomen is uniformly distended, tympanitic, and not tender on pressure. Very commonly in marked cases there results a diastasis of the external recti of the abdomen, well shown when the child attempts to raise itself from a recumbent position (Fig. 216). Constipation depends upon the muscular weakness of the intestinal and abdominal walls.

General Condition.—The skin is usually pale and transparent, with a tendency to enlargement of the veins in many localities, especially over the scalp and at the root of the nose. Although often apparently well-nourished, so far, at least, as the presence of adipose is concerned, children with rickets nearly always possess flabby tissues. Severe cases eventually lose considerably in weight and in general health and become anemic. The general power of resistance is slight.

Digestive System.—Adenoid growths and tonsillar hypertrophy are common. The sensitiveness of the mucous membrane is shown by the readiness with which rachitic subjects develop obstinate catarrhal disturbances of the stomach and intestines from slight causes. This aids in producing the tympanitic distention of the stomach and intestines and the characteristic pot-belly referred to. Constipation is common, often alternating with diarrhea. The liver is not infrequently enlarged, but this may be only apparent, due to the displacement downward, the result of the distortion of the chest.

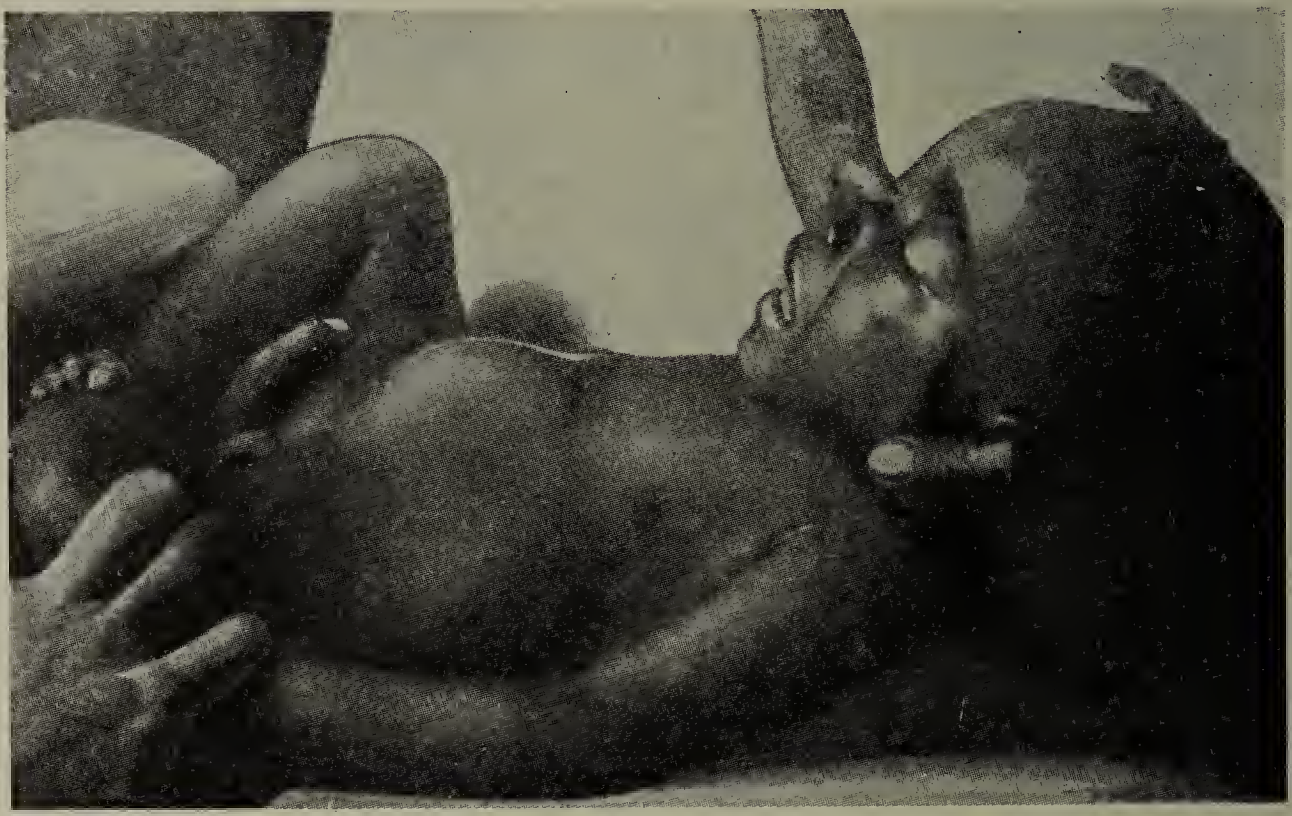

Fig. 216.—Rachitis, with Diastasis of the Recti Abdominis

The bulging intestine is seen in the center of the abdomen between the layers of muscle. Colored child of 15 months. (*Francine, Arch. of Pediat.*, 1904, Feb., 116.)

Respiratory System.—The mucous membranes in rickets are particularly prone to the development of diseased conditions, among them bronchitis and bronchopneumonia. Acceleration of respiration, without fever or other discoverable cause, and often accompanied by moving of the alæ nasi and other evidences of dyspnea, is very characteristic. The breathing in well-marked cases is largely diaphragmatic, due to the softening and yielding of the framework and the weakness of the muscles of the thorax. The deformities of the chest produce alterations in the physical signs of auscultation and percussion, which may be very misleading in the diagnosis of possible intra-thoracic diseases.

Circulatory System.—In mild cases the blood is unaffected, but in the severer ones more or less anemia usually develops. Morse's[1] studies of his own cases and of the writings of other investigators led to the conclusion that the blood in rickets usually shows the red cells normal or reduced in number, and the hemoglobin always reduced to a greater degree.

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[1] Med. and Surg Rep Boston City Hosp., 1897.

Moderate leucocytosis is present in about ½ the cases. Dilatation of the superficial veins is decided.

Nervous System.—Nervous symptoms are very common, prominent among them being restlessness at night, with rocking of the head upon the pillow, and constant tossing off of the bed-clothes. The unusual instability of the nervous system characteristic of rickets would appear to predispose to convulsive conditions of various sorts, as tetany, laryngospasm, and particularly eclampsia. These symptoms are those, however, of spasmophilia, which seems in many instances to be associated in some way with rickets. In a study of 1766 rachitic children, Oliari[1] found evidences of spasmophilia in 542. While this subject will be more fully discussed later (see Spasmophilia, Vol. II, p. 526), it may be stated here that spasmophilic symptoms are present when the blood-calcium is low, and, as we have pointed out, this is not usual in rickets uncomplicated by spasmophilic symptoms. It is also of interest to note that the type of rickets which is associated with spasmophilia is that in which there is rather slight bony deformity. Pain in the bones, especially evident when the infant is lifted by grasping the chest, is sometimes seen. Although perhaps due in some cases to the rachitic lesions, it is probably oftener dependent upon a complicating scorbutus or epiphyseal separation. In the category of nervous affections may be placed the profuse sweating of the head, which occurs during sleep irrespective of the weather, and which is sometimes sufficient to moisten the pillow. Fever may be present, but does not appear to be a symptom of the disease. In severe cases there may be decided mental backwardness associated with the general weakness.

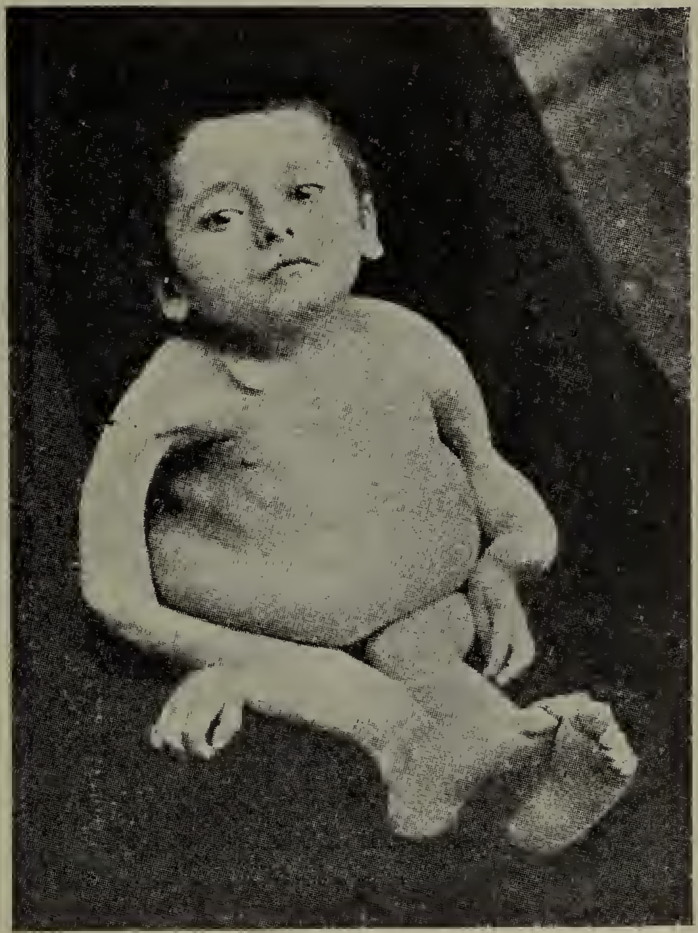

Fig. 217.—Late Rickets.

Patient 28 years of age, in whom the rachitic changes began at the age of 8 years. Photograph taken at the age of 21 years. (*von Bókay, Arch. de méd. des enfants*, 1910, *XIII*, 444.)

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[1] La Pediat., 1910, XVIII, 581.

Urine.— No special alterations are discoverable.

Glandular System.—The external lymphatic glands are frequently found enlarged. Moderate enlargement of the spleen is common and great increase in size occasional, but it is still undetermined whether this hypertrophy is a symptom of rickets or only a complication. It must be remembered, too, that the wide costal flare so often present, and the diminished capacity of the thorax, renders the spleen much more readily felt.

FETAL RICKETS.—Whether or not such a condition as fetal or congenital rickets exists has been much disputed. The diagnosis in cases of so-called fetal rickets has been based upon the presence of craniotabes or beading of the ribs in the new-born. These things in themselves are not proof of the existence of rickets. It is claimed as very common by some, but denied absolutely by other investigators. Certainly it is rare in the United States; and it is, moreover, probable that the majority, if not all, of the reported cases are instances of osteogenesis imperfecta or of chondrodystrophy fetalis (see Vol. II, pp. 728 and 734), which differ pathologically from the rachitic process. An elaborate review of the subject by Wieland[1] leads him to the conclusion that not a single case beyond criticism has been reported.

ACUTE RICKETS.—Although cases of this condition have been described in which the symptoms develop with great rapidity, it is doubtful whether this is not something entirely different from rickets—in many cases certainly infantile scurvy. Other cases are, perhaps, only instances of rapid development of rickets in a severe form, but this cannot properly be called acute.

LATE RICKETS.—The pathological processes of osteomalacia are so similar in some respects to those of rickets, although the causes are different, that it is probable that the instances of so-called late rickets, developing in later childhood or at puberty, are for the most part properly to be classified as early osteomalacia. Schmorl,[2] Rendu and Werteimer,[3] and others, however, from a careful study of the subject maintain that genuine rickets may begin in later childhood. Its development is usually slow, the course prolonged, and the final deformities very decided (Fig. 217). The condition has certainly been rare, although reports by Hochstetter,[4] Bittorf,[5] Engel,[6] and others indicate that a condition with *x*-ray pictures of osseous disturbance similar to rickets, and otherwise resembling the disease, occurred in Germany after the World War with considerable frequency in patients from 15 to 18 years of age. It is a question, however, how many of these cases were of the nature of a simple osteoporosis. Bony deformities and changes somewhat resembling rickets may be associated with chronic intestinal nephritis in childhood.[7]

Complications and Sequels.—The great tendency to the development of respiratory affections, especially bronchitis and bronchopneumonia, has already been alluded to. Atelectasis not infrequently occurs in infants with severe rachitic deformities of the chest. The gravity of all respiratory diseases in rachitic subjects is greatly increased by the yielding character of the thoracic walls and the insufficient expansion of the lungs. The disposition to diarrhea or constipation has also been mentioned. A certain degree of hydrocephalus is a complication frequently observed in cases with marked rachitic heads, this being the result of the yielding of the soft cranial bones, which allows the fluid to accumulate in the ventricles. General convulsions are common, as are laryngospasm and tetany. This tendency to the development of spasmophilic symptoms, particularly convulsions, is especially marked in rickets. Umbilical hernia is of frequent occurrence, due to the distention and thinning of the abdominal walls. Infantile scurvy usually manifests itself in subjects already rachitic, although to this there are frequent exceptions. Bony deformities of various sorts remain as sequels, sometimes permanent.

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[1] Jahrb. f. Kinderheilk., 1908, LXVII, 675, 1916, LXXXIV, 360; Ergebn. der inn. Med. u. Kinderheilk., 1910, VI, 64.

[2] Deutsch. Arch. f. klin. Med., 1905, LXXXV, 170.

[3] Rev. d'Orthop., 1921, XXVIII, 215.

[4] Münch. med. Wochenschr., 1919, LXVI, 776.

[5] Berl. klin. Wochenschr., 1919, LVI, 652.

[6] Berl. klin. Wochenschr., 1920, LVII, 35.

[7] See Renal Infantilism, Vol. II, p. 857.

Course and Prognosis.—Rickets is essentially a chronic disease. The active symptoms usually continue not longer than the termination of the 1st and seldom more than the end of the 2d year, their retrogression being evidenced by the gradual spontaneous cessation of head-sweating; the improvement of the general restlessness, irritability, and other nervous symptoms; disappearance of anemia; closure of the fontanelles; and cessation of the disordered intestinal and respiratory states and of the softness of the bones. Recovery from the bony deformities is very slow. In most cases the enlargement of the epiphyses will finally almost entirely disappear, as will the deformity of the skull. Sometimes, however, the head remains always larger than normal. Even moderate bowing of the legs and other deformities disappear spontaneously in a remarkable manner. Recovery is usually complete by the end of the 3d or 4th year. In well-marked cases of the disease, however, permanent alterations are likely to be present throughout life, in the form of curvature of the bones of the thigh, leg, or forearm, producing bow-legs, knock-knees, and other deformities; distortions of the chest; scoliosis; flat-foot; rachitic pelvis; rachitic coxa vara, and dwarfing from arrested growth of the bones in length. It is also possible for rickets undergoing recovery to exhibit exacerbations, or even to relapse after recovery has apparently taken place; the cause being the renewal of the etiological factors or the occurrence of some intercurrent disease. That recurrence is not uncommon is indicated by the report of Hess[1] of 31 cases personally observed.

The disease in itself is not dangerous to life, but through its numerous complications or through other intercurrent diseases it is the cause of many deaths. The contraction of the chest and the lack of resiliency in its walls are very serious factors should pneumonia or pertussis be contracted, and severe cases of rickets may die from ordinary bronchitis through the development of atelectasis. The debility and general loss of resisting power predispose to a fatal ending in cases of intestinal disease. Convulsions attending rickets are the cause of death in numbers of instances.

Diagnosis.—Well-developed rickets is not readily confounded with any other disease. Early in its course it is not so easily recognized. The principal early diagnostic symptoms are the head-sweating and the restlessness at night. Later are evident the enlarged fontanelles characteristic shape of the head, distended abdomen, alteration in the shape of the chest, rachitic rosary, and the various other deformities described.

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[1] Amer. Jour. Dis. Child., 1926, XXXI, 380.

X-ray pictures, especially as seen in the long bones (see p. 692, Figs 205; 206), are valuable aids in diagnosis. Although typical roentgenological evidence in the epiphyses will not appear as early as does the rachitic rosary, this method of examination is particularly serviceable because, as has been stated, slight beading of the ribs is not diagnostic without the presence of other symptoms.

Certain other conditions occasion difficulty in diagnosis. *Hydrocephalus* has a superficial resemblance to the rachitic head in some instances. In both the fontanelles are large and the sutures open, but in hydrocephalus the head is of a more globular shape and the sides protrude somewhat beyond the ears, while in rickets the shape is more rectangular, with areas of decided thickening of the bones, especially over the parietal and frontal eminences and about the fontanelles. The tension over the fontanelle is greater in hydrocephalus. *Craniotabes* is not positive diagnostic evidence of rickets, as it may occur in syphilis also.

Delayed dentition, although a diagnostic symptom of rickets, is not necessarily so. If rickets is somewhat late in developing, the first teeth may erupt promptly; and, on the other hand, it is not infrequent for entirely healthy infants to have no teeth until the age of 10 or 11 months, or even a year. *Infantile scurvy* has often been confounded with rickets because the two are so frequently combined; and the names "scurvy-rickets" and "hemorrhagic rickets" have been applied to infantile scurvy. Either disease can occur without the other, and the two have, in reality, no symptoms in common. The tenderness of the bones which sometimes seems to be a symptom of rickets is, in most cases, as already pointed out, probably due to a certain scorbutic element. *Infantile osteomalacia* has been described, but is to be considered identical with rickets in most instances, although probably some of the cases reported belong to the category of osteogenesis imperfecta.

Osteogenesis imperfecta has often been called "fetal rickets." In it there is usually associated a remarkable thinness of the flat bones, especially of the skull, with more or less deformity of the long bones. It has little in common with rickets. *Osteopsathyrosis,* or *fragilitas ossium,* has likewise often been confounded with rickets. It is true that in severe rickets there is a decided tendency to the occurrence of fractures, yet these are usually of the green-stick variety, due to softness of the bone. In true fragilitas ossium there is nothing of the soft character present, and neither chemically nor microscopically is there anything found resembling rickets. The two conditions are absolutely distinct, as I[1] have pointed out elsewhere and osteopsathyrosis is more closely associated with some forms of osteogenesis imperfecta.

Infantile myxedema in its early stages bears certain resemblances to rickets. In both there is delay in dentition and in the closing of the fontanelles, and slowness in learning to walk. In cretinism, however, there is an unusual slowness of growth in length, and the peculiar physiognomy of the disease, with mental impairment and the general physical characteristics, soon makes the diagnosis easy. *Paralytic conditions* of various sorts, with wasting of the muscles, sometimes lead to the suspicion of rickets. In all the learning to walk may be long delayed, but in none are the other symptoms of rickets present; while careful study may show the characteristic electrical reactions and other evidences of poliomyelitis, or the spastic condition of a cerebral paralysis.

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[1] Amer. Jour Med. Sci., 1897, CXIII, 426.

Syphilis has little in common with rickets. The syphilitic pseudoparalysis occurs usually in the early months of life, antedating the weakness of rickets, and the sabre-tibia of later syphilitic manifestations is dependent solely upon a thickening of the anterior border of the bone and is unaccompanied by other conditions suggesting rachitic deformities. The spinal curvature of *Pott's Disease* is short and angular in shape, in contra distinction to the long anteroposterior curve of rickets, and when well-marked does not lessen when the child is lifted by the arms.

Treatment. **Prophylaxis.**—Rickets is to a large degree preventable if the proper diet is given and if advantage is taken of the protection afforded by sunlight and cod liver oil. The use of proprietary amylaceous foods and of condensed milk should be avoided. Breast-feeding is to be employed whenever possible. What will be said later regarding heliotherapy and dietetic treatment of rickets already developed applies as well to its prevention. Cod liver oil has been considered as completely protective, especially in experimental studies, and interchangeable with sunlight. Hess and Unger[1] and others believe it inferior to sunlight as a prophylactic. This has been the conclusion as well of others, such as DeBuys and von Meysenbug.[2] The failure of cod liver oil to protect in some cases may be due to insufficient dosage or to the administration of oil low in the antirachitic factor, about which there is as yet little known in the matter of quantitative requirements. Until the exact nature of this is discovered, oil should be used which has been found experimentally to be efficient in protecting rats against rickets. Because of the widespread prevalence of the disease, it would not be amiss to administer cod liver oil to infants almost as a routine measure in the temperate zone during the winter-season. Infants of 2 or 3 months of age may be started with 5 drops 3 times a day, and the amount slowly increased until at 6 months of age the dosage is ½ dram (1.8). Older infants may be given a dram (3.7) 3 times a day. If intolerance develops, as shown by vomiting, loose stools and loss of appetite, egg-yolk has been recommended as a substitute (Hess;[3] Casparis, Shipley and Kramer[4]), although its protective action is much less than that of the oil. One half of an uncooked egg-yolk in the day's formula is started as early as the 6th week of life, and this is increased later to the entire yolk. In many infants the egg-yolk causes yellowish pigmentation of the skin (see Carotinemia, Vol. II, p. 898), and in others it is not at all well-tolerated. The administration of cod liver oil does not do away with the necessity of sunlight, fresh air, exercise and proper diet. The special predisposition of premature infants and of those of certain races has been mentioned previously.

Regarding the prevention of rickets by prenatal measures Hess and Weinstock[5] claimed that cod liver oil given to the mother during pregnancy did not prevent rickets but did mitigate it to some extent, and the same was true of alteration of the diet of the female in animal experimentation. A good deal of study has been given to this subject chiefly by experimentation on animals. It must be said that the possibility of influencing rickets by prenatal measures is not settled. In some painstaking studies carried out in the Department of Pediatrics at the University of Cincinnati by Greenebaum and others, in which the diets of pregnant mothers were increased in total caloric and mineral intake, it appeared that these factors alone probably decreased the incidence of rachitic manifestations in the offspring. The administration of cod liver oil or of a satisfactory diet to a mother during lactation does not protect her nursing infant from rickets, if other conditions are favorable to the development of the disease.

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[1] Amer. Jour. Dis. Child., 1917, LXIX, 1583.
[2] Amer. Jour. Dis. Child., 1924, XXVIII, 329.
[3] Jour. Amer. Med. Assoc., 1923, LXXXI, 15.
[4] Jour. Amer. Med. Assoc., 1923, LXXXI, 818.
[5] Jour. Amer. Med. Assoc., 1924, LXXXIII, 1558.

Treatment of the Attack.—This should be commenced as early as possible. Defects in *diet* should be sought for and removed, as much care being taken to avoid overfeeding as underfeeding. Dietetic changes are necessarily, to an extent, experimental in this disease; but some alteration should be tried, even although the infant seems to be thriving in other respects. If the feeding is manifestly faulty, it should, of course, be corrected in a way to accord with the needs of the patient. (See Feeding, p. 107.) The use of proprietary infants' foods should be stopped. If amylaceous foods have constituted a large proportion of the dietary, these should be abandoned largely for a time. Possibly an increase of the fat in the food may be of service, if well-tolerated by the digestion. Beef-juice, orange-juice, cooked fruit, and broths may be added to the diet. Sometimes scraped or minced underdone meat is of advantage. Fresh vegetables must be tried as soon as the age permits. If no cereal food has yet been given, its administration may be begun. In fact, a varied diet is an excellent means for the cure of rickets; and this should be commenced earlier than is commonly done with normal children; continuing milk at least in large enough amount to supply sufficient calcium to the organism. Caution must be used in making any change, not allowing it to be too sudden or too radical, lest other evils than rickets follow.

All *other diseased conditions*, especially digestive disturbances, must be remedied as far as possible. *Hygienic treatment* is important, particularly life in the open air, massage, cool baths when well borne, and change of residence to the seashore or country. *Heliotherapy* is curative (Hess and Unger).[1] It is not necessary in rickets to expose the entire body, and only the face, forearms and, weather permitting, lower legs need be bare. The short ultra-violet rays of less than 313 millimicrons in length must impinge on the skin, and these rays are almost entirely filtered out by window-glass and to a less extent by clothing, especially if the latter is dark and heavy. The effect of smoke, moisture and altitude on the ultra-violet rays must be taken into account in exposure to the sun, but it is especially the influence of season that is of practical importance (see Etiology and Pathogenesis). The summer-sun is of much more value than the winter-sun. Excessive heat should be avoided by utilizing the cooler morning and afternoon hours in the summer, and excessive cold by using the midday-sun and avoiding draughts in the winter. There is not enough known quantitatively of the ultra-violet rays present in the sunlight, or of their effect, to state with certainty how long exposure is necessary in an individual case, but it should be as long as is possible and practicable (see Heliotherapy, p. 281). Ordinarily ½ hour a day will be sufficient. Ultra-violet radiation produced by artificial means is very effective, but is to be regarded in the nature of a substitute for sun, and to be used when sufficient exposure to the latter is not possible.

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[1] Jour. Amer. Med. Assoc., 1921, LXXVII, 39.

Huldschinsky[1] was the first to use the mercury-vapor quartz-lamp for this purpose. Exposure of the entire body to the rays from the lamp is begun for 3 minutes at a distance of 120 cm. (3.93 ft.). A treatment is given every 2 or 3 days and the time of exposure increased to 20 minutes, and the distance decreased to 75 cm. (2.46 ft.). The carbon arc-light is very satisfactory, the lamp being adjusted to 15 amperes. Daily exposure is begun at 3 feet (91.4 cm.) for 3 minutes and increased to 30 minutes or more, care being taken to protect the eyes with dark glasses. A group of several children may be treated for 1 to 2 hours at a distance of 9 feet (274 cm.) (Hess and Unger.)[2] A few weeks treatment will often result in great improvement or even in cure, as witnessed by clinical evidence and by studies of the blood and with the *x*-ray. *Medicinal treatment* of various sorts has been employed. General experience has shown cod-liver oil to be of unquestionable benefit. It should be given in doses of 1 fluidrachm (3.9 c.c.) 2 or 3 times a day after food. It is usually well borne except in the hottest weather, and even then may be given tentatively. Egg-yolk may be employed as a curative measure, in the manner described under Prophylaxis. The irradiation of cod-liver oil and of milk by ultra-violet light increases their antirachitic value. Phosphorus in doses of $\frac{1}{300}$ or $\frac{1}{200}$ grain (0.0002 or 0.0003) 3 times a day, has been especially recommended by Kassowitz,[3] and Phemister, Miller and Bonar,[4] and used very extensively by others, but its value when given alone is very doubtful in the opinion of many. The experience of Schabad[5] indicates that although phosphorus exerts no influence upon the calcium-metabolism in normal infants, yet when combined with cod-liver oil it favors the retention of calcium in cases of rickets. This supports the clinical experience of the value of the combination of phosphorus with cod-liver oil as reported in many quarters, and the low blood-phosphorus of the disease is an additional argument in favor of its use. Lime in various forms was formerly much given, on the ground that a deficiency of lime in the food produced the disease. It is now well recognized by most authorities that this theory is incorrect, and that the administration of lime can have no direct influence upon the rachitic process. There is sufficient lime in the usual food of infancy. It is only during convalescence, when special demands may exist for an unusually large amount to replace the deficiency which has developed, that the administration of lime may be of possible value. It may be given in the form of calcium citrate or lactate. In this connection the following table is instructive:

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[1] Deutsch. med. Wochenschr., 1919, XLV, 712; Zeitschr. f. orthop. Chir., 1920 XXXIX, 426.

[2] Jour. Amer. Med. Assoc., 1922, LXXVIII, 1596.

[3] Wien. med. Blätter, 1883, VI, 1492.

[4] Jour. Amer. Med. Assoc. 1921, LXXVI, 850.

[5] Zeitschr. f. klin. Med., 1909, LXVIII, 94; 1910, LXIX, 435

TABLE 91.—CALCIUM-CONTENT AND PHOSPHORUS-CONTENT OF COMMON FOOD-MATERIALS[1]

Animal Foods, Cereals, etc.	Per cent.		*Vegetables*	Per cent.		*Fruits*	Per cent.	
	Cal.	Phos.		Cal.	Phos.		Cal.	Phos.
Meats	0.054	1.004	Beans, pea, dried	0.154	0.270	Apples	0.008	0.011
Fish	0.128	1.222	Beans, string, fresh	0.053	0.040	Apricots	0.015	0.025
Eggs	0.071	0.160	Beets	0.014	0.041	Bananas	0.006	0.027
Butter	0.016	0.014	Cabbage	0.041	0.035	Blackberries	0.056	0.036
Butterine	0.016	0.014	Carrots	0.055	0.041	Blueberries	0.032	0.020
Buttermilk	0.123	0.095	Celery	0.067	0.044	Grapes	0.010	0.028
Milk, whole	0.123	0.095	Greens; turnip-tops	0.363	0.043	Oranges	0.031	0.021
Cream	0.105	0.081	Lettuce	0.032	0.032	Peaches	0.011	0.021
Barley, pearl	0.018	0.201	Potatoes	0.011	0.063	Pears	0.013	0.018
Cornmeal	0.006	0.200	Rhubarb	0.043	0.045	Prunes	0.045	0.089
Hominy	0.010	0.309	Spinach	0.046	0.045	Raisins	0.030	0.105
Oatmeal	0.056	0.425	Tomatoes	0.014	0.020	Raspberries	0.051	0.041
Rice	0.009	0.086				Strawberries	0.041	0.030
Wheat flour	0.020	0.094						
Crackers and macaroni	0.020	0.094						
Graham flour	0.026	0.290						
Bread	0.015	0.071						
Maple-syrup	0.088	0.044						
Honey	0.004	0.028						

Iron, and sometimes arsenic, are of advantage when anemia is marked, and general tonic treatment should be given when indicated. Various organs and tissues have been used therapeutically for rickets, among them thymus gland (Mettenheimer),[2] thyroid gland (Heubner,[3] and others), suprarenal body (Stöltzner)[4] and bone-marrow (Amistani).[5] The value of none of these has been established.

The development of *deformities* must be carefully guarded against. Rachitic children should be discouraged from walking until the bones have become firm. The wearing of too thick a diaper predisposes to the development of bow-legs. Carrying the infant always on the one arm is very likely to produce scoliosis. Sitting cross-legged may occasion rotation of the femora, indentation of the tibiæ, and curvature of the forearms from pressure of the hands against the bed or floor. Irregular distortion of the head is to be avoided by altering the position in which the child lies. The treatment of deformities already acquired is considered fully in text-books upon orthopedic surgery. Here only may be mentioned the very great value of massage in strengthening the feeble muscles; the importance of favoring free movement of the limbs by the patient by creeping and by any other form of exercise, properly regulated, which does not directly increase deformity; and the correction of deformities by postural treatment and the like, such as lying upon one side with a pillow under it; lying upon the abdomen; the gentle bending of curved extremities by the nurse; etc., etc., according to the nature of the deformity present. Such measures, combined with general treatment, are often completely efficacious without operative or special orthopedic procedures. By the age of 3 years, however, and often decidedly before this, the ossification of the bones has become too complete to permit of benefit being obtained in this way.

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[1] Bull. No. 27, U. S. Dept. of Agriculture.
[2] Jahrb. f. Kinderheilk., 1898, XLVI, 55.
[3] Berl. klin. Wochenschr., 1896, XXXIII, 700.
[4] Path. u. Therap. d. Rachitis, 1904.
[5] La Pediat., 1903, XI, 560.

CHAPTER II

SCORBUTUS

(Infantile Scurvy)

This disease as seen in early life does not differ materially from that in adults, long known as occurring among sailors and others deprived of suitable food. The adjective "Infantile" is not strictly correct, since the disorder is occasionally observed in children after this period. In infancy it was first described by Möller,[1] but considered by him to be "acute rickets." Barlow,[2] in 1883, made a careful study of it and recognized its true nature, and the disease has often been called after his name. Numerous contributions to its literature have since been made, one of the most extensive being the Collective Investigation of the American Pediatric Society[3] in 1898, in which I[4] chanced to be interested. An able review of the subject has been published by Concetti,[5] based upon these cases with others collected from medical literature, equalling 682 in all. A study of 93 personal cases is given by Morse,[6] and of 60 by Comby,[7] and series have been published by others. My own experience is based upon probably considerably over 100 cases. The statistics which follow are taken largely from the report of the American Pediatric Society, in which 379 cases were analyzed.

Etiology.—*Age* is a predisposing cause of importance, the majority of cases occurring in the latter half of the 1st year, and nearly all the remainder before the end of the 2d year. In Concetti's series 359 were between the age of 6 and 12 months. The youngest case in the Society's report was 3 weeks old. The disease was observed by Owen[8] in a boy of 12 years and by Miller[9] in a child of 6 years. The occurrence of the disease in older children is confirmed by the report of over 200 cases by Tobler,[10] 9 by Weill and Dufourt,[11] and 40 by Chick and Dalyell.[12] *Geographical distribution* seems to have some influence. Infantile scurvy, although nowhere frequent as compared with other nutritional disorders, appears much more common in some countries, such as England, Germany and the United States, than in some others. The previous condition of health seems to have no definite influence. About ¼ of the cases of the American Pediatric Society's series had previously suffered from some digestive disturbance, while ¾ had been in entirely good health. Bad hygiene and similar conditions seem in no way to predispose, and the disease is as frequent, or even more so, among the better classes as among the poor. The active factor is the employment of an *unsuitable diet* through a period of weeks or months. In 356 cases in the series of the American Pediatric Society, the diet was as follows:

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[1] Königsberger med. Jahrb., 1856–7, I, 377; 1862, III, 135.
[2] Med.-Chir. Transac., 1883, LXVI, 159.
[3] Transac. Amer. Pediat., Soc., 1898, X, 5.
[4] Chairman of the Committee.
[5] Archiv. f. Kinderheilk., 1909, L, 174.
[6] Bost. Med. and Surg. Jour., 1914, CLXX, 504.
[7] Arch. de méd. des enf.; 1919, XXII, 225.
[8] Brit. Med. Jour., 1899, II, 1719.
[9] Transac. Amer. Pediat. Soc., 1924, XXXVI, 43.
[0] Zeitschr. f. Kinderheilk., Orig., 1918, XVIII, 63.
[11] Arch. de méd. des enf., 1919, XXII, 561.
[12] Zeitschr. f. Kinderheilk., Orig., 1920, XXVI, 257.

Table 92.—Influence of Food on the Incidence of Scurvy

Breast-milk	alone 10 cases, in combination	2—total	12
Raw cows' milk	alone 4 cases, in combination	1—total	5
Sterilized milk	alone 68 cases, in combination	39—total	107
Pasteurized milk	alone 16 cases, in combination	4—total	20
Condensed milk	alone 32 cases, in combination	6—total	38
Other proprietary foods		total	214

It is evident that scurvy may develop in infants on a variety of foods, and this argues for a common fault in the diet. Looking at the disease as a whole, it seems beyond question that proprietary infant-foods, including condensed milk, are especially liable to produce it, and that this is also true of the heating of milk in a considerable number of instances. In those instances of scurvy in which no heating of the food has been employed, there is clearly the absence from the beginning of the necessary element. In fact the experience of Nobel,[1] demonstrating that infants could recover when given milk which had been boiled for an hour, is in accord with that of many other physicians, to the effect that it may be an original lack of vitamin in certain milks which renders them dangerous, rather than the influence of high temperature alone. Undoubtedly, prolonged heating of the milk diminishes the vitamin-content, but it is possible that this is not to the harmful extent often believed, at least in the case of infants.

It is, in fine, clear that scurvy is a deficiency disease and is due to the lack of antiscorbutic vitamin (vitamin C) in the diet. The antiscorbutic vitamin is present in certain common fruits and vegetables, and the amount of it in milk is dependent on the fodder of the cow. To be absent in breast-milk the mother's diet must be deficient. In other words animals cannot manufacture the substance but must derive it from food. Heat, especially if prolonged, inhibits the vitamin or destroys it. (For a further consideration of the special vitamin concerned see the section on Vitamins, p. 195.)

Active symptoms of scurvy do not at once appear when an infant is fed on an improper food. This may be due to the factor of *individual predisposition*. In support of this, I have seen scurvy develop in several members of the same family during their infancy and have even observed it in twins, as though there might be a family tendency sometimes existent. Wallgren[2] has seen the disease in one of twins on the same diet, arguing still further for individual predisposition. A similar case is reported by Brachi and Carr.[3] Still further illustrating the existence of some individual predisposition is the fact that many hundreds of infants may be fed safely upon some proprietary or other food, in which there is certainly a diminution of the antiscorbutic element, while another infant readily develops scurvy and is promptly cured by a change in diet. It is known from animal experimentation that there is a period of latency before active manifestations appear. For the infant this probably lasts several months. Further, it may well be that a certain amount of storage of the vitamin is possible, or that the body can do without it for a while. It is also true that most foods, even if sterilized or dried, will retain vitamin to a certain extent, especially when they have originally been rich in the substance. The age-incidence of scurvy is readily explained by the influence of the introduction toward the end of the 1st year of a mixed diet which is vitamin-containing, and it is possible that a latent scurvy slowly developing is eliminated by the change in food. In cases reported in older children the food was always deficient in fruits and vegetables.

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[1] Zeitschr. f. Kinderheilk., Orig., 1921, XXVIII, 348.
[2] Acta paediat., 1923, II, 37.
[3] Lancet, 1911, I, 662.

Pathological Anatomy.—The principal lesions are those of the bones, combined with a tendency to hemorrhage in various regions. There occurs a replacement of the bone-marrow by an embryonic connective tissue with few vessels and cellular elements. There is an arrest also in the formation of bone from the osteoblasts, which are few in number, but no production of osteoid tissue devoid of lime-salts such as occurs in rickets (Schmorl).[1] Lust and Klocman[2] and Frank[3] have shown that there is calcium-retention in the early stage and calcium-excretion during healing. The bone-marrow is altered in nature. It loses its lymphoid elements, and the cells are reduced in number, and in place is found a somewhat homogeneous reticulated substance containing few blood-vessels. These changes are most marked at the ends of the diaphyses. Separation of the epiphyses may occur in severe cases as a result of slight trauma. The most characteristic lesion, however, consists in the occurrence of hemorrhage depending upon weakness of the blood-vessel walls, due to degenerative cellular changes (Findlay).[4] This is then always present beneath the periosteum, oftenest in the bones of the lower extremities but frequently elsewhere as well (Fig. 218). Hemorrhage may take place, too, in various other tissues of the body. It is liable to be observed in the muscles and skin about the periosteal lesion, or in the neighborhood of the joints, producing a large, tender swelling, easily recognized during life. Small hemorrhages may occur in any of the serous or mucous membranes, often from the kidneys, sometimes from the stomach or intestine, or in the bone-marrow and the internal organs including the brain. The commonest seat of hemorrhage next to that of the periosteum is the mucous membrane of the gums, especially about the upper incisor teeth. Wolbach and Howe[5] find that there is inability of the supporting tissues to produce intracellular substances, and that this lack of cement-substance in the vessels explains the extravasation of blood. The abnormal permeability of the capillaries also allows the leakage of serum, resulting in edema. In some cases the clinical evidence of edema is slight or even absent, but its presence accounts for the marked changes in weight especially noted as loss of this during convalescence (Nobel;[6] Wallgren[7]).

Symptoms.—Although anemia, loss of appetite, and irritability usually precede the more characteristic symptoms, the first manifestation observed in most cases is *pain in the limbs*. This was reported present in 314 of the 379 cases of the American Pediatric Society's series, in all but 3 of these being in the lower extremities, either alone (149 cases) or combined with pain elsewhere. It often develops so suddenly in infants who have appeared perfectly well, that it may be attributed by the parents to some accident. In other cases the onset is slower, the pain at first being intermittent, and the child often passing a number of days at a time in entire comfort. But in all severe cases the pain soon becomes constant, the tenderness intense, and the slightest attempt at passive movement occasions loud outcries. When the localization can be determined, it is found oftenest in the neighborhood of the ankles or knees. Even approach to the bed-side may cause the infant to scream through fear of being touched. In the majority of cases there is suffering only when the limbs are moved. In 91 of the American Pediatric Society's series, however, pain was reported present even when the infant was entirely at rest. In nearly all instances some degree of *pseudoparalysis* exists, dependent upon pain, and in some cases the legs may be kept so still that poliomyelitis or other form of paralysis is suspected. The absence of motion may range from mere disinclination to move the limbs up to complete disability; varying with the case. At times this is the first symptom noticed (36 of the American Pediatric Society's series). Thoracic pain is sometimes produced by lifting the child in the ordinary manner.

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[1] Beiträge z. path. Anat. u. z. allg. Path., 1901, XXX, 232.
[2] Jahrb. f. Kinderheilk., 1912, LXXV, 663.
[3] Jahrb. f. Kinderheilk., 1920, XCI, 21.
[4] Jour. Path. and Bacteriol., 1921, XXIV, 446.
[5] Arch. Path. and Lab. Med., 1926, I, 1.
[6] *Loc. cit.*
[7] Zeitschr. f. Kinderheilk., Orig., 1921, XXXI, 35.

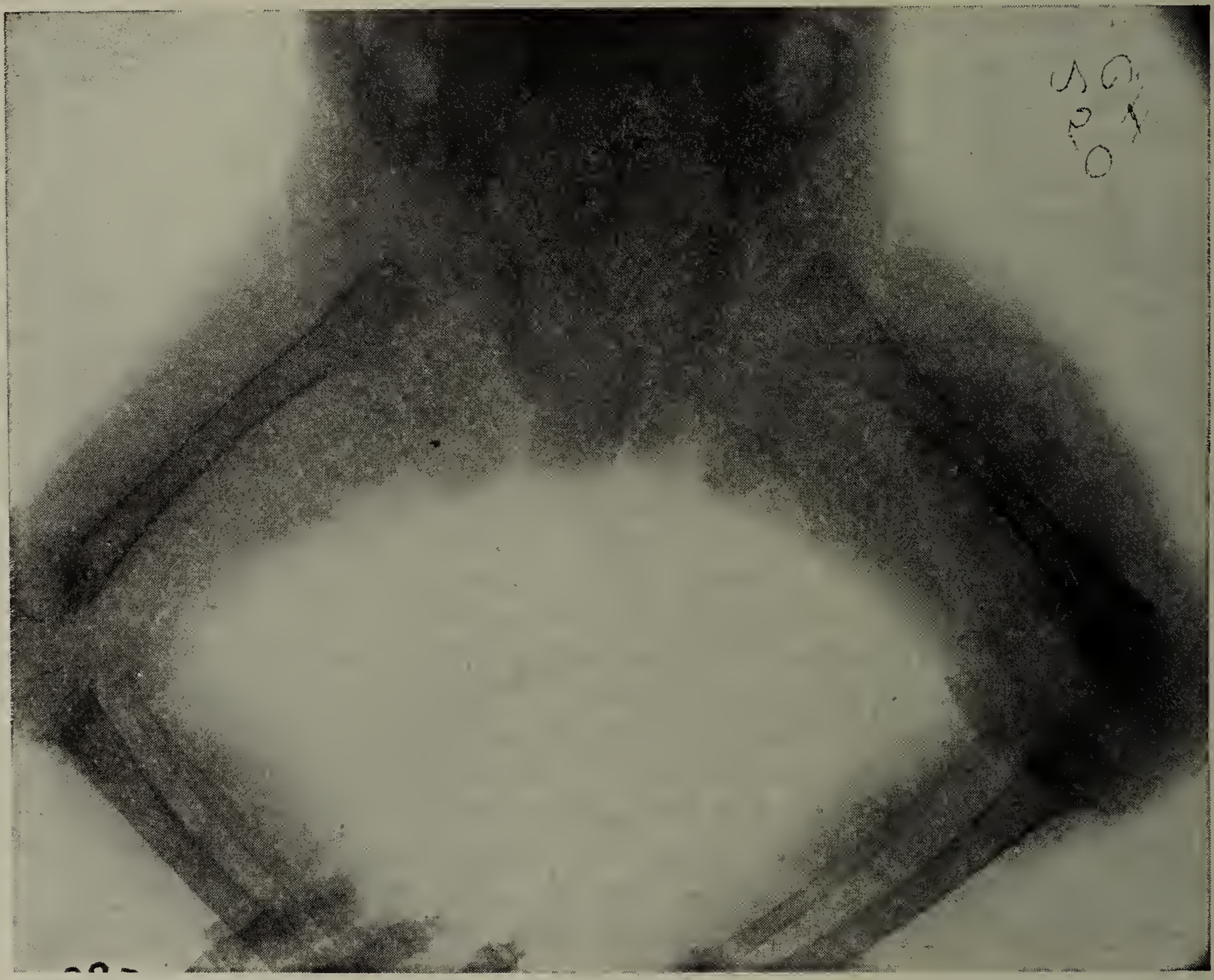

Fig. 218.—Radiograph of Subperiosteal Lesions in Infantile Scurvy.

Child of 11 months, admitted to the Children's Hospital of Philadelphia, Jan. 25. Well until 3½ months previously, when pain and tenderness without swelling developed in the left thigh. A month later the right side became tender and swollen, and has continued so. Condition had been supposed to be sarcoma. Radiograph (viewed postero-anteriorly) shows decided swelling about the right femur, and beginning involvement of the left. Orange-juice given freely. Discharged, entirely well.

About the same time with the development of pain the characteristic *affection of the gums* appears. It was seen in 313 cases in the American

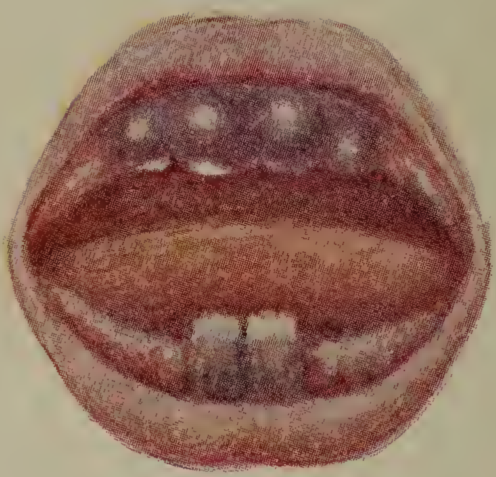

FIG. 219.—INFANTILE SCURVY.

Infant of 8 months. Well-nourished. Fed upon Horlick's Malted Milk. Fretful, nervous, pain in the legs. Had been suffering for 3 weeks. Recovered in 3 days. Illustration shows the swollen, purple gums about the upper incisors, and the purple streaks in the gums of the lower jaw.

Pediatric Society's series and was the first symptom in 42 cases. It consists, when well-developed, in a deep bluish-purple, spongy swelling of the mucous membrane, generally over the upper incisior teeth, sometimes so decided that the teeth become entirely concealed (Fig. 219). Slight hemorrhage from the gums or from the palate or pharynx takes place readily and ulceration is not infrequently present. As a rule the involvement of the gums occurs only in infants whose incisor teeth have already erupted, but this is because the disease is seen so much oftener in those past the age of the first apperance of teeth. In 45 cases in the American Pediatric Society's series no teeth had been cut and in 24 of these the gums were affected. Often at this time *swelling along the shaft of the long bones* can be found. This is hard and tender and is situated usually in proximity to a joint, oftenest the ankle or the knee, but does not involve it. The swelling may be bilateral, with the skin over it shining but usually not reddened (Fig. 220). It may be only slight, but is not infrequently extensive, increasing greatly the general size of the limb. In severe cases the affected limb may become extensively edematous. Quite often *hemorrhages* in the form of ecchymoses or petechiæ are seen elsewhere in various regions of the cutaneous surface (182 of 353 cases in the American Pediatric Society's series). They may be wide-spread and may be found also on the mucous membrane of the mouth and pharynx; sometimes upon the conjunctivæ. They are often among the earliest evidences of the disease. Orbital hemorrhage occurs in a rather small proportion of cases (49 of the American Pediatric Society's series) producing purplish swelling of the eyelids and protrusion of the eyeball on one or both sides (Fig. 221). In severe cases blood may be vomited or passed by the bowel. Hemorrhage from the kidney, usually slight, is commoner than usually supposed, its frequency not depending upon the severity of the case. I[1] have known it to be the only positively characteristic symptom in otherwise doubtful instances, and have observed it present in a large number of cases where the urine has been examined. Blood-casts and granular casts may occur. The loss of blood may sometimes be discoverable only by microscopical examination; in other instances the urine is distinctly red or smoky. Albuminuria independent of the loss of blood is not infrequent.

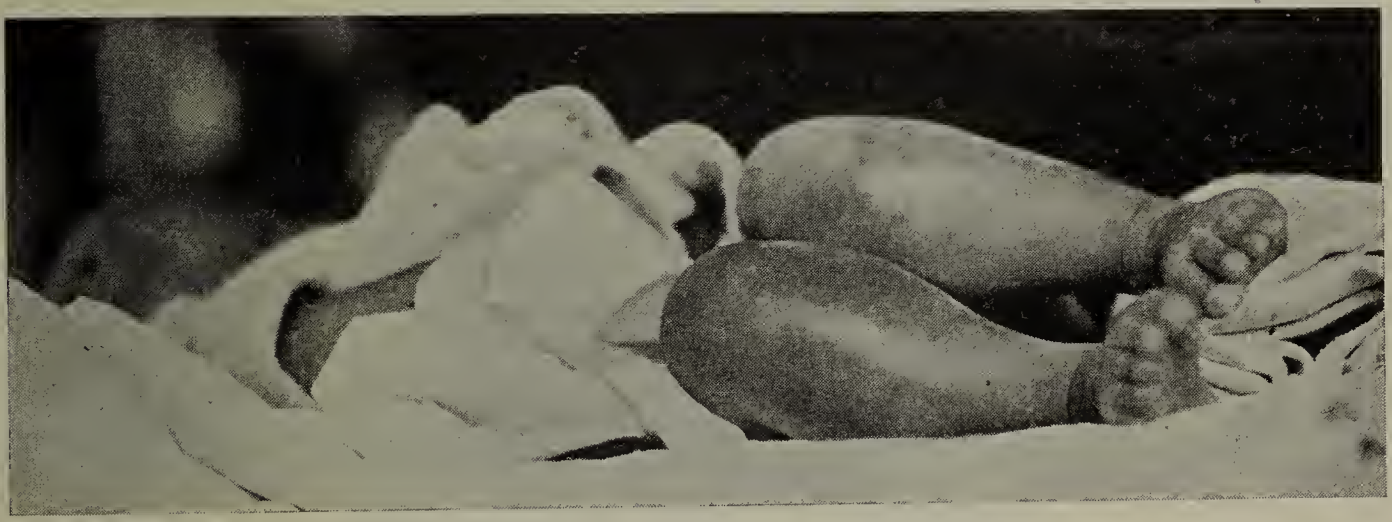

FIG. 220.—INFANTILE SCURVY, SHOWING SWELLING OF LEGS.

Infant of 1½ months, in the Children's Ward of the University Hospital, Philadelphia. Been fed on proprietary food. Pain and swelling in both legs and about the knees. Right arm also involved. Improved on orange-juice, but died of diarrheal disorder.

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[1] Phila. Med. Jour., 1901, VII, 213.

In the meantime the *general condition* of the child suffers. In severe cases there is a progressive wasting accompanied by the development of a cachectic appearance and one expressing the constant presence of pain. The infant lies almost motionless and may cry almost continuously and sleep but little, and its condition is most wretched. There may be moderate fever. The heart is weak, the appetite diminished, and the stools often diarrheal or constipated. Anemia is little marked at first, but becomes decided as the disease advances, being a secondary anemia with especial reduction of the hemoglobin as compared with that of the red cells and with a moderate and inconstant leucocytosis. Hess and Fish[1] and others found the coagulability of the blood slightly diminished, but not to any noteworthy degree. Both Brandt[2] and Bedson[3] have observed an increase in red blood-cells early in the attack of scurvy. After hemorrhages occur the cells diminish. The platelets are usually normal in number but may be increased (Brandt). Beading of the ribs is not infrequent in scurvy. It is perhaps due to a complicating rachitis, but it may disappear under antiscorbutic treatment, indicating its independence of rickets, as maintained by Hess and Unger.[4]

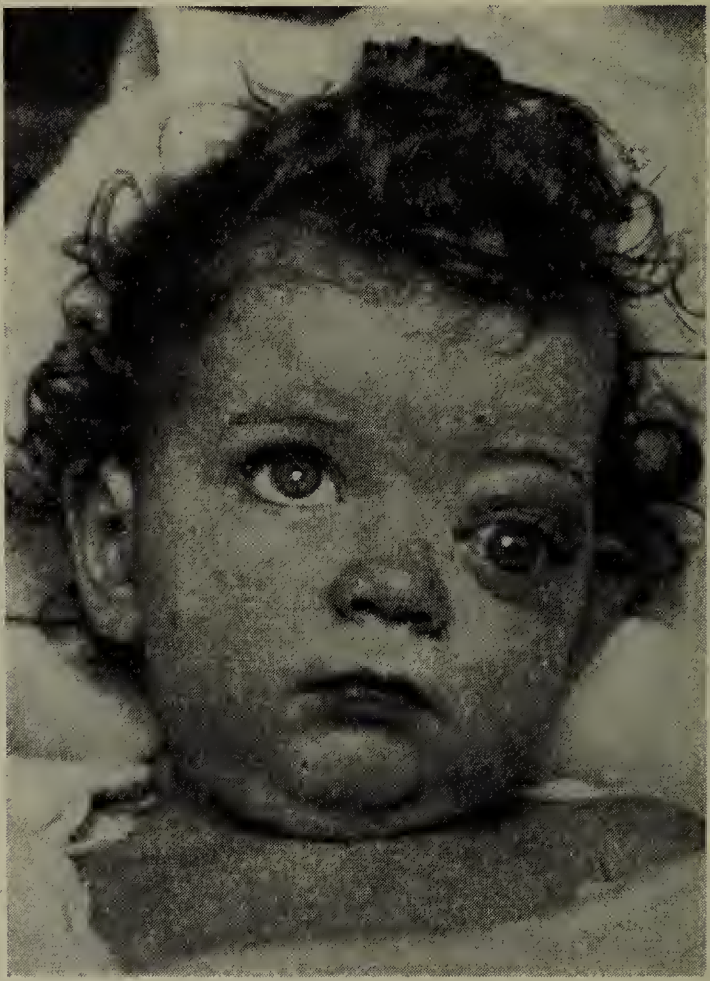

Fig. 221.—Exophthalmos in Infantile Scurvy.

Retta W. Aged 14 months; Children's Medical Ward of the Hospital of the University of Pennsylvania. Fed on pasturized cow's milk-mixture to age of 6 months. Then milk, not boiled; bread; crackers; no orange juice. Pain in left leg began 2 months previously; 3 weeks ago right arm involved, and 10 days ago left eye suddenly became prominent. In hospital exhibited tenderness all over; screamed if touched; no subperiosteal hemorrhage found; gums purple; left eye bulging outward and directed downward, and skin of the lid discolored.

In some cases the classical symptom of subperiosteal hemorrhage is slow in appearing. Yet such manifestations as failure to gain weight, loss of appetite, irritability, and punctate hemorrhages in the mucous membranes or skin may occur, these being, in all probability, due to avitaminosis. If the vitamin is supplied these signs disappear, but if the deficient diet is continued frank scurvy develops. To such a condition Hess[1] applies the term "subacute or latent scurvy." Nassau and Singer[2] use the title "incipient scurvy." They found that 50 per cent. of 30 children had punctate hemorrhages in the skin; 37 per cent. blood in the urine, and 50 per cent. anorexia for several weeks or months before they developed the ordinary evidences of the disease.

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[1] Amer. Jour. Dis. Child, 1914, VIII, 386.

[2] Arch. f. Kinderheilk., 1919, LXVII, 395.

[3] Brit. Med. Jour., 1921, II, 1992.

[4] Amer. Jour. Dis. Child., 1920, XIX, 331

Complications.—By far the most frequent is rickets, and the association of this with scurvy is so common that a necessary connection has been supposed. This, however does not appear to exist, and the two diseases are entirely independent. In 45 per cent. of 340 cases of scurvy in the American Pediatric Society's series it was distinctly stated that rickets was not present, and even admitting that slight rickets may have been overlooked in some of these, it is certainly improbable that it was so in all. Bronchopneumonia or gastroenteric disturbances are occasionally fatal complications.

Course and Prognosis.—The prognosis is excellent in cases promptly treated, and recovery is very rapid. All pain ceases in a week or less. There is scarcely another disease in which the results of treatment are so rapid and remarkable. The swelling of the bones and the constitutional symptoms disappear within 2 or 3 weeks even in severe cases, unless some serious complication be present. The subperiosteal hemorrhage, if extensive, may require months to disappear completely. Any failure of growth, either in weight or length, which may have occurred during the development of the disease is usually quickly made up (Hess).[3] If untreated, however, the disease is chronic, the symptoms gradually becoming more marked and the general condition constantly worse and, although spontaneous recovery may eventually take place in the mild cases, death is liable to occur in the severer ones from malnutrition, exhaustion, or some intercurrent disease after a period of several months. In severe cases it may occasionally happen that there has been enough involvement of the epiphysis to result in shortening of a limb after recovery (Frank).[4]

Diagnosis.—Although the disease is usually readily recognized by those familiar with it, it is the source to others of numerous errors. The failure to make a diagnosis nearly always depends not upon any difficulty attending it, but upon the fact that the possibility of the existence of scurvy has not occurred to the observer. The diagnosis in typical cases rests upon the combination of great pain and tenderness of the limbs, with swelling and hemorrhage beneath the periosteum and in the gums and elsewhere. In general it is safe to assume that pain in the limbs and pseudoparalysis developing rapidly in an infant between 4 months and 2 years of age is due to scurvy, unless examination reveals other causes, even although no affection of the gums is discoverable. Considerable attention has been directed to the "white line" first described by Fränkel,[5] existing in *x*-ray plates at the junction of the epiphyses and diaphyses in the long bones in cases of scurvy. In the print the line, of course, is black. It would appear to be a reliable diagnostic symptom

[1] Jour. Amer. Med. Assoc., 1917, LXVIII, 235.
[2] Jahrb. f. Kinderheilk., 1922, XCVIII, 44.
[3] Amer. Jour. Dis. Child., 1916, XII, 152.
[4] Zeitschr. f. Kinderheilk., 1920, Orig., XXVII, 127.
[5] Fortschr. aus d. Gebiete der Röntgenstrahlen, 1906, X, 1.

(Figs 222 and 223). Wimberger[1] describes a circular shadow surrounding the centers of ossification in the epiphyses of the long bones, and Pelkan[2] refers to a ground-glass appearance of the shaft of the bones, a broadening of the epiphyseal line, and the shadow about the centers of ossification; these being apparent before the onset of clinical symptoms.

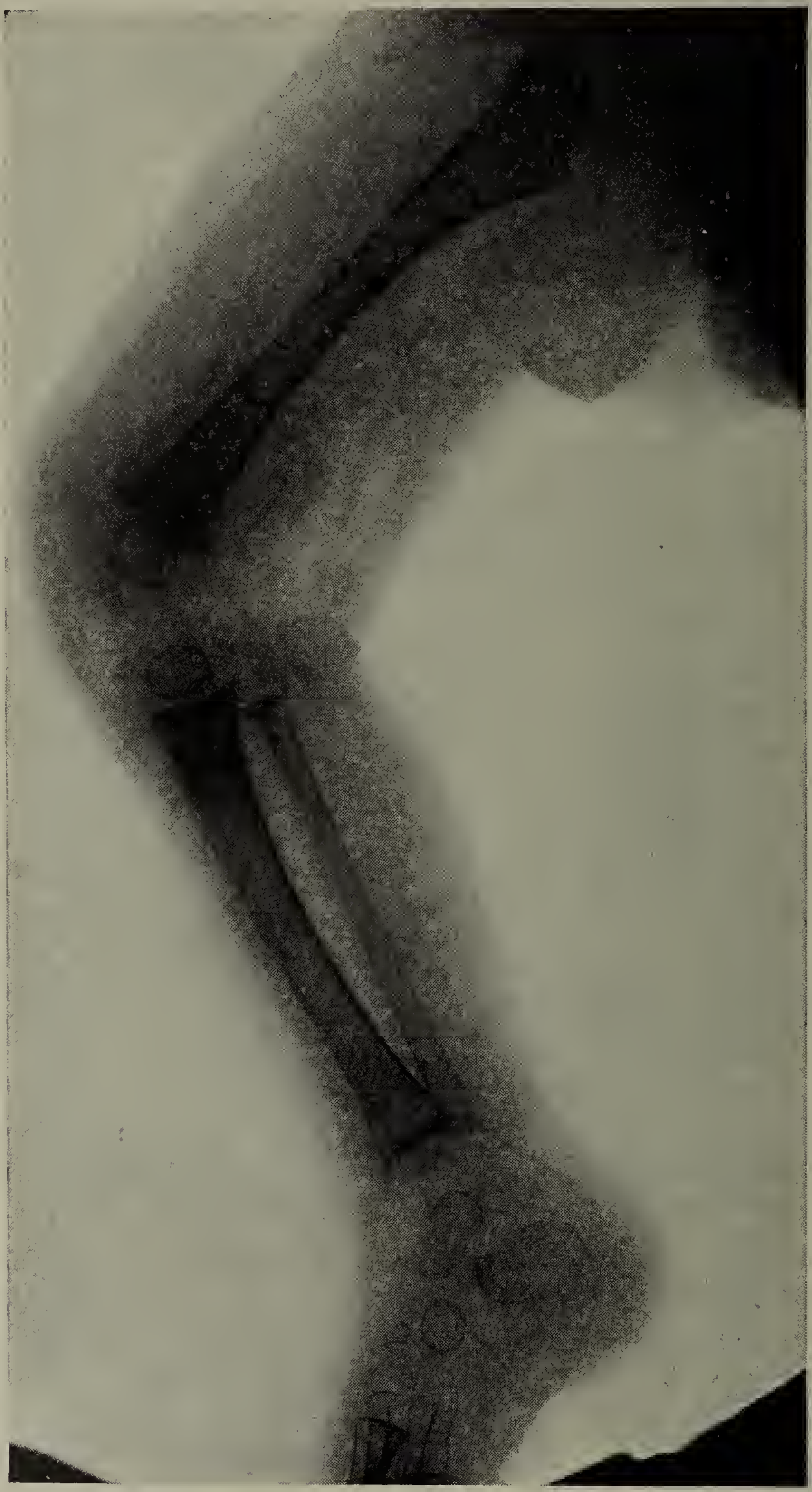

Fig. 222.—Infantile Scurvy. White Line.

Subacute case in an infant admitted to the Children's Hospital of Philadelphia, aged 10 months. Knees swollen; tibiæ tender on pressure; moves the legs very little; gums swollen and mottled. Radiograph taken Jan. 17 shows the "white line," (in the negative; naturally black in the print).

The diagnosis of *rheumatism* is the one oftenest falsely made. This disease, however, is exceedingly rare in infancy. When present it affects the joint and not the bone; there is no involvement of the gums, and the employment of anti-scorbutic treatment is inefficacious. The pseudoparalysis of *syphilis* may suggest scurvy. It occurs, however, usually at an earlier age, produces less tenderness, is oftener limited to the arms, is always situated at the epiphyseal junction and not in the shaft of the bone, and is associated with other symptoms of syphilis. When epiphyseal separation occurs in scurvy, the other symptoms of the disease are so well-marked that mistakes in diagnosis can hardly occur. *Poliomyelitis* is sometimes suggested by scurvy on account of the apparently flaccid paralysis of the legs. There is, however, in scurvy far more tenderness present and no alteration of the electrical reactions. *Osteomyelitis* invades the joints, if it does not begin there, and is attended by fever and pyemic constitutional symptoms quite different from those of scurvy. The painful immobility of the limbs of scurvy may suggest *hip-joint disease* or sometimes *disease of the spine*, or the swelling may be mistaken for that of a *malignant growth* of the bone. The frequent presence of *rachitis* as a complication leads sometimes to the overlooking of the

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[1] Zeitschr. f. Kinderheilk., 1923, XXXVI, 279.
[2] Amer. Jour. Dis. Child., 1925, XXX, 174.

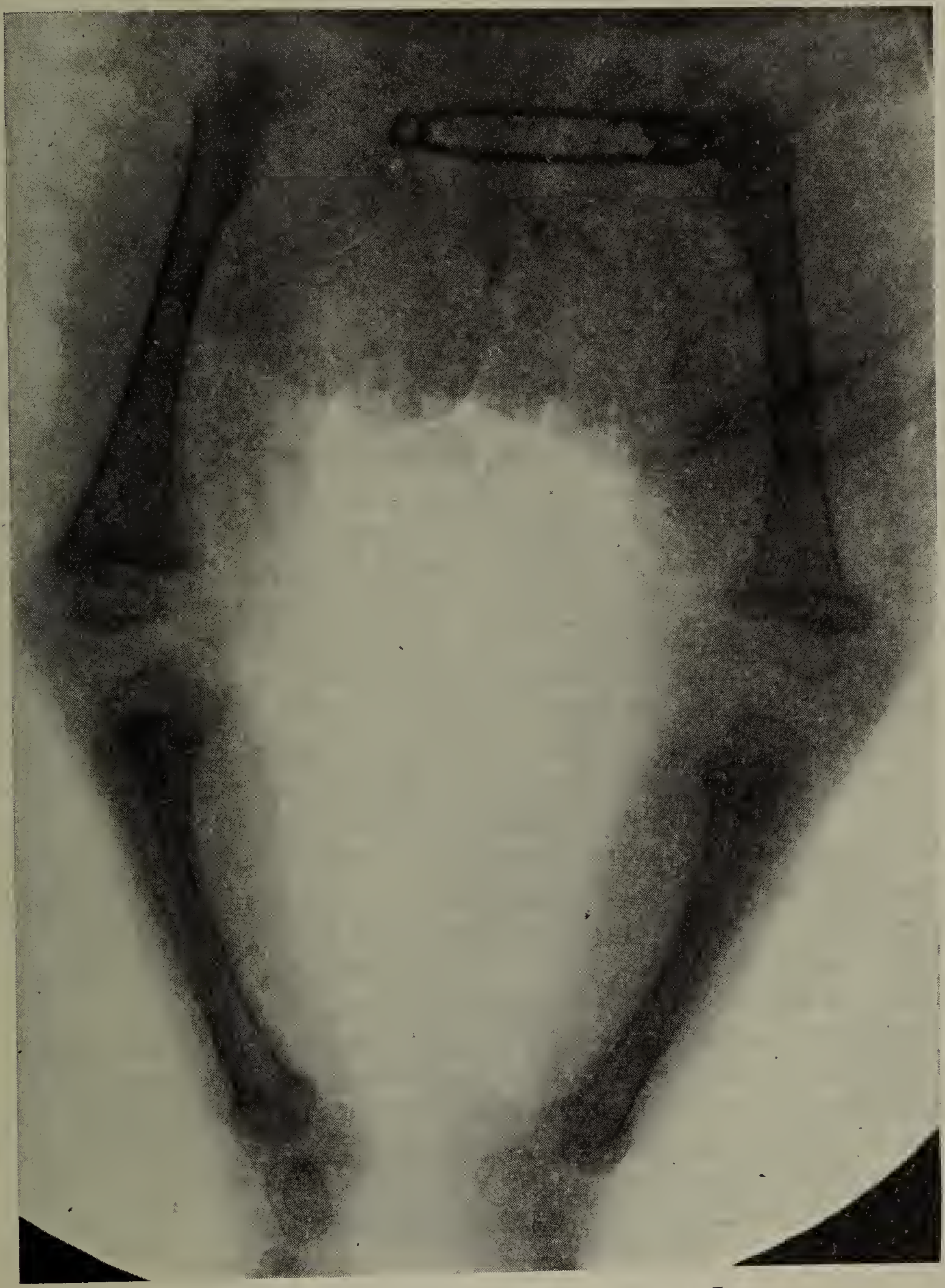

Fig. 223.—Infantile Scurvy. White Line.

Same case as in Fig. 222. Radiograph made Feb. 11. Treatment with orange-juice was instituted, and symptoms were promptly relieved. Radiograph shows the disappearance of the "white line."

existence of scurvy also. There is, however, no hemorrhagic tendency in rickets, and improvement under dietetic treatment is slow. The hemorrhagic rickets of earlier writers is in reality scorbutus.

Treatment.—**Prophylaxis** consists in the avoidance of diet known to predispose to the development of the disease. Inasmuch as there seems reason to believe that the heating of milk and, especially, the employment of proprietary foods, predisposes, the administration of fruit-juices should begin early. The pasteurization of the food is often so important a matter that it should never be avoided merely on account of the possibility of scurvy developing. The evils which threaten to follow the ingestion of raw milk not of the very best quality are far greater and much more dangerous than those produced by scorbutus, and the latter are readily prevented.

Treatment of the Attack.—This consists primarily in correcting the diet. In some cases the mere change from a cooked milk-mixture to one of raw milk is sufficient. In others the abandoning of a proprietary food answers. When scurvy appears the change in diet should always be made promptly unless there is some reason against this. On various grounds, however, it may seem inadvisable to make any sudden alteration in a diet which has, in other respects, been found satisfactory. Fortunately we have almost a specific for the disease in fresh fruit-juice, and this should always be administered. Orange-juice is one of the best, the juice of one-half and later of one orange of average size, sweetened if necessary, being given either in divided portions or in a single dose, usually between feedings. Diarrhea, if present, offers no contra-indication, and may even be controlled by the treatment. One of the few fatal cases I have seen was one in which orange juice was mistakenly avoided on account of the presence of intestinal disturbance. Tomato-juice, canned or freshly cooked, in doses of 1 to 2 tablespoonfuls a day is an excellent antiscorbutic, and is useful in prevention as well as cure (Hess and Unger).[1] Freshly expressed beef-juice is also of service, but not to so great a degree. For children over 1 year, or even younger, potato or other fresh vegetables may be given with advantage. Cooking of vegetables should not be more prolonged than is necessary for digestibility (see Vitamins, p. 195). If the diet is for any reason not altered immediately, the change should certainly be made as soon as possible in order to prevent a recurrence of the disease. Should the anemia and debility not disappear promptly iron or cod-liver oil may be needed later. Of course the infant must be handled with the greatest care while the tenderness remains.

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[1] Proc. Soc. Exper. Biol. and Med., 1918, XVI, 1.

CHAPTER III

INFANTILE ATROPHY

(Marasmus. Athrepsia)

This title, probably first employed by Soranio in the 16th century (Albarel)[1] denotes a progressive and finally extreme wasting of the infant without other symptoms or discoverable anatomical lesions to account for it. The term "athrepsia" was applied to it by Parrot.[2] From this *primary atrophy* must be distinguished all cases of *secondary atrophy* the result of malnutrition dependent upon starvation, congenital syphilis, tuberculosis, or evident chronic disorders of the gastro-enteric tract. This leaves the term primary infantile atrophy certainly useful, since the condition is common, but unsatisfactory, since many cases so designated are undoubtedly secondary to positive but unrecognized causes. The title to a large extent covers the condition designated "decomposition" by Finkelstein (see Vol. II, p. 58), applied to those cases in which the lack of power of digestion and assimilation finally reaches a point at which the infant fails to gain no matter what the diet may be. Whatever the cause of infantile atrophy may be, it is evident that the condition, like icterus, is a symptom rather than a disease.

Etiology.—The disorder is seen chiefly in infants under 1 year living under bad hygienic conditions in cities and fed artifically. It is the cause of many deaths in asylums for infants and in crowded hospital wards, where the title "hospitalism" has often been well applied to it. Some gastroenteric affection frequently precedes it. It has been claimed to depend upon an infection of unknown nature, but of this there is no proof offered. Another theory (Baginsky),[3] assumed an anatomical basis, the lesions being a destructive change in Lieberkühn's follicles, especially in Paneth's cells (Bloch).[4] The presence of any anatomical lesions has, however, not been confirmed by most investigators. That the disease is not dependent upon the absence in the artificial food of ferments or other bodies natural to human-milk, is indicated by the frequency with which infants thrive who are fed entirely artificially. Marfan,[5] however, believes that it is the lack of the specific enzymes of breast-milk which is the factor in infants with feeble digestive power; the vigorous ones being able themselves to produce sufficient of these. Abnormal proliferation of bacteria in the upper intestinal tract has been observed by Bessau and Bossert,[6] and this would interfere with normal digestion. Lack of vitamins has been thought to be causative, at least to some extent. Milk is low in the antineuritic vitamin (vitamin B). Daniels and Byfield[7] have found that they could stimulate growth in infants without changing food-intake when vitamin B was added to the diet; yet it must be noted here that infants dying from atrophy do not present the picture of avitaminosis. It has also been maintained that the imperfect metabolism depends upon defective internal secretions. The presence of diminution in the size of the thymus gland, for instance, has been pointed out by several observers (Mettenheimer;[1] Stokes, Ruhräh and Royal[2]) as accompanying atrophic conditions in infancy. No etiological relationship, however, has been proven. Anderson and Schloss,[3] who demonstrated the absorption of antigenic cow's milk protein, and later observed the appearance of precipitin to cow's milk in the blood, believe that it is not impossible that the absorption of foreign protein may play a rôle in the production of infantile atrophy.

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[1] Ann. de méd. et de chir, inf., 1905, IX, 1.
[2] Prog. méd., 1874, II, 637.
[3] Brit. Med. Jour., 1899, I, 1084.
[4] Jahrb. f. Kinderheilk., 1906, LXIII, 421.
[5] Le Nourisson, 1921, IX, 193.
[6] Jahrb. f. Kinderheilk., 1919, LXXXIX, 269.
[7] Amer. Jour. Dis. Child., 1919, XVIII, 546.

The prevailing view makes primary infantile atrophy a defect in the physiological processes of the organism, as a result of which the infant cannot utilize the food given. This may depend either upon disturbance of the alimentary secretions, including ferments, or upon faulty metabolism causing a loss of power of assimilation in the tissues themselves. According to the first of these, the intestinal ferment is absent which completes the breaking down of foreign (cow) protein and the reconstruction of the homologous protein which the infant can properly utilize. In the latter cause may perhaps be included the suggestion that an acidosis or some other intoxication develops and acts as a determining impulse through the poisoning of the system. Yet the lowering of the alkaline reserve in these cases is but slight (Hutchison)[4] and not constant. It is probably secondary, and has nothing to do with causation (Marriott).[5] Marriott[6] and Utheim[7] have studied many of the phases of atrophy (athrepsia), especially alterations in the chemical and physiological processes of the body. Among the results of their investigations is the discovery of a low concentration of protein in the blood; a diminution in red blood-corpuscles and in hemoglobin, and an absolute and relative diminution in blood-volume; all these indicating destruction of the blood and of other tissues of the body. The low rate of blood-flow also present adds to the destruction of tissues by depriving them of food. The oxidizing power of the tissues is decreased. These various abnormal conditions are end-results rather than primary etiological factors. According to Marriott (see p. 113) the beginning of atrophy may be due to an absence of sufficient gastric hydrochloric acid to permit of cow's milk, with its high buffer-content, reaching an optimum hydrogen-ion concentration in the stomach. Because of this, bacterial growth is not inhibited, rennin action is incomplete, and the chyme is not acid enough properly to initiate the formation of secretin and of hormone-stimulation of the pancreas and biliary secretions. Digestive difficulties and improper assimilation naturally follow.

Atrophy is, therefore, essentially a condition of starvation and dependent either upon insufficient food intake or defective absorption. This latter possibility is supported by the fact that there is greatly increased loss of food-material in the stools (Utheim), and upholds the view of Fleming[8] whose metabolism-studies showed that fats and carbohydrates can be utilized if they can gain access to the tissues. It seems the most reasonable view that the underlying factor in infantile atrophy is failure of absorption and utilization, and that the various pathological, chemical and physiological changes which occur in the body are probably secondary to this.

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[1] Jahrb. f. Kinderheilk., 1898, XLVI, 55.

[2] Amer. Jour. Med. Sci., 1902, CXIV, 847.

[3] Amer. Jour. Dis. Child., 1923, XXVI, 451

[4] Glasgow Med. Jour., 1919, XCII, 227.

[5] Amer. Jour. Dis. Child., 1920, XX, 461.

[6] *Loc. cit.*

[7] Amer. Jour. Dis. Child., 1920, XX, 366; 1921, XXII, 329.

[8] Quart. Jour. Med., 1921, XIV, 171.

Very defective hygiene is another influential factor, especially confinement in poorly ventilated rooms in the poorer parts of the cities, or the occupying of crowded institutions for infants. It is on this account that the disease is comparatively uncommon in rural practice, or in civic practice among the better classes. A further cause often of importance is the presence of a constitutional debility, whether due to prematurity or to ill-health of the parents.

Pathological Anatomy.—Any lesions found must be regarded only as secondary. Fatty changes in the liver are often seen, but no more frequently than in many other disorders in infancy. Bronchitis, bronchopneumonia, atelectasis, hypostatic pulmonary congestion, and intestinal derangements are also often observed, and may, as complications, have precipitated the fatal ending. The lymphatic glands are sometimes enlarged. Atrophy of the mucous membrane of the intestine may be present but appears to be the result of the general atrophy of the soft tissues of the body, and is frequently entirely absent. The pathological picture is that of starvation.

Symptoms.—The essential symptom is progressive loss of weight. The emaciation finally becomes excessive, the face wrinkling all over with each feeble cry, giving the infant the expression of a withered old man (Figs. 224; 225). The fontanelle is depressed and usually small; the bones of the cranium overlap; the chin and cheek-bones are prominent and the eyes large and sunken; the skin of the body is pale and hangs in loose, wrinkled folds. The arms and legs seem to consist of bones with only a thin layer of skin over them; the hands are like claws and they and the feet cold and cyanosed. The outlines of the collar-bones and the ribs suggest a washing-board. The abdomen is sometimes very prominent; sometimes sunken with the thin skin over it showing dilated veins and revealing the outlines of the intestines beneath. The temperature is usually subnormal (Fig. 227), the respiration superficial, the circulation poor, more or less anemia is present, and, toward the end of life, edema also, especially of the face and extremities but sometimes of the whole

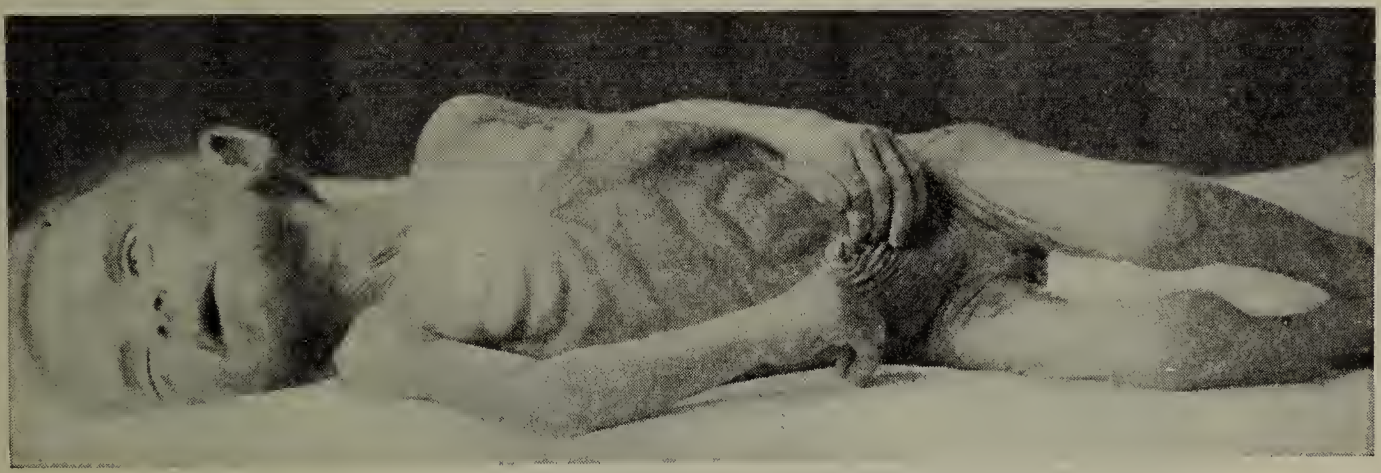

FIG. 224.—INFANTILE ATROPHY.

From a patient, aged 7 weeks, in the Children's Hospital of Philadelphia. Had been boarded out since birth. Temperature 95° to 98°F. (35° to 36.7°C.). 2 to 3 soft stools daily. Death.

surface. (See also p. 728.) At this period a gain in weight may give false encouragement. It is, however, a bad symptom, due to the deposit of liquid in the tissues. The appetite is usually diminished, sometimes voracious; the stools are often regular and well-digested, oftener contain mucus and undigested food. Yet no matter how normal the stools, the loss of weight continues. Vomiting occurs easily; albuminuria is absent, and if edema develops the chlorides of the urine are much diminished. The infant is at first fretful and cries often; later apathetic and lying with little movement. The muscles are usually flabby and relaxed, but in some cases a condition of hypertonia is observed, producing arching of the back, retraction of the head, and flexion of the thighs upon the abdomen. (See Vol II, p. 535.) The whole aspect is one of the most shocking in the realm of pediatrics. The infant appears to be merely a skeleton with a thin covering of skin.

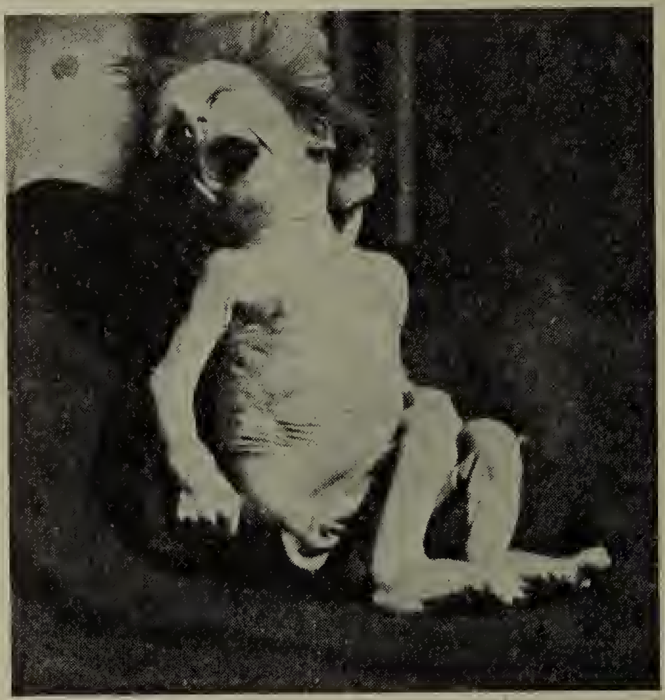

Fig. 225.—Infantile Atrophy.

Child of 4 months, admitted to the Children's Hospital of Philadelphia, weighing 6 pounds, 4 ounces (2835). Been fed on condensed milk and various fresh-milk mixtures. Progressive loss of weight. No active digestive disturbance until within 2 weeks.

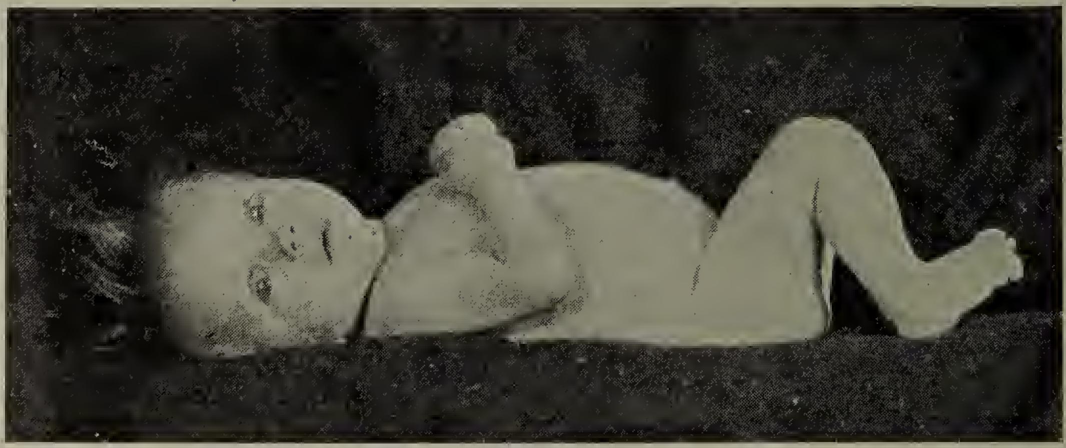

Fig. 226.—Infantile Atrophy.

Same infant as in Fig. 225, 2 months later, weighing 10 pounds (4536), after careful dietetic and other treatment.

Complications.—Infantile atrophy is often complicated by the development of furunculosis and other cutaneous abscesses; erythema chiefly of the nates, scrotum, and back of the thighs; hernia; and thrush. Petechiæ, especially on the abdomen, often develop shortly before death.

(See Vol. II, p. 788, Fig. 416.) Bronchopneumonia, atelectasis, and gastrointestinal derangements are frequent. Convulsions may terminate the scene.

Prognosis.—This is always grave and the majority of well-established cases die after weeks or months of wasting, in spite of the greatest care. The disease steadily grows worse, or in some cases exhibits temporary periods of arrest of loss of weight or even a slow gain, to be followed by sudden, rapid renewed loss. The reduction of the amount of food taken, on the ground that some digestive disturbance may be present, is followed by increased loss of weight; and the same happens if the quantity is augmented. In fact, marantic infants are extremely susceptible to changes in the nourishment, and may be made rapidly worse by this. Death may occur quite unexpectedly from causes not ascertainable, the infant being perhaps found dead in bed, although when last seen it was apparently no worse than usual. In other cases some complication, often of very slight severity, is the cause of rapid failure of strength and of a fatal ending. Anything which causes loss of water from the tissues, such as the excessive evaporation occurring during hot weather, or vomiting or diarrhea, is especially liable to precipitate the termination. It is obvious that atrophic infants cannot stand rapid dehydration. In addition to the loss of water through perspiration, there is in very hot weather apparently a direct effect of the high temperature. Yet under favorable circumstances a certain number of cases can be saved. The longer the condition has lasted and the older the infant, the greater the prospect of cure. Recovery may be slow but finally complete (Fig. 226), and the child in later years may appear no worse for the illness of infancy.

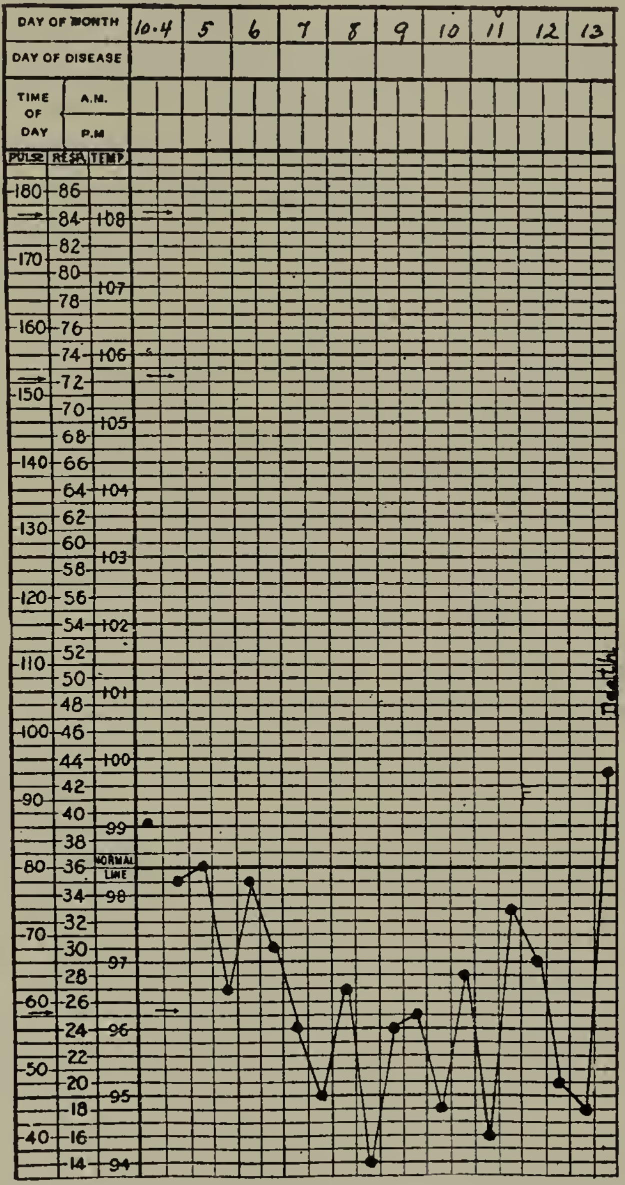

FIG. 227.—INFANTILE ATROPHY, WITH HYPOTHERMIA AND TERMINAL INCREASE OF TEMPERATURE.

Robert M., aged 3 months. Said to have been vomiting more or less since age of 3 weeks. Bottle-fed. Last 2 weeks some retraction of head. Entered in the University Hospital in Philadelphia, Oct. 4, cries a great deal, much emaciated, no vomiting since admission, stools normal. No improvement. Death. Autopsy findings negative.

Diagnosis.—The condition does not differ in symptomatology from the secondary atrophy dependent upon starvation, tuberculosis, or other cause, and the object of diagnosis is to exclude these factors. This is at times most difficult. The existence of tuberculosis may become evident only at autopsy. In other cases the presence of the tuberculin-reaction or the discovery of some localized symptoms indicate tuberculosis. A coincidence of the date of the commencement of failing health with that of weaning or some other change of diet suggests marasmus. Starvation from mere lack of sufficiently nourishing food is readily differentiated by the rapid improvement which follows a proper diet. This applies as well to the gain in weight in starvation-cases which follows operation for pyloric stenosis.

Treatment.—In the way of prophylaxis the continuance of breast-feeding in whole or in part, or, when this is not possible, the careful avoidance of digestive disturbances, accomplished by a judicious selection of cows' milk mixtures, is of great value, as is the obtaining of an abundance of fresh air and the avoidance of excessive summer-heat. For the developed condition, dietetic measures are of much service, yet these can be but experimental as long as the nature of the cause is not understood. The employment of a suitable wet-nurse is greatly to be desired, in fact is almost imperative, although it by no means follows that life can be saved in this way. When it is impossible to feed otherwise than artificially, the same rules hold good, and the same difficulties obtain, as attend the treatment of indigestion. (See Vol. II, p. 138.) Care must be taken in advanced cases to avoid any prolonged starvation-treatment, such as would properly bc employed in acute digestive disturbances. The strength of the infant is not sufficient to tolerate this well. On the other hand, the initial food should be of only such a caloric strength as would, under normal conditions, maintain the weight, without effort to increase it, until tolerance is established. More than this cannot be hoped for at the beginning. The food should be weak, given frequently and in small amounts. As regards the selection of the nourishment, the principles, apply which are operative in the treatment of chronic digestive diseases (Vol. II, pp. 88, 133). In general protein-foods such as buttermilk and caseinmilk offer the best of the unfortunately often slim chances for success. Saccharose or dextrin-maltose preparations, including corn-syrup, are usually preferable to lactose. Weak cereal decoctions may sometimes be of benefit. Tisdall, Drake and Brown[1] have shown that carbohydrate absorption and digestion are usually normal. The greatest difficulty encountered is in finding a food of sufficient caloric value which, at the same time, can be tolerated by the infant's alimentary tract; since the digestive power is feeble in this condition and disturbances of digestion are very easily brought about. Murlin and Hoobler[2] and Talbot[3] have shown that the basal metabolism of atrophic infants is higher than that of normal ones, and it is observed clinically that to produce a gain of weight an unusually large number of calories must be supplied. From 80 to 100 calories per pound (176 to 221 clinically per kilogram) may be required. Because of the intolerance for fat so often present this had better be avoided or given in small amounts at first. This naturally decreases the caloric value of the food, and the deficit must be supplied in other ways. The attempt to increase the fat unduly only increases the loss of weight by producing digestive disturbances. Yet it should be remembered that some cases, apparently those due in part to starvation, may tolerate a normal amount of fat. Some clinicians, indeed, maintain that there is good fat-absorption in infantile atrophy (Parsons;[4] Fleming and Hutchison.[5]) A distinction should be made, not only with fat but also with the other food-elements, between the cases with diarrhea and those with little clinical evidence of indigestion. In the latter stronger food can be given and often will be tolerated remarkably well.

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[1] Amer. Jour. Dis. Child., 1925, XXX, 829.
[2] Amer. Jour. Dis. Child., 1915, IX, 81.
[3] Amer. Jour. Dis. Child., 1921, XXII, 358.
[4] Lancet, 1924, I, 687.
[5] Quart. Jour. Med., 1924, LXVIII, 339.

In milder cases foods with higher fat-content may be tried quite early. Vitamins may be needed by these infants, and the food supplied is often necessarily low in vitamin-content. Consequently orange-juice should be given if it agrees, as it usually will if begun in small amounts; and reliable and tested vitamin preparations especially those containing vitamin B are useful. (See Vitamins, p. 195.)

While the attempt is being made to adjust the food, other measures of relief must be followed. Whereas about 3 ounces of fluid per pound of body-weight (195 c.c. per kilogram) per day are normally required, more than this must be supplied where wasting is great and need of liquid in the tissues is evident, and if it cannot be taken orally it must be introduced in other ways. A certain amount may be retained by the bowel if a few ounces are taken slowly every 3 or 4 hours. Hypodermoclysis and intraperitoneal injections may often be given with greater advantage. It should be remembered, however, that these measures are to be used only as indicated by evidence of dehydration; the primary need of atrophic infants being food and not water. Glucose, so readily utilized by the organism, may be administered by mouth in 5 per cent. solution in place of plain water, and in the same strength may be used by hypodermoclysis. The intravenous injection of a 5 per cent. solution of glucose by the longitudinal sinus, giving of the solution ⅙0 of the body-weight as recommended by Dunn,[1] Helmholz and Sauer[2] and others, is a useful procedure, and may act in other ways than simply that of supplying nourishment. The injections may be employed daily. Pittfield[3] was the first to recommend the employment of insulin with carbohydrate in the treatment of atrophy and malnutrition. It may well be used according to the manner advised by Marriott[4] for "buffering" stronger solutions of glucose than could be employed without it. By this method ½ fluidounce of a 20 per cent. solution of glucose is given intravenously for each pound of body-weight (32.6 c.c. per kilo), insulin being added in the proportion of 15 units per 100 c.c. (3.38 fl.oz.) of glucose solution. The combination may also be given by hypodermoclysis. Barbour[5] advises administering the glucose by mouth and the insulin subcutaneously. Tisdall, Brown, *et al.*[6] could discover no good results which were clearly attributable to the combination, and found that hyperglycemia might be produced even when the glucose is apparently sufficiently buffered by the insulin. This has been our own experience.

Of great value are repeated injections of blood. Fifteen c.c. of citrated blood per pound of body-weight (1.12 fl. oz. per kilo) may be injected intravenously (See Intravenous Injection, p. 274), or into the peritoneal cavity as suggested by Sansby and Siperstein[7] (See Intraperitoneal Injection and Intraperitoneal Transfusion, pp. 260 and 277). Intramuscular injections of blood are less effective.

Freeman[8] claimed good results from the subcutaneous employment of horse-serum and Epstein[9] used serum from other animals. Daily injections are given for 8 or 10 doses beginning with 0.5 c.c. (.017 fl. oz.) and increasing to 2 c.c. (.07 fl. oz.). Clarke and Dow[1] report stimulation of growth and tonic effects from subcutaneous injections of sodium cacodylate, giving ¼ to 1 grain (.016 to .065) every 4 days.

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[1] Arch. of Pediat., 1917, XXXIV, 425.

[2] Transac. Amer. Pediat. Soc., 1919, XXXI, 256.

[3] N. Y. Med. Jour., 1923, CXVIII, 217.

[4] Jour. Amer. Med. Assoc., 1924, LXXXIII, 600.

[5] Arch. of Pediat., 1924, LXI, 707.

[6] Amer. Jour. Dis. Child., 1925, XXX, 10.

[7] Jour. Amer. Med. Assoc., 1923, LXXX, 1763.

[8] Arch. of Pediat., 1917, XXXIV, 425.

[9] Monatsschr. f. Kinderheilk., 1923, XXV, 72

Of great importance is the obtaining of fresh air. The disease diminishes in hospital-practice in proportion as the air-space of the room is increased, and especially through the use of open sun-parlors or roof-gardens, unless the presence of unusually low body-temperature precludes this. Indeed in hospital-practice, often one of the best plans of treatment is to send the child with beginning hospitalism home, if it can be taken care of there in a manner at all suitable. The fact, however, is too often evident that the bad hygienic conditions of the home are even worse than the retention of the infant in the hospital.

General massage with oil is of value in many instances. Alcoholic stimulants in moderate dose are serviceable if the fontanelle is much depressed and the general circulation poor. Should the temperature of the body be below normal it must be maintained by the employment of hot water-bottles, electric pads and the like. In the case of young infants with threateningly low temperature it is best to envelope the child in cotton and keep it in a suitable warm room, which must, however, receive sufficient previously warmed fresh air. (See Premature Infants p. 284.)

CHAPTER IV

MALNUTRITION

By the general and rather vague term Malnutrition we may cover all the secondary atrophies and instances of failure to gain in weight, independent of whether the causes are organic or functional; reserving the title "Infantile Atrophy" or "Marasmus" for the severe cases with the etiology and symptomatology as described in the previous chapter. The term Inanition, literally "emptiness," may be regarded as expressing an unusual degree of malnutrition in reality dependent upon a starvation. The distinction is, however, usually not very sharply drawn.

Etiology.—Malnutrition is extremely common, and depends upon various causes. Based upon a strict application of the 7 per cent. deficiency weight-height relationship (see pp. 19, 729), Emerson[2] estimated that at least ⅓ of school-children are malnourished, and at least ½ decidedly under weight. A constitutional debility may be present in children from the time of birth, and render them always below normal in their strength, weight, and resisting power to disease of any sort. (See Congenital Asthenia p. 322.) Many cases of premature birth are to be placed in this category. Children with a congenital excessively nervous development constitute a considerable proportion of the instances of malnutrition in childhood. Not only may tuberculosis and syphilis of the parents produce a state of malnutrition in the offspring, who, however, may not show evidences of the existence of either of these diseases, but parents delicate

[1] Amer. Jour. Dis. Child., 1920, XIX, 260.

[2] Nutrition and Growth in Children, 1922, 230.

or diseased from any other cause may be unfit to produce children capable of thriving normally. The frequent occurrence of poor and of good nutrition in different children of the same family and even in twins has led Emerson[1] to the belief that heredity has little to do with malnutrition.

In probably the majority of cases, however, the condition is not a constitutional one observable at birth, but is acquired later, the infants appearing healthy when born. The presence of chronic disease of any nature may be the cause, to be mentioned here being tuberculosis, syphilis, rachitis, diabetes, malignant growths and chronic disturbances of any of the organs of the body, most frequently the gastroenteric tract. Even the occurrence during early infancy of an acute disorder, oftenest digestive in nature, may leave a chronic malnutrition in its train. Continued improper or insufficient diet and the constant living under bad hygienic conditions with lack of sufficient fresh air are perhaps the most frequent factors; and this applies not only to the children of the poor to whom too little care is given, but equally well to the many over-housed, pampered and over-cared-for children of the well-to-do. Finally, a more or less acute inanition from insufficient food may occur at any period of early life, but oftenest in infants. Here the food may as a whole be ingested in much too small an amount, either because it it too weak in character or scanty in quantity, or because the child refuses it; or may be sufficiently strong in some respects but very deficient in others, and inanition result. In other cases which have suffered from some long-continued digestive disease, severe inanition rapidly develops in consequence of the curtailing of the amount of food which was intended to act as a therapeutic measure.

Symptoms.—As the name denotes, the symptoms of malnutrition are those of imperfect nutritional development. In *infants* the growth of the body in weight and often in length as well is much below normal. The strength is poor; the muscles flabby; the child cannot sit or stand at the age when this should normally have been learned; evidences of digestive disturbances are a not infrequent accompaniment; rickets is not uncommon, although in the severest cases it is noteworthy that rickets is absent or but little developed. The digestive and assimilative powers are feeble, and the physician is constantly occupied by the effort to find a diet which will be sufficiently nourishing and yet which will not disturb the digestion and produce a loss of weight.

The cases of **acute inanition** present symptoms of interest. In many such, as a result of the causes mentioned, a very rapid and dangerous loss of weight takes place, often following upon a previous moderate degree of malnutrition; sometimes occurring rapidly without this. With the loss of weight is the development of low body-temperature (Fig. 228), sunken fontanelle, hollowness about the eyes, pallor, feeble pulse, and all the signs of great depression of strength. The condition suggests infantile atrophy, but is distinguished by the prompt improvement which occurs when sufficient proper nourishment is given, if this has not been too long delayed.

Another interesting form in infants is the **malnutrition from excess of starch.** This appears to be a common disorder in those regions, as in parts of Germany, where a diet largely of cereal decoctions is employed for infants by the laity. It has been designated *Mehlnährschaden* (Czerny and Keller;[2] Klose[3]). In the United States it is certainly not so common. Sometimes it follows the too long continuance of an amylaceous diet, prescribed for infants who have been suffering from diarrhea with inability to digest milk. It is to be noted that the occurrence of this form of malnutrition generally develops only when such a diet has been continued for a considerable time. The temporary substitution of milk by starchy food is a well-approved course of treatment of some digestive disturbances; but the danger of the continuance must be borne in mind. The appearance of symptoms would seem to depend not so much upon the presence of the excess of starch as upon the relative deficiency of protein and of fat, the disease being one of deprivation. Three types may be recognized: First the *hypertonic form* (see Vol. II, p. 535) in which there is a rigid condition of the muscular system with opisthotonos, adduction of the arms and thighs, and flexion of the forearms and legs. This is the least common variety; and hypertonia may be seen in other nutritional disturbances than those dependent upon a starchy diet. Second, an *atrophic form* which does not differ much in appearance from ordinary inanition from starvation, although the condition of hypertonia is often associated. In this variety there has been no addition of salt to the diet. Third, the *hydremic form* in which salt has been added freely to the food or has not been sufficiently excreted in the urine, and as a result the water is retained in the tissues. This retention of water makes the infant at first appear well-nourished, but gradually the presence of edema becomes very noticeable, and the increase in weight is decided. In all forms the skin is pale and of a pasty appearance, and the infant is especially liable to the development of infections, chief among them being suppurative processes of the skin and xerosis of the cornea and conjunctiva with destruction of the eye often following.

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[1] Nutrition, Health and Growth, 1922.

[2] Des Kindes Ernährung, etc., 1906, II, 62.

[3] Med. Klinik. 1915, XI, 881.

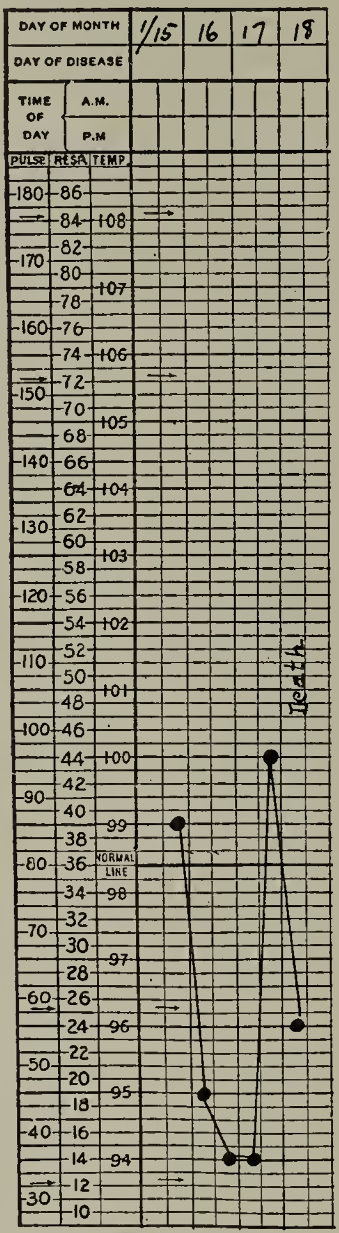

Fig. 228.—Inanition from Starvation on Greatly Diluted Condensed Milk.

Samuel S., 9 weeks old, Jan. 15. Breast-fed 1 month, then given ½ drachm condensed milk in 5 ounces of water. Grown progressively weaker. Now very weak, slow pulse and respiration, subnormal temperature, scarcely able to move, mucous membranes pale, abdomen scaphoid, child remains in any position placed, almost unable to swallow, fed by pipette. Death on 3d day after admission to the Children's Ward of the University Hospital, Philadelphia.

In *older children* the symptoms of malnutrition show themselves in various ways, such as anemia; coldness of the extremities; flabbiness of the muscles; fretfulness; nervousness; poor appetite; constipation; coated tongue; insomnia; fatigue on slight exertion, the child being unwilling to exercise, or too restless and active and growing fatigued and irritable before evening comes on. Sometimes the children are above the average mentality; sometimes they easily become mentally tired, and the ordinary school-work is difficult and burdensome. Of great importance in diagnosis is the so-called fatigue-posture, in which there is flat chest, round shoulders and prominent abdomen. Often this posture may be well-developed and the body thin, although the face may be fat. Very slight causes are liable to produce outbreaks of indigestion. There is very little resisting power to attacks of disease of any sort, illnesses developing readily and leaving a distinct impression on the system.

What shall be designated malnutrition so far as the weight is concerned has been a matter of some discussion. It is manifest that a child may be of short stature with weight below that for his age, but normal for his height. Such a child cannot be said to have malnutrition, unless there are other symptoms present. On the other hand, if he has grown rapidly and is tall for his age, his weight may not have kept pace with this, but he is not necessarily a subject of malnutrition. The physical development in other respects is to be taken into consideration. It has been the custom not to speak of malnutrition in this connection unless the weight is more than 7 per cent. lower than it should normally be for the height attained (see Tables 13–15, pp. 18–20), but this rule should not be applied too rigorously.

Course and Prognosis.—This varies with the age and with the cause. Children who are born constitutionally predisposed to malnutrition cannot be expected to become hardy with any rapidity if at all. In those in whom the disorder depends upon some existing or previously existing disease, the duration of the condition and the ultimate outcome depend upon the nature of this and the possibility of removing it if still present. Very often the state of malnutrition disappears to a large extent when later childhood is passed; in other cases it is permanent. There is always, too, the danger of a fatal termination from the occurrence of some of the numerous complicating disorders to the development of which the subjects of malnutrition are especially liable. The immediate danger from malnutrition is much the greatest in young infants. The older they grow the greater the chance of final recovery, provided proper therapeutic measures can be enforced.

In the nutritional disturbances dependent upon an excessively starchy diet, the prognosis is very uncertain, being worse in proportion to the youthfulness of the infant, the length of time the faulty diet has been employed, and the duration of the symptoms. The mortality in young infants is high. Yet patients will sometimes improve rapidly when a proper diet is instituted. The danger from infections of different sorts is great, and the infants appear to have but little resisting power to them.

The acute inanition from insufficient nourishment generally offers a favorable prognosis, if it has not advanced too far; and recovery is rapid when the dietetic defect is remedied. Very often, however, the patient has developed through lack of food an intolerance for it, and the prognosis is then unfavorable. In this connection must be emphasized the danger of starvation-treatment as the first step in the course of the management of cases of indigestion in infants much debilitated. It is the proper treatment in acute cases with good general health; but in those with but feeble strength it may precipitate a most serious state of inanition if continued unduly.

Treatment.—The treatment of malnutrition depends naturally upon the cause. Inasmuch as this is so often dietetic and hygienic, the first effort must be made to discover what may be the error in this particular and to correct it.

As regards the malnutrition of **infants,** inanition occurring in those breast-fed requires a careful study of the composition and amount of milk secreted, as well as the determining, in the event that nothing is wrong in this particular, whether the infant, perhaps weakly, is actually drawing sufficient nourishment from the breast. It may be necessary to pump or express the milk and to give it from a bottle or dropper or by gavage. In infants artificially fed who refuse their nourishment, feeding by gavage may be necessary. In young infants in whom the state of malnutrition depends upon digestive disorders, the securing of breast-milk is often an essential for recovery. This may be fed in small amounts at frequent intervals, or in larger quantities with longer pauses, depending upon the attendant symptoms and the results obtained. When the employment of substitute-feeding is unavoidable, the food must be carefully selected according to the indications of the symptoms. (See Chronic Gastritis, p. 88; Chronic Intestinal Indigestion, p. 133 etc.) In general the high-protein foods, such as casein-milk and buttermilk, are most efficacious. Sometimes malt-soup gives excellent results; in other cases whey, white of egg, and the like are valuable as temporary expedients. It is necessary to make sure that the food is of sufficient strength to supply the caloric needs. When the digestion permits excellent results may be obtained from the use of such high caloric foods as butter-flour mixture (p. 169) or lactic acid milk with corn-syrup (p. 156). In the case of malnutrition dependent upon an excessive amount of starch, the dietetic remedy is obvious. Inasmuch as the trouble is not so much the starch as the nearly or complete absence of fat and protein and in some instances of salts, these ingredients must be supplied and the starch withdrawn or reduced in amount. In cases of other nature the addition of 5 per cent. or more of starch in some form to the mixture is a very helpful procedure, and thick cereal-feedings of the type used in the treatment of pyloric stenosis have been employed with benefit (Mixsell).[1]

Next to the management of the diet in infancy that of the hygiene is of the most importance. The chief error here, both among poor and rich, is deficient fresh air, the child being confined by night and often by day to close, poorly ventilated rooms. Whatever risk there may be from exposure, that of keeping the infant housed is certainly much greater. There is naturally a middle course advisable in all cases of much debilitated infants, and enthusiasm for open-air treatment should not lead to exposure to chilling, especially in patients with a tendency to low body-temperature, who bear this very badly. The employment of hot water-bottles and the careful screening against cold wind will permit of open-air treatment in many cases, while in others the keeping of the infant in a well-warmed room with the windows open may constitute a satisfactory compromise.

A number of other measures may be serviceable, according to the needs of the case. In atrophic infants with vomiting or diarrhea, who are taking or retaining but little nourishment, frequently the most pressing need is that of water in the tissues. In such an event enteroclysis with normal saline solution is often of the greatest value. When it is not satisfactorily retained, hypodermoclysis or intraperitoneal injections may be required. (See Treatment of Infantile Atrophy.) Cardiac stimulants should also be used as the occasion demands. In other cases of malnutrition gentle massage, such as the mother herself can give, is of service. Ferreira[2] confirms the experience of Freeman[3] on the stimulating action of injections of horse-serum (see p. 725).

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[1] Arch. of Pediat., 1919, XXXVI, 449; 1920, XXXVII, 486.

[2] Arch. Lat.-Amer. de Pediat., 1920, XIV, No. 2. Ref. Jour. Amer. Med. Assoc., 1920, LXXV, 278.

[3] Arch. of Pediat., 1917, XXXIV, 428.

In the treatment of malnutrition in older children the causes are so manifold that only a careful study of the conditions which have obtained from infancy onward can guide to a successful result. Here, too the diet is often at fault, although not so predominatingly as during infancy. In all classes of society the greatest ignorance and carelessness are often shown by parents in these matters. The appetite for the meals is spoiled by allowing cakes, candies, and the like between meal-times. The fancies of the child are made the guide rather than what is best for it; sometimes on the ground that if denied the manifestly improper articles of food it will take nothing. The ultimate result is, however, always bad. The diet should be simple, yet sufficiently varied if food of one nature palls, as is often the case. The proper diet for normal children of various ages has been described earlier (Chap. VII, p. 181). It may require great modification in malnutrition, dependent upon what digestive disturbances may be present. Whether the child shall be urged to eat depends upon the individual. As a rule, if voluntarily going without a meal the patient will often have a better appetite for the next and will make up the deficiency. Yet some children seem to have been born with a constitutional lack of appetite, and in these unobtrusive persuading to eat is often a necessity. This is, however, frequently much overdone by parents, and awakens in the child a hysterical objection to the taking of food of any sort. (See "Anorexia nervosa," Vol. II, p. 68.) When the appetite is poor proper selection should be made of the sort of food which will nourish most. Thus, for instance, I have seen children satisfy themselves when beginning a meal with a thin soup, and refuse the following more substantial substances; and have succeeded in modifying this by giving the soup as a dessert, it making no practical difference whether this article was then declined or not. If the child prefers liquid to the exclusion of solid food, the former may be withdrawn for a time and only the latter given, the fluid intake as water being insisted upon. Association with other children who have normal food-habits is helpful. As a rule not more than 24 ounces (710 c.c.) of milk should be allowed per day, but to this there are exceptions.

The whole course of the child's daily life must be supervised by the physician. There is in many instances far too much confinement to the house or in school, and far too much pushing in an educational line. The child spends the greater part of both morning and afternoon in school, and on returning home has lessons in piano-music, modelling, foreign languages, drawing, and so on. There is no opportunity given for active outdoor amusement, and it is not long until a distaste for this arises, and the patient has to be driven from the house to partake in it. In other children, on the other hand, the fondness for outdoor play exists to such an extent that long before evening the child is in an over-tired state, ready to cry on the slightest provocation, and a night troubled by insomnia follows. Parents often fail to remember that a child never knows enough to stop play before becoming tired out. The same nervous condition arises from over-excitement of any other sort, such as going frequently to the theater, moving-picture shows, children's parties, and the like.

Enough has been said to illustrate the great necessity of a thorough study of the child's daily life and the proper regulation of this, and to emphasize the fact that dietary measures alone are not sufficient to effect a cure. In addition to the correction of the bad food and bad health-habits already enumerated physical defects, such as diseased adenoids and tonsils and carious teeth, should be treated. Certain other therapeutic measures may be considered. Massage and gymnastic training are useful for children who take too little exercise, but not suitable for those who take too much. It is often difficult to determine whether the fatigue-posture is congenital and the cause of symptoms, or is acquired and the effect of the malnutrition. In any event it should be corrected. We have had good results from the employment of the following exercises in which the child must use specific muscles: (1) Stand with heels together and 6 inches (15 cm.) from the wall; inhale, rising on toes; raise arms forward and upward and backward; touch wall with tips of fingers; exhale, slowly lowering the arms to the sides and heels to floor. (2) Lie flat on floor. Toes hooked under chair or dresser; raise body from floor, keeping knees flat and elbows and hands folded across chest. These exercises to be done 5 and later 15 times morning and night. When the fatigue-posture is marked and has been long-continued, it may be difficult to correct by exercises alone, and a proper support worn for several months should be used as an adjunct to other measures. A type with which we have had considerable success is that recommended by Nicholson[1] and consists of an abdominal pad and a back-support to which shoulder-braces are attached. Rest, recumbent, in the middle of the day, not insisting upon sleeping in the case of those in later childhood, is sometimes remarkably efficacious, and improvement may be impossible without it. During the rest-hour or at night the child may sleep on its back with a small pillow between the shoulders, or on its face with a pillow under the abdomen. A cool morning sponging or affusion is often an excellent stimulating measure if the brisk after-rubbing brings about a good reaction. The bath-room should be warm and the child should stand in the tub in a few inches of warm water, this being used for cleansing purposes. Finally, the common observation that the children enjoy the best health when out of town during the summer-months, indicates that in many instances life in the country during the whole year is what will do most good.

The employment of drugs occupies a very minor position in all cases of malnutrition of any age. They should be used chiefly symptomatically as the need arises. Particular attention must be paid to the relief of the chronic constipation which is so often an attendant. The anemia which is a frequent symptom may need a prolonged course of iron. Cod-liver oil is excellent in many instances, provided it does not produce coating of the tongue and loss of appetite. Children usually take it readily if made into an emulsion or given combined with a syrupy extract of malt. Further dietetic and other treatment of the poor appetite which is so commonly present will be considered when discussing Chronic Indigestion (pp. 88 and 133).

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[1] Penna. Med. Jour., 1923, XXVI, 296.

CHAPTER V

RHEUMATISM

Rheumatism is often classified among the infectious disorders, and certainly shows a close similarity at times to infections of a septic nature. For various reasons, however, it seems more convenient to consider it provisionally among the general and nutritional diseases; at least until such time as the invariable causative relationship of a specific germ may be definitely proven beyond possibility of question.

Etiology. Predisposing Causes.—*Age* is important among these. In infancy rheumatism of any form is very rare. I[1] have seen but 1 instance of articular involvement at this period, occurring in a male infant of 8 months, and Miller[2] writing in 1899 found recorded under 1 year of age only 19 cases, in addition to 1 of his own, which seemed properly to belong to this class. Yet a few instances have been reported which appear to have been congenital (Abrahams,[3] Poynton and Paine[4]), and Richdorf and Griffith[5] record polyarthritis in an infant of 6 days whose mother was suffering from acute articular rheumatism. It is uncommon in early childhood, but after this period, especially in some of its varied forms, steadily increases in frequency. Langmead[6] analyzed the conditions existing in 2556 school children from 3½ to 14 years of age, and found definite rheumatism in 133. Smith[7] reports 4 cases with rheumatic manifestations, 1 at 4 years, 1 at 2 years and 2 months, 1 at 2 years, and 1 at 22 months. In the series of 185 patients studied by Ingerman and Wilson,[8] 173 were from 5 to 14 years of age; 10 from 3 to 5 years of age, and 2 below 3 years. Denzer[9] records 3 cases of rheumatic heart-disease under 2 years of age. The individual predisposition at this time is, however, not very great, Baginsky[10] observing articular rheumatism in but 1.4 per cent. of 10,375 children coming to the hospital, and Wachenheim[11] 113 instances in about 8000 children. Girls appear to be attacked rather oftener than boys; 71 per cent. of Ingerman and Wilson's patients being girls and 29 per cent. boys. In the fatal cases in children there about 3 females to 2 males (Poynton, Agassiz and Taylor).[12] Inheritance is a factor of importance, certain families seeming especially predisposed, either the parents or other children having suffered from the disease. Kephalliós[13] discovered evidences of inheritance in 50.7 per cent. of 69 cases. St. Lawrence[1] found that in 100 families in which rheumatic cardiac disease existed, there were 2 or more instances of this in 29 per cent., an incidence too high to be accidental. Faulkner and White[2] observed that in families in which there had been no rheumatic disease the incidence was less than ½ that which existed in families in which a case of rheumatism had previously occurred. Poynton, Patterson and Spence[3] found heredity present in 40 per cent. of 172 cases. The fact that the attacks of rheumatism in the different members of the family were often simultaneous, or at least closely associated in time of occurrence, seemed to argue against hereditary influence and more for that of environmental condition, such as cold and dampness, or even for direct infection; and yet the influence of heredity, although not proven, cannot be denied. Defective hygiene, especially exposure to cold and dampness or to sudden alterations of temperature, is perhaps the most potent predisposing factor. For this reason rheumatism is more prevalent in the cooler and damper season of the year; and this, too, explains the fact that some years and some countries produce many more cases of rheumatism than do others.

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[1] Arch. of Pediat., 1908, XXV, 265.
[2] Arch. of Pediat., 1899, XVI, 686.
[3] Med. Rec., 1896, L, 547.
[4] Researches on Rheumatism, 1914, 389.
[5] Amer. Jour. Dis. Child., 1926, XXXI, 250.
[6] Lancet, 1911, II, 1133.
[7] Arch. of Pediat., 1922, XXXIX, 799.
[8] Jour. Amer. Med. Assoc., 1924, LXXXII, 759.
[9] Jour. Amer. Med. Assoc., 1924, LXXXII, 1243.
[10] Berl. klin. Wochenschr., 1904, XLI, 1213.
[11] Arch. of Pediat., 1908, XXV, 669.
[12] The Practitioner, 1914, XCIII, 445.
[13] Wien. klin. Wochenschr., 1906, XIX, 563.

Exciting Cause.—The direct cause of the disease is unknown. Excessive acidity of the blood, dependent especially upon lactic acid, has been claimed to be influential, but there appears to be no proof of this. Microörganisms have been discovered in the blood, heart and joints by various observers, probably first by Popoff[4] in 1887. One of the most important contributions is that of Poynton and Paine,[5] who found cocci (streptococcus rheumaticus) in fatal cases of rheumatism, present in the joints, blood, heart and tonsils. They considered these the cause, and that they probably entered by way of the tonsils, since rheumatic individuals appear especially predisposed to tonsillar inflammation, and the characteristic rheumatic symptoms are in many cases immediately preceded by an acute angina. Later studies by them[6] confirmed their past experience, and Coombs, Miller and Kettle[7] and others produced arthritis and carditis in rabbits inoculated with streptococci obtained from rheumatic individuals. Whether or not any variety of microörganism can be considered the specific cause remains to be proven. In the great majority of cases of rheumatism blood-cultures have given negative results. Much of the experimental work is inconclusive. There should be mentioned in this connection the studies of Rosenow[8] who claims that the streptococcus is not only causative but selective in its action.

Certainly if infection is the exciting cause there must be some powerful constitutional susceptibility present as well. Family outbreaks, it is true, point decidedly to an infectious nature of the disease (Allaria)[9] yet but few such instances have been reported, and it is difficult to understand the association of infection with the sudden development of such nervous manifestations of rheumatism as seen, for instance, in many cases of chorea. The relationship of diseased tonsils and adenoids to rheumatism is still a question of dispute and it is hard to evaluate statistics in this matter. Poynton, Patterson and Spence[1] noted that of 172 cases of severe rheumatic disease in children 17 had had their tonsils and adenoids removed; 38 had enlarged and diseased tonsils, and in 22 cases acute tonsillitis had immediately preceded the attack. St. Lawrence[2] stated that after tonsillectomy there was no return of acute rheumatic fever in 84 per cent. of 42 children who had had this condition previously. His patients were followed for from 2 to 6 years. On the other hand, Ingerman and Wilson[3] found that in their cases, followed from 1 to 11 years, 76 per cent. of 88 children had rheumatic manifestations after as well as before tonsillectomy, while of 97 children whose tonsils were not removed 80 per cent. had rheumatic recurrences. Hunt[4] observed recurrences of rheumatic symptoms in 35 of 66 children after tonsillectomy and in only 33 of 78 children whose tonsils were not removed. It seems clear that although the removal of diseased tonsils and adenoid growths is perhaps indicated in rheumatic families as a precautionary measure, yet that there is no certainty that this will prevent the occurrence of rheumatic disease. It is still an open question whether tonsillitis in many instances is the source of the rheumatic disease or itself a rheumatic manifestation, as was formerly generally believed.

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[1] Jour. Amer. Med. Assoc., 1922, LXXIX, 2051.

[2] Jour. Amer. Med. Assoc., 1924, LXXXIII, 425.

[3] Lancet, 1920, II, 1086.

[4] Medit. Prebavlena K. Moskowa Sboneskie, 1887, 401. Ref., Dunn, Jour. Amer Med. Assoc., 1907, XLVIII, 494.

[5] Lancet, 1900, II, 861.

[6] Lancet, 1910, I, 1524.

[7] Lancet, 1912, II, 1209.

[8] Jour. Amer. Med. Assoc., 1913, LX, 1223.

[9] Revista crit. di med. clin., 1901, Nov. 23. Ref., Brit. Med. Jour., 1902, I. Current Lit., 5.

The etiology of the different varieties of chronic rheumatism is even yet little understood. There is some reason to believe that all the forms depend upon a chronic infection arising from the tonsils, sinuses, joints or the roots of the teeth. It has also been claimed that the disorder may be metabolic in origin (Pemberton);[5] and Fletcher[6] as well as Pemberton[7] asserts some connection with a diminution of the sugar-tolerance. Cecil, Barr and DuBois,[8] on the other hand, concluded that there is no relationship to metabolism. Rhonheimer[9] claims that juvenile arthritis is of neurogenous origin, and that it has no connection with acute articular rheumatism.

Pathological Anatomy.—There are no characteristic articular lesions in acute cases beyond hyperemia with turbidity of the fluid, and slight infiltration of the neighboring connective tissue. As a rule no bacteria are found, in the experience of most observers. Purulent inflammation occurs only as a complication. In the chronic cases there is decided effusion into the joint, with thickening of the capsule and ligaments, erosion of the cartilage, and finally involvement of the bones resulting in ankylosis. Fibrous nodules are sometimes present beneath the skin in various regions in children. These consist of connective tissue of an inflammatory nature, fibrin, and cells. The lesions of rheumatic endocarditis and pericarditis will be considered in the chapter upon Disease of the Heart. Here only may be mentioned the bodies described by Aschoff[10] in 1904 and known by his name. These are microscopic nodules attached to the adventitia of the blood-vessels of the myocardium, and are composed of peculiar large cells containing what look like numerous giant nuclei, together with leucocytes and connective tissue fibres. Aschoff considered them characteristic of rheumatism, and this has been confirmed by others, among them Thalhimer and Rothschild[1] who found them even in fatal cases of chorea without articular involvement.

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[1] *Loc. cit.*
[2] Jour. Amer. Med. Assoc., 1920, LXXV, 1035.
[3] *Loc. cit.*
[4] Guy's Hosp. Rep., 1923, LXXXIII, 383.
[5] Amer. Jour. Med. Sci., 1914, CXLVII, 423.
[6] Arch. Int. Med., 1922, XXX, 106.
[7] Amer. Jour. Med. Sci., 1921, CLXI, 517.
[8] Arch. Int. Med., 1922, XXIX, 583.
[9] Jahrb. f. Kinderheilk., 1917, LXXXV, 173.
[10] Brit. Med. Jour., 1906, II, 1103.

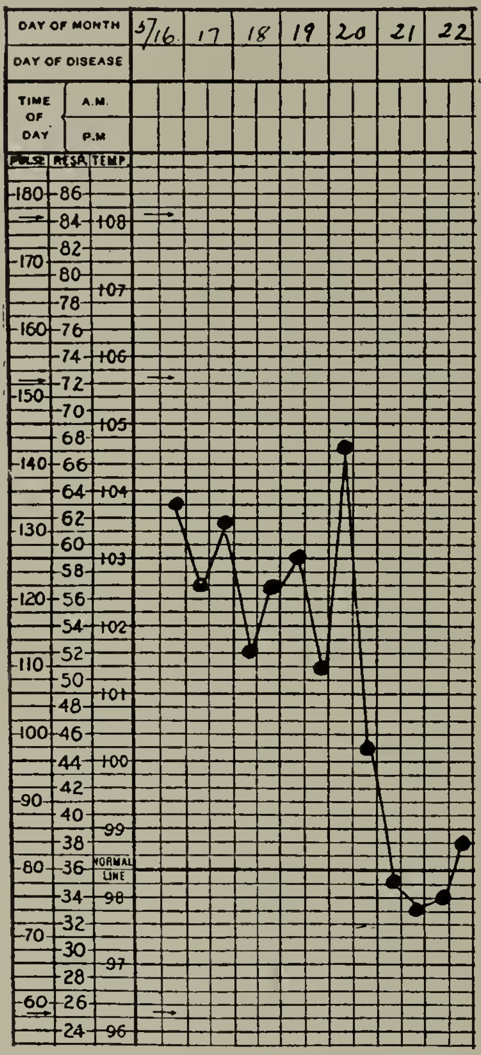

Fig. 229.—Acute Articular Rheumatism.

Alice W., aged 6 years. After becoming overheated by skating, developed headache, fever, and pain in the knees, ankles and feet. On entrance to the Children's Ward of the University Hospital, Philadelphia, May 16, exhibited pain, redness and swelling of both knees, tongue coated. No cardiac murmur present. Leucocytes 30,200; May 22, swelling and pain nearly gone. A faint systolic murmur audible.

Symptoms.—For the correct understanding of the manifestations of rheumatism in children, the idea must be banished that the affection must show itself in the same manner as in adults. The symptoms of rheumatism in early life are manifold. While an acute arthritis is the type, yet there may be no involvement of the joints at all, or this may occur secondarily. Particular notice must be taken of the vague, trifling pains in the joints or muscles, which are ignored by parents, but which can readily be followed by affections of the heart or by chorea. Cardiac involvement is especially common in children and is often the first manifestation. Tonsillar inflammation or torticollis may be quickly followed by arthritis, or, even without this, by pericarditis or endocarditis. Chorea may succeed articular inflammation or may precede it, or be followed by cardiac involvement without any arthritis. Subcutaneous fibrous nodules may be the first symptom or may occur subsequently to articular disease. Indeed, any one of these conditions may be the first to appear, but is liable soon to be followed by others. Although, therefore, articular rheumatism may be considered the type, the other conditions are rather to be viewed as forms of rheumatic manifestation than as complications.

Acute Articular Rheumatism.—Occasionally preceded by malaise, or sometimes by sore throat for several days, the attack generally begins acutely with fever, loss of appetite, and swelling, pain, and redness in one or several joints. The intensity of these symptoms varies greatly. In early childhood they are less marked than later, and at any time in early life the articular symptoms are usually not so decided as in adults, the redness, swelling and pain are less evident, sweating absent or slight, and the fever is less, reaching 103° to 104°F. (39.4° to 40°C.) at the onset but soon falling to about 101°F. (38.3°C.) (Fig. 229). Generally the joints of the lower extremities are first affected, the ankle or knee on one side being much oftenest attacked, with the hip usually next in order of frequency. The disease may remain limited to a single joint, but as a rule the corresponding joints of the other side are soon involved also, and perhaps the wrists, elbows and shoulders as well; or not infrequently the joints of the upper extremity may be attacked alone or primarily. Sometimes the disease appears in the cervical vertebræ, less often in the fingers and toes or other joints. Quite often one joint improves as another becomes affected, but it is not uncommon for the inflammation to reappear in the first joint as the course of the disease progresses. As a rule not many are involved.

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[1] Jour. Exper. Med., 1914, XIX, 417.

In the mild attacks, such as are observed especially in early life, the patient may not feel ill, the fever is insignificant, and the child is not confined to bed unless the lower extremities are affected. Often there is only sufficient pain to produce lameness without actual inability to walk. The discomfort is frequently so insignificant that it is given by the parents the common title of "growing pains."

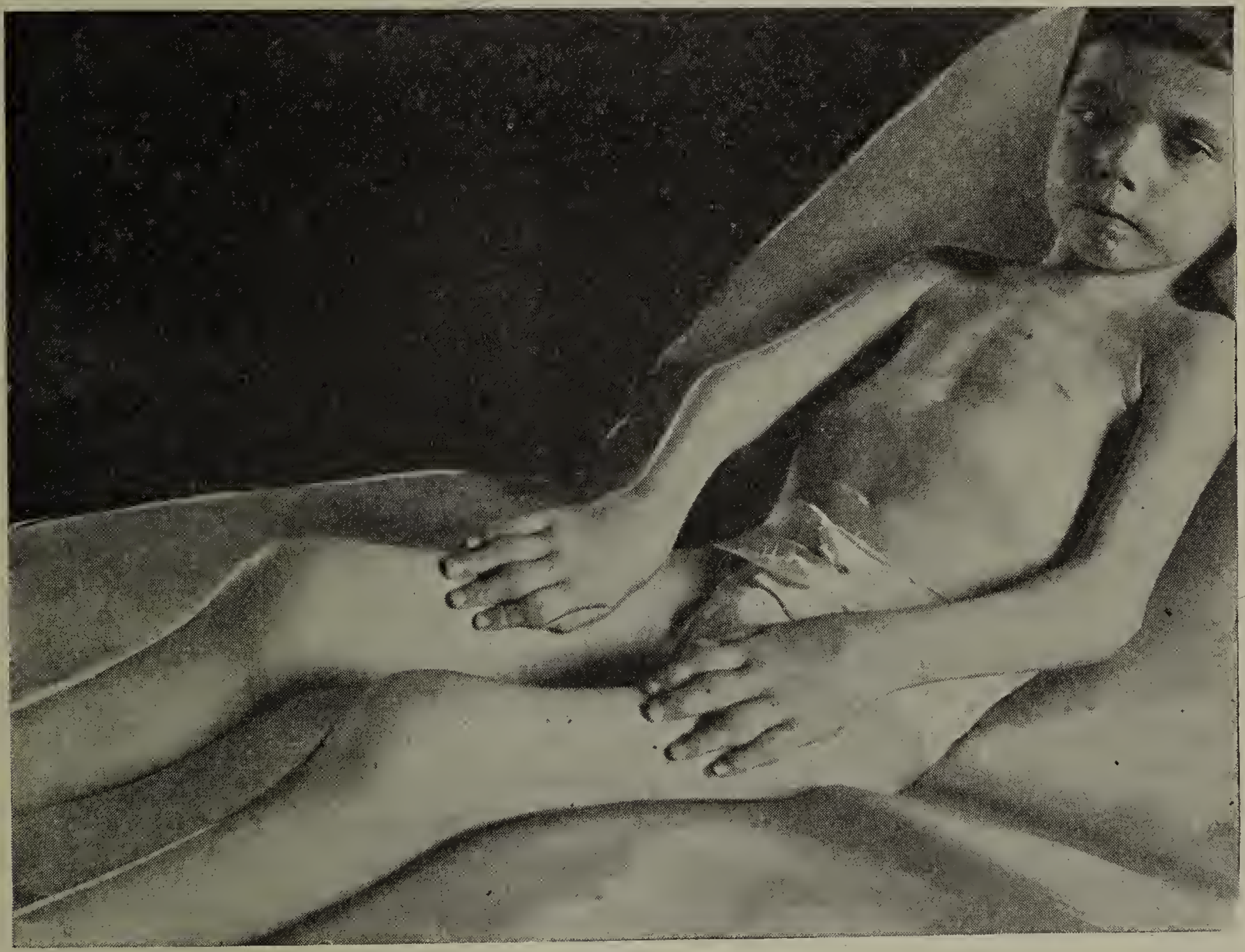

Fig. 230.—Chronic Articular Rheumatism.

Showing enlargement of the elbows, hands and knees. Same case as in Fig. 231.

The well-marked typical attacks of the adult type are not often seen until toward the end of later childhood. Here the expression of the face is one of pain and the tenderness of the inflamed joints is exquisite, any change of position causing severe suffering. The urine is high-colored and diminished in amount and usually acid, and there is abundant acid, sour-smelling perspiration. The blood in these cases exhibits a moderate leucocytosis, and anemia develops if the case is long-continued. There is coating of the tongue and loss of appetite. The temperature remains elevated, sometimes with exacerbations as fresh joints are attacked. Delirium and other cerebral disturbances are rare at any period of childhood.

The *duration* of acute articular rheumatism is variable. In average cases it is from 1 to 2 weeks, but the remarkable tendency to relapse may lengthen the course very greatly. Some cases pass into the chronic from, but this is much less common than in adults.

Chronic Articular Rheumatism.—Under this heading may be included disorders described as rheumatoid arthritis, arthritis deformans, and by other titles. The relationship of these to each other and to acute articular rheumatism is still far from determined. Although many of the cases are probably not at all truly rheumatic, yet provisionally they may all be denominated chronic rheumatism, inasmuch as it seems impossible to draw any sharp line of demarcation between them. Chronic rheumatism is, fortunately, comparatively uncommon in early life. Ibrahim,[1] however, reported upon 273 collected cases including certain of his own. Slightly more than ½ were less than 6 years of age. I have seen a very considerable number of instances. It may follow an acute attack of rheumatism without any cessation of symptoms, or may be the final result of a series of relapses or recrudescences. A second class of cases has certain points of difference, yet cannot be clearly differentiated clinically, although in nature probably not truly rheumatic. In this the course is subacute or chronic and more or less progressive from the beginning (*Rheumatoid arthritis*) (Fig. 230). Either form may begin in the larger joints usually attacked in acute articular rheumatism, but there is a tendency to primary localization in the hips, small joints of the fingers,

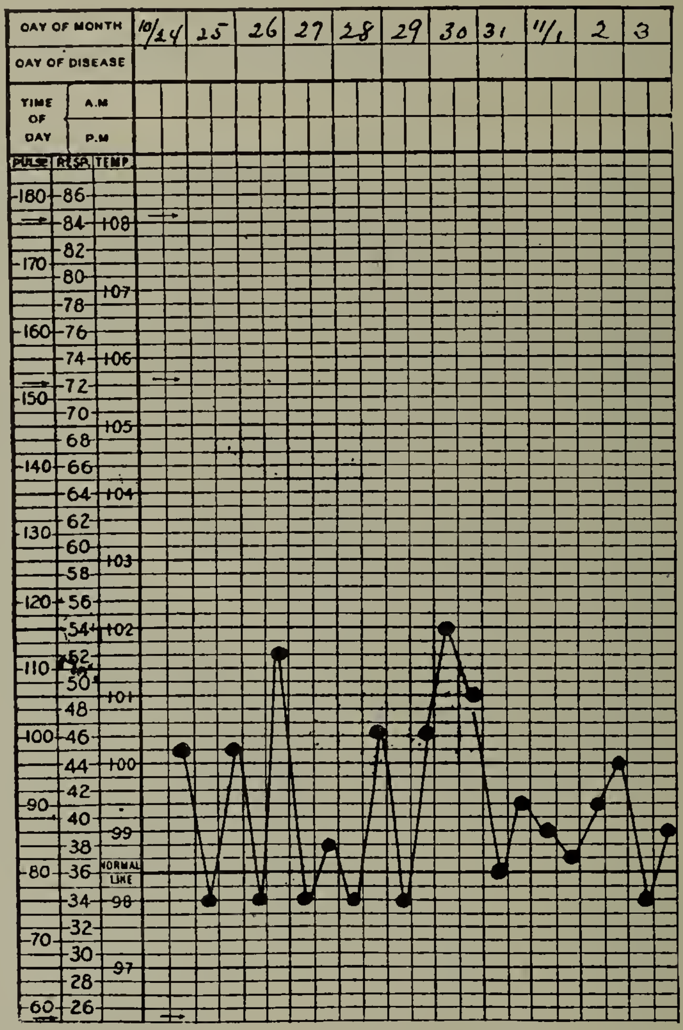

FIG. 231.—CHRONIC ARTICULAR RHEUMATISM.

Ignatio C., aged 7 years. At 20 months developed painful swelling of knees and fingers. In bed 3 or 4 months. Then entirely well until Dec., 1912, when gradually all joints of lower limbs, the hands and the elbows became swollen and painful, neck stiff. Been confined to bed ever since. Admitted to the Children's Hospital of Philadelphia, Oct. 25, 1913. Poorly nourished; very bad teeth; wrists, elbows, ankles, fingers and toes swollen but not tender; neck stiff; fixation at hips; knees swollen and tender; spleen not enlarged. Leucocytes 12,000. Pirquet-reaction negative. Teeth treated by dentist. Removed unimproved.

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[1] Zeitschr. f. orthop. Chirurg., 1914, XXXIV, 213.

jaws, or the neck. This is especially true of the second class of cases. The disease spreads from joint to joint without disappearing in those first affected, until the majority of the articulations are involved. There is increasing debility and anemia, and a tendency to cold, clammy sweating. Endocarditis may occur in either group, but less often in the second. Fever is usually moderate (Fig. 231) or absent. Periods of improvement occur but usually without complete disappearance of symptoms. At these times pain on movement may be moderate, but it becomes severe during the exacerbations. The joints are swollen and distorted and grate when moved; the skin over them shining and the neighboring muscles atrophied. There is involvement of the cartilages and bony structures and the periarticular tissues, and the final production of great deformity of the joints and of ankylosis. Arthritis deformans is considered by many as identical with cases of the class of rheumatoid arthritis; by others as a distinct affection. A special form of chronic arthritis has been described by Still[1] which is possibly rheumatic, possibly tuberculous, or dependent upon some chronic infection. It is characterized by progressive involvement of the joints, which become enlarged and stiff although not very painful, but which do not undergo destructive processes. There is more or less fever and always enlargement of the spleen and of the lymph-nodes.

Cardiac Rheumatism.—As already stated, this is sometimes the first manifestation of the disease, being evidenced by fever and vague general symptoms, the cause of which is unknown until a cardiac murmur becomes audible, and perhaps other characteristic symptoms developing later prove that the affection is of a rheumatic nature. Oftener, however, cardiac symptoms are secondary to chorea or arthritis. Involvement of the heart is especially liable to occur in childhood, and although more frequent during the severe attacks of articular rheumatism, it is common even in the mildest. In fact, the articular inflammation may have been so slight as to have been overlooked. Endocarditis is the most frequent lesion. A certain degree of this is present in probably the majority of cases of articular rheumatism, as shown by the acceleration of the pulse-rate, which is so often out of proportion to the elevation of temperature. Cohn and Swift,[2] as a result of electrocardiographic study, claimed that myocardial involvement was present in practically all cases of rheumatism. It appears usually about the end of the 1st week of the articular attack, or earlier. Coombs[3] found cardiac lesions in 60 per cent. of 75 children with rheumatic disease, Kephallinos[4] in about 70 per cent. of 129 cases and Ingerman and Wilson[5] in 69 per cent. of 185 cases. A valvular lesion, generally mitral, persists in probably ½ or more of the cases of articular rheumatism in children, developing in later attacks if not in the first. Pericarditis, although less common, is very characteristic of rheumatism in children, occurring in from 10 per cent. to 20 per cent. of the cases of articular rheumatism. It is seen much oftener than in adult life. Nearly always accompanied by endocarditis it has generally a sudden, acute onset, followed by effusion, and finally often by more or less obliteration of the pericardial sac. (See Diseases of the Heart, Vol. II, pp. 391, 398, 399.)

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[1] Med.-Chir. Transac., 1897, LXXX, 47.
[2] Jour. Exp. Med., 1924, XXXIX, 1.
[3] Bristol Med.-Chir. Jour., 1907, XXV, 193.
[4] *Loc. cit.*
[5] *Loc. cit.*

Chorea.—This is a frequent manifestation of rheumatism, although not all instances of chorea can be called rheumatic. In 226 cases of chorea reported by Still[1] at least 126 (55.7 per cent.) exhibited rheumatism, and in Ingerman and Wilson's[2] 185 children with rheumatic manifestations 34 per cent. had chorea. The question of the relationship of the two conditions is further discussed in the chapter upon Chorea. It may be the primary disorder and may be followed by arthritis, but oftener, if the combination exists, the latter is the first to develop. As the symptoms of chorea appear those of articular inflammation may suddenly cease. Very frequently it occurs without any articular involvement whatever, and is then often followed by endocarditis. All three conditions may be present at the same time.

Subcutaneous Fibrous Nodules (Fig. 232).—Although first described by Meynet[3] this affection has been most frequently recorded by English physicians. Barlow and Warner[4] in 1881 reported upon 27 cases in children. It has been much less frequently recorded in the United States, but Bronson and Carr[5] found the nodules in 45 per cent. of 38 children with rheumatism; Ingerman and Wilson[2] in 11 per cent. of 185 children, and Wallace[6] in 12 per cent. of 124 children. The condition appears to be more frequent in children than in adults. The nodules vary in size from that of a pinhead to that of a small nut, are not reddened or tender, and are found oftenest upon the back of the head, about the joints, especially the elbows, knees and ankles, along the tendons and the vertebræ and upon the pinna of the ear. They are distributed somewhat symmetrically, vary in number from very few to 50 or more and generally develop after the articular inflammation appears, although I have seen them abundant before this. They usually take some weeks to disappear, and may exceptionally last many months. They would seem to be especially likely to develop in cases with endocardial involvement.

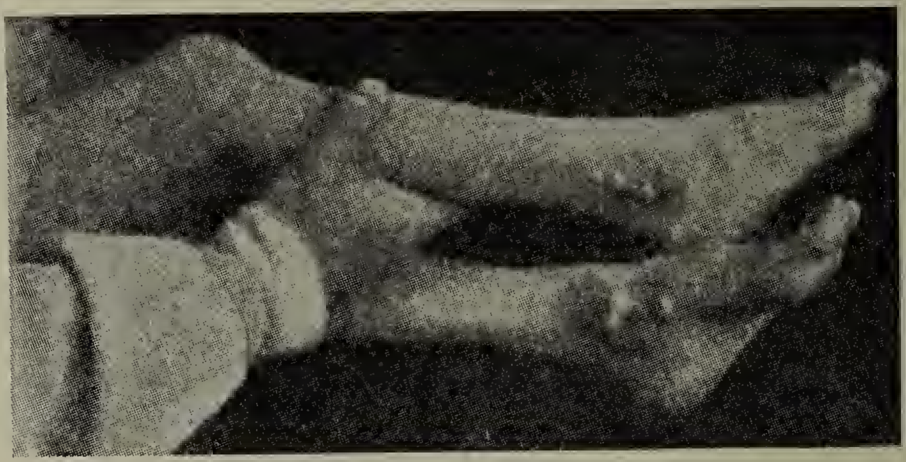

Fig. 232.—Subcutaneous Fibrous Nodules.

Boy of 4½ years. Chorea, chronic endocarditis, nodules about the wrists and ankles (*Berkowitz, Arch. f. Kinderheilk.*, 1912, *LIX*, 6.)

Cutaneous Manifestations.—Rheumatic subjects are prone to attacks of erythema of various forms, with which articular inflammation is often associated. Purpura too, is sometimes combined with rheumatic arthritis, with or without erythema. It is far from certain, however, that the majority of cases of arthritis with purpura have any connection with rheumatism. (See Purpura, Vol. II, p. 791.)

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[1] Practitioner, 1901, LXVI, 53.
[2] *Loc. cit.*
[3] Lyon méd., 1875, XX, 495.
[4] Transac. Internat. Med. Cong., 1881, IV, 116
[5] Amer. Jour. Med. Sci., 1923, CLXV, 781.
[6] Arch. Pediat., 1924, XLI, 731.

Tonsillitis.—As already stated, (p. 734) inflammation of the tonsils or pharynx may be promptly followed by articular inflammation or, even without this, by cardiac disease. There seems to be a special tendency to tonsillitis in evidently rheumatic subjects. It is a question whether the tonsillar inflammation is a manifestation of rheumatism, or merely the source from which the rheumatic involvement of the joints arises. Possibly it varies with the case.

Muscular Rheumatism. Rheumatic Myalgia.—The question whether this condition is actually rheumatic has been much disputed. There appears to be no doubt, however, that at least some of the cases of myalgia are instances of rheumatic disease. This is true, in children especially; of the acute torticollis which is characterized by sudden onset, great soreness and stiffness of the neck chiefly in the sternocleidomastoid muscles, rapid disappearance, and association with tonsillitis or other rheumatic manifestations. It is to be distinguished from rheumatism of the cervical vertebral articulations. I have seen arthritis and cardiac involvement quickly follow torticollis. (See Torticollis, Vol. II, p. 709.)

Another evidence of muscular rheumatism common in children are the so-called "growing pains" located in the extremities, to which little attention is paid as a rule, but which may be followed by cardiac involvement. Most frequently, however, these growing pains are probably located in the joints. Lumbago, pleurodynia, and other localized myalgias are less common in early life.

Complications.—A number of disorders may occasionally complicate rheumatism. Among these are vomiting, peritonitis, pleuritis, pneumonia, nephritis, bronchitis, iritis, trigeminal neuralgia, sciatica, venous thrombosis, epistaxis and mastitis.

Recurrence and Relapse.—One attack of rheumatism is extremely liable to be followed by later ones, especially in childhood. There is rather the reverse of a protective influence. The severity of the recurrences is in no way dependent upon that of the primary attack.

The tendency for the disease to relapse is very great. These relapses may take place in spite of the greatest care and entirely without discoverable cause; or they may be brought on by slight exposure, such as leaving the bed, a week or so after convalescence seems established. The disease may appear in the joints originally affected or in others. Repeated relapses may occur one after the other, the severity being independent of that of the first attack.

Prognosis.—The prognosis in children is better than in adults so far as recovery from the individual attack is concerned. The course of arthritis is shorter and the symptoms milder. Endocarditis, if slight, is frequently recovered from; if more severe, valvular disease remains but compensation is much more easily acquired in children than in later life. (See Endocarditis, Vol. II, p. 399.) The prognosis of rheumatism on the whole, however, is more serious in early life, on account of the greater tendency to recurrence of arthritis and chorea, and the greater probability that endocarditis will develop in later attacks, if not in the primary one; or that if already present it will become worse on each reappearance of rheumatic disease. This great disposition to the development of cardiac involvement in early life makes the prognosis of rheumatism in children always doubtful. Rheumatic pericarditis in children is exceedingly liable to lead to loss of cardiac compensation. The presence of subcutaneous fibrous nodules is unfavorable, since they are especially liable to accompany the development of an endocarditis or pericarditis (Cheadle).[1] In 21 children with rheumatism but without nodules there was only 1 death, whereas in 17 with nodules there were 6 deaths. (Bronson and Carr.)[2] The studies of Poynton, Agassiz and Taylor[3] upon 350 fatal cases of rheumatism indicated that the disease was at its worst from the 6th to the 12th year, and that the large majority of deaths occurred before the age of 20 years. The cause of death in early life was nearly always cardiac involvement.

The prognosis of chronic articular rheumatism is grave. Though the course of the disease is always slow, it is more rapid than in adults. Some of the milder cases may recover, but this is exceptional. Death finally occurs from exhaustion or some intercurrent disease, especially tuberculosis.

Diagnosis.—Diagnosis in early life is not always easy on account of the different guises under which the disease may appear. The discovery of a slight valvular lesion of the heart should always awaken the suspicion of rheumatism, and an investigation should be made into the previous existence of moderate pain in the joints, growing pains, torticollis, repeated sore throat, chorea, and other rheumatic manifestations. So, also, the development of chorea should lead to a careful study of the personal and the family history with regard to the earlier occurrence of rheumatism, and to a search for the presence of endocarditis or other rheumatic symptoms.

An attack of acute articular rheumatism is generally easily recognized, its characteristic symptoms being sudden development of some degree of pain, swelling, tenderness, and redness in one or, generally, more joints. A number of other conditions may, however, be confounded with it and must be eliminated. *Osteomyelitis* is among these. It is less often multiple in its localization, exhibits more severe constitutional symptoms, and involves the shafts and epiphyses rather than the joint itself. *Secondary arthritis* after acute infectious diseases, such as scarlatina, is usually monarticular, sometimes polyarticular, and is recognized by the previous history of the case. *Gonorrheal arthritis,* although generally monarticular, is more often multiple in early life than later. It is, however, nearly always combined with vulvovaginitis or ophthalmia. The *syphilitic arthritis* sometimes occurring as a later manifestation of syphilis is localized in both knee-joints, runs a very chronic course with little pain, and is generally associated with keratitis and other late syphilitic symptoms. *Septic arthritis* is polyarticular, purulent in character, and is always accompanied by other symptoms of sepsis. Acute rheumatism of the hip-joint may strongly suggest *appendicitis.* In infancy *scorbutus* and *syphilitic epiphysitis* are sometimes wrongly called rheumatism; but rheumatism is so rare at this period that it is proper to assume its absence unless its symptoms are unquestionable. Nearly all the cases of pain in the legs in infants, with disability and without fever, are due to scurvy. (See Scurvy, p. 709.) *Multiple neuritis* and *sciatica* may at first suggest rheumatism but exhibit no articular involvement; and the fever and pain on being moved often seen in *poliomyelitis* may cause confusion, but are distinguished by the absence of articular effusion. *Tuberculous coxitis* may also at times occasion difficulty. Careful examination will soon reveal the true condition. *Retropharyngeal abscess* may in some instances simulate torticollis. The course of the case soon renders the diagnosis clear.

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[1] Keating's Cyclop. of Dis. of Child., 1889, I, 802

[2] *Loc cit.*

[3] *Loc cit.*

In the case of chronic rheumatism, the principal diagnostic problem is to determine the cause; whether the disease is a direct sequence of an acute rheumatic attack, is the result of some infectious process such as syphilis, tuberculosis, scarlet fever, sepsis or the like, or has been chronic from the beginning and apparently of a primary nature.

Treatment. Prophylaxis.—This is by all odds the most important, since little can be done to influence a cardiac rheumatism when present, and it is the cardiac disease upon which the gravity of the prognosis depends in most cases. Any possible foci of infection such as diseased tonsils, teeth, sinuses and the like should be treated. It should be emphasized that the removal of tonsils is to be done only when they are diseased, and even then prevention of rheumatic manifestations is by no means certain (see Vol. II, p. 44). Children with a decided family history of rheumatism or who have suffered from previous attacks should be scrupulously guarded against exposure to cold and damp, including wetting of the feet; dressed warmly in woolen underclothing, yet not so warmly that free perspiration is produced; enjoy an abundance of fresh air on suitable days, and live in dry, healthful dwellings. At the same time care must be taken against rendering the child susceptible by too great precaution. The effort should be made to improve the general tone and to increase the resisting power to influences which might prove harmful. A system of hardening should be cautiously instituted, through cool, morning sponging followed by vigorous rubbing, systematic gymnastic exercises and massage, the fullest use of these measures being approached by degrees. In specially susceptible subjects change of residence in winter to a warm, dry climate is most advisable.

Whether diet has any influence in preventing the disease is disputed. The giving of nitrogenous food and the restriction of carbohydrates has been recommended, but the actual value of these is questionable.

For the prevention of cardiac involvement the cases of even slight articular rheumatism should be kept quiet in a warm room, and, if there is the slightest fever, in bed. This is all that can be done.

Treatment of the Attack.—In cases of acute articular rheumatism the diet during the acute febrile stage should consist of milk or broths. Later a simple, digestible regimen may be prescribed. The patient should be at rest in bed in an equably heated but well-ventilated room, and the slightest chilling should be avoided since a relapse of the disease may readily follow. The clothing should consequently be preferably of flannel, made easily removable to facilitate changing when wet by the sweating. For local treatment the affected joints may be wrapped in cotton or with warm, moist, applications for the relief of pain; all motion avoided; and the bed-clothes prevented from pressure on tender regions. The treatment of developing endocarditis will be considered in the section upon Endocarditis, Vol. II, p. 405. For the direct medicinal treatment of the attack salicylic acid has been considered a specific. Whether or not it may be justly deemed so is a matter of much dispute, and no definite proof of its specificity has ever been brought forward. There is no doubt at least that the salicylates control both pain and fever. To be of value, sufficiently large doses should be given, 5 to 8 grains (0.3 to 0.52) of the salicylate of soda being administered every 3 hours to a child 8 to 10 years of age. Very much larger initial doses are recommended by some clinicians. The amount mentioned can be rapidly increased if necessary, and if the stomach tolerates it, until moderate tinnitus and deafness are produced. The addition of an aromatic, such as ginger, often aids in the tolerance of the drug. Salicin, oil of wintergreen, acetyl-salicylic acid, phenol salicylate, and other salicylic acid derivatives, may be given in place of the salicylate; or, if the digestion will not bear any internal administration, the oil of wintergreen (salicylate of methyl) may be used freely by inunction in the armpits and similar suitable regions. The constitutional effect can be produced in this way. Salicylates may also be given by rectum. When the pain and fever are decidedly lessened the dosage may be reduced, but the treatment should not be abandoned entirely for some time after convalescence seems established. Where the salicylates fail to relieve pain, antipyrine or phenacetin may be employed, alone or in combination with these. Some of the newer preparations such as neocinchophen, novatophan, tolysin, etc. may be tried. It is claimed that they are as effective as salicylates but less irritating to the stomach and less toxic. For the pain in severe cases opiates may be necessary. I have never witnessed any of the depressing effects from salicylates which have sometimes been reported, yet there is no doubt that they can occur and the drug must be given with due caution and with this possibility in mind. The alkaline treatment, as with bicarbonate of soda or citrate of potash, has been much esteemed. It may be combined with the salicylates and should be given in sufficient dose to render the urine alkaline.

Experiments with serum and vaccine treatment have been made, but the results, which have been reported as encouraging, demand further corroborative experience.

During convalescence from acute articular rheumatism tonic remedies may be required, such as arsenic, iron, or cod-liver oil. The treatment of acute manifestations of other forms of rheumatism will be considered in the different chapters discussing these subjects.

In the treatment of subacute and chronic rheumatism the effort must be made to discover the cause. In this direction the condition of the tonsils, nasal sinuses, mouth and the like should be carefully examined and appropriate measures taken if needed. Autogenous vaccines have been tried with some reputed success. The benefit, however, may be not through any specific effect, but as a result of a parenteral protein treatment, since good results have followed the subcutaneous injection of various non-specific proteins. DeCourcy[1] found no benefit from the injection of milk although many reports in foreign literature are favorable. Apart from these methods the persistent administration of iodide of potassium has much in its favor. In other cases arsenic or cod-liver oil is of benefit. Thyroid extract has occasionally given good results. Always the effort to improve the general health must be made and to procure the most favorable hygienic conditions. In Pemberton's[2] studies limitation of the diet has been useful. Massage and passive movement of the joints are very important, except during exacerbations, when rest is required. Baking the joints is very helpful in some cases. Local applications of ichthyol ointment or preparations of iodine or mercury may be employed. Benefit has followed the production of passive congestion by the Bier method, as well as by baking in a suitable apparatus. Treatment at some one of the thermal springs is of decided help and should always be employed when possible.

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[1] Ohio State Med. Assoc. Jour., 1923, XIX, 416.

[2] *Loc. cit.*

CHAPTER VI

THE DIATHESES

The views of many years ago concerning the existence of various diatheses passed to a large extent into the class of forgotten things, under the increasing trend of the attributing of nearly every disordered state to the influence of some infection. In recent years it has become evident that neither infection nor any other active cause is sufficient by itself to account for the development of certain disorders in certain persons; and that diatheses do, in fact, exist. By this term is meant a constitutional tendency to the development of certain sorts of diseases varying with the individual; a tendency which makes the same acting cause vary in the character of the symptoms produced. It is to be noted, however, that in the strict sense a diathesis is not a disease, but a constitutional peculiarity which acts as a predisposition.

Various diatheses have been described, the boundary-lines between then being not sharply marked, and the list of symptoms attributed to their influence varying with different writers. Among them may be mentioned the spasmophilic, lymphatic, neuropathic and exudative. Additional varieties have sometimes been described, as the chronic constitutional, the metabolic, the hemorrhagic, and the endocrine. It is further to be observed that there is frequently a combination of two diatheses, the evidences of both appearing in the same individual. The symptoms of the lymphatic diathesis are to a large extent by many attributed to the exudative diathesis, and it is certain that there exists in any event a close relationship between them. Whether they should, in fact, be classed as one is not yet possible of determination. There is also witnessed the presence of allergic manifestations in cases assigned to the exudative diathesis. Spasmophilia is by many considered as one form of the neuropathic diathesis, and its occurrence in combination with the exudative diathesis is very common. It is questionable, however, whether it can properly be ranked among the diatheses. In other cases lymphatic disturbances occurring in nervous subjects give rise to the title "neurolymphatic diathesis." All this renders the subject confusing and divisions arbitrary and conflicting.

Spasmophilia and the neuropathic diathesis are described elsewhere (see Vol. II, pp. 526 and 551) in connection with Diseases of the Nervous System. Here will be considered only the exudative and the lymphatic diatheses

EXUDATIVE DIATHESIS

Etiology.—This title was applied by Czerny[1] to subjects in which, with other symptoms, there is a tendency to *exudation*, or inflammation, of the skin and mucous membrane. It covers many of the symptoms formerly described as "scrofulous," but is in no way connected etiologically with tuberculosis. It occurs chiefly in the 1st year of life, but to a lesser extent also after this period. It is to a considerable degree hereditary and familial, several children of the family exhibiting the same symptoms, and the parents perhaps having suffered from eczema, gout, asthma, or some nervous disorder. Apart from the constitutional tendency, the symptoms are brought on or increased by an improper diet, such as one containing an excess of food of any sort, particularly one too largely of milk, and, most of all, of fat. It is possible that in some cases the protein may be especially at fault. Recent studies have demonstrated that not all cases of eczema can be attributed to the exudative diathesis, as commonly understood. The influence of certain proteins, varying with the case, seems to be beyond doubt in the production of eczema, and the same is true of urticaria and asthma. The question arises whether the allergic phenomena shown in some instances by the development of eczema, asthma or urticaria, indicates a difference in the etiology of these disorders in different subjects, or whether there is some direct association between the exudative diathesis and allergy. (See Allergy p. 750.)

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[1] Jahrb. f. Kinderheilk, 1905, LXI, 199.

Symptoms.—The disease manifests itself in infancy by an unusual disposition to the development of seborrhea of the scalp and face, and later of eczema; the latter often extending over much of the body. Catarrhal inflammation of the nose and pharynx, as well as bronchitis, is common, and otitis a frequent consequence. Some enlargement of the neighboring lymphatic glands may be found, but decided and extensive hypertrophy is not a characteristic of this condition. The infants are usually much too fat, but flabby and anemic, and readily suffer elevations of temperature. In other cases they are thin even before symptoms appear, or become debilitated and lose flesh through the constant irritation and loss of sleep brought about by the itching of an eczema, or without this. The blood shows an increase of the eosinophilic cells, perhaps up to 10 or 20 per cent. Whether this depends upon eczema, if this is present, or occurs coincidentally and from the same cause, is uncertain. The fact that it is seen also in asthma is suggestive of the latter relationship. It has been claimed that the sugar-content of the blood and the retention of chlorides is increased, but this demands further proof. Similarly the fat-content of the blood is said to be diminished (Schippers).[1]

In older children, after the period of infancy, eczema may be replaced by asthma, recurrent bronchitis, obstinate cough, pruritus, lichen, and urticaria. The geographical tongue is a common symptom. Vasomotor disturbances are frequent, such as palpitation, rises of temperature, and the like. It is uncertain whether the phlyctenules which appear on the conjunctiva in children are symptoms of the exudative diathesis, or are always actual tuberculous lesions.

In the category of the exudative diathesis perhaps belong, too, many of the cases of *arthritism* in children, described by Comby[2] and others. The symptoms are variable and multiform. Prominent among them are those just detailed, in addition to attacks of vomiting (recurrent vomiting), arthralgia, headache, neuralgia, vertigo and various other nervous and vasomotor disturbances.

Course and Prognosis.—The tendency to eczema diminishes greatly after the 1st year, and in general the exudative manifestations are prone to ameliorate and soon to cease. Sometimes, however, the symptoms characteristic of the condition after this period are very slow in disappearing. Generally they are gone by the time puberty is reached. They do not often in themselves constitute an element of danger. Fatal results generally depend upon an attendant spasmophilic or lymphatic condition.

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[1] Nederl. Tijdschr. v. Geneesk., 1920, II, 1081

[2] Arch. de. méd. des enf., 1902, V, 1; 65.

Diagnosis.—This rests upon the symptoms as already outlined. Scrofulo-tuberculosis may, it is true, develop in children with the exudative diathesis, but the eczema and catarrhal condition of the latter has nothing in common with the glandular inflammation, chronic conjunctivitis and keratitis, and severe chronic catarrhal processes of the former; although it is possible that it is the existence of the exudative condition which predisposes to a tuberculous infection producing the symptoms often called scrofula. Similarly the status lymphaticus is characterized by decided hypertrophy of the lymphatic tissues throughout the body, including the thymus gland, and by a tendency to sudden death; but it is not a producer of eczema or catarrhal processes. It is, however, frequently combined with the exudative diathesis.

Treatment.—Only the passing of time will cure the constitutional predisposition, and treatment must be directed against the immediate exciting cause of the symptoms. This is chiefly dietetic. The fat in the food must be reduced, and, indeed, the total amount of food given usually diminished as well, and care taken to prevent the children from gaining weight rapidly. Starchy addition to the diet should be commenced at once on the appearance of symptoms, and this made to replace milk to a considerable extent.

LYMPHATIC DIATHESIS

(Lymphatism. Status Lymphaticus)

This is a disorder much discussed and not yet entirely understood. It would appear to be a constitutional condition in which there is present a diminished resistance of the entire organism to morbid influences, and a certain hypersensitiveness of the nervous system producing a predisposition to sudden death from cardiac failure brought on by slight and in themselves insufficient causes, or occurring entirely unexpectedly and apparently without reason. Anatomically there is a tendency to general hyperplasia of the lymphoid tissues throughout the body, including the thymus gland. It must be borne in mind, however, that in normal children the glandular apparatus is well-developed, and this must not be confounded with hyperplasia. Even in the new-born cervical, axillary and other glands are discoverable with careful palpation (see p. 57). The close association of lymphatism with sudden death and thymic enlargement was emphasized by Paltauf;[1] while Escherich[2] maintained that the condition was a toxemia, the origin of which was the hypertrophied thymus gland. The relative relationship of thymic hypertrophy and of general lymphatic enlargement is not yet clear. It is possible that lymphatic overgrowth is the direct cause of the symptoms, and that the enlargement of the thymus gland is a concomitant accidental or compensatory process. Whether, however, a lymphoid hyperplasia is a necessary condition, or even one always present has been disputed; and the theory has been advanced by Heubner[3] and others, and has much in its favor, that the disorder is a constitutional anomaly consequent upon some chemical alteration of the tissues, independent of lymphatic or thymic enlargement, although these are likely to appear among the later symptoms. Personal experience has led me to the belief that the enlarge-

[1] Wien. klin. Wochenschr., 1889, II, 877; 1890, III, 172.

[2] Berlin. klin. Wochenschr., 1896, XXXIII, 645.

[3] Lehrb. d. Kinderheilk., 1911, II, 33.

ment of the thymus, and perhaps too of the lymphatic glands, is a secondary matter, and can be entirely absent in cases of sudden death, and present in those dying of other causes. The extensive studies of Hammar[1] confirm this view. (See also Sudden Death, p. 243, and Enlargement of the Thymus, Vol. II, p. 837.)

There are cases with glandular enlargement and symptoms of other sorts to which the title "scrofulous" was formerly, and is still often, applied, the lesions being in reality tuberculous manifestations, although the constitutional condition would place the child in the class of those suffering from the lymphatic or oftener from the exudative diathesis (p. 745. See also Tuberculosis, p. 648 and Adenitis, Vol. II, p. 815.) There is an undoubted, close relation between lymphatism and spasmophilia (see Vol. II, p. 527) on the one hand, and the exudative diathesis on the other.

Etiology.—A distinct family disposition is seen in many instances. A tendency to glandular swelling may have existed in the parents as well as the children; and as regards the most severe form, I[2] have previously reported the occurrence of 9 sudden deaths from lymphatism in one family of children. The disease is observed especially in infancy and early childhood, although in the disposition to sudden death from slight causes it has not infrequently been witnessed in adult life. Even the new-born and very young infants may exhibit it; and many instances have been wrongly attributed to death from suffocation through overlying or brought about in other ways. Sex, race and season exert no influence. Rickets is often associated with the lymphatic diathesis but has no etiological connection, other than that both may possibly be produced by allied causes acting simultaneously and consequently combined; and the same is true of spasmophilia and of the exudative diathesis. The constitutional tendency may be present at birth, or may be acquired especially through errors in hygiene and diet; or may disappear under regulation of these or perhaps as a result of increasing age.

Pathological Anatomy.—The noteworthy feature often found at autopsy is the hyperplasia of lymphoid tissue. This is seen particularly in the thymus gland, which is frequently abnormally large. In determining the existence of enlargement the great variation in the size of the normal thymus gland in different children is to be taken into account, as well as the age of the patient. (See Physiology, p. 58; Diseases of the Thymus Gland, Vol. II, p. 836.) In addition to this lesion there is commonly more or less hypertrophy of the lymphoid tissue throughout the body, particularly well seen in the glands of the neck and head, and in the tracheo-bronchial and mesenteric glands, as also in the nasopharynx, the posterior pharyngeal wall, the base of the tongue, the tonsils, the entrance to the larynx, Peyer's patches, and the solitary follicles of the intestine. There is usually moderate enlargement of the spleen, with prominence of the Malpighian bodies. The liver is often fatty and the cardiac muscle may show degeneration. Hypertrophy of the heart has been observed by Riesenfeld[3] and by Rieder.[4]

Symptoms.—These are often vague or even unnoticed; often sufficiently well-marked, but varied. The frequent association of the exudative diathesis accounts for the tendency to seborrhea and eczema

[1] Zeitschr. f. Kinderheilk., 1916–17, XV, 225.
Upsala Läkaref. Förhandl., 1922, n.f. XXVII, 147.

[2] New York Med. Jour., 1909, XC, 444.

[3] Jahrb. f. Kinderheilk., 1917, LXXXVI, 419

[4] Jahrb. f. Kinderheilk., 1922, XCVII, 9.

so often seen. Lymphoid hypertrophy is generally discovered in some part of the body. It is often and sometimes early shown in the development of adenoid growths of the nasopharynx, or increase of lymphoid tissue at the base of the tongue or on the posterior pharyngeal wall. In other cases there may be discovered persistent and sometimes decided enlargement of the lymphatic glands in the region of the neck, occiput, axillæ, or groins. The spleen is often found enlarged on palpation, and it may be possible to detect by percussion or by radiography, or occasionally by palpation, an enlargement of the thymus gland. Generally, however, thymic enlargement cannot be determined with certainty during life, owing to the possibility of the percussion-dulness or the *x*-ray shadow being produced in other ways. (See Diseases of the Thymus Gland, Vol. II, p. 837.) Other cases show a tendency to asthmatic respiration or to attacks of asphyxia dependent sometimes upon pressure of an enlarged thymus gland; much oftener upon the nervous disturbance. Ordinary asthmatic bronchitis is a symptom rather of the exudative diathesis. The combination of the neuropathic diathesis or of spasmophilia frequently observed accounts for the occurrence of convulsions, dyspnea, fever from insignificant causes, and larynogospasm; but it is not certain how often this is the case and how many instances, especially of the last mentioned, depend purely upon lymphatism. The children are usually phlegmatic, flabby, fat, inactive, pale, and of little strength. There is a very notably diminished resisting power and increased susceptibility to disease, and infections of any sort may produce an unusually well-marked reaction.

Of all symptoms, however, the most important is the danger of sudden death. This often appears to be respiratory from asphyxia and cyanosis, but probably in most cases is cardiac, as was pointed out by Pott,[1] the accident occurring without discoverable reason; or following very insignificant trauma, such as a hypodermic injection, an exploratory puncture, the giving of diphtheria-antitoxin, and the like; or being produced by the shock from cold water, the use of a tongue-depressor, the administration of an anæsthetic, or the occurrence of some mild acute disease; or often without there having been any previous symptoms whatever. In any event the patient suffers from a sudden, apparently suffocative attack, throws the head back, turns pale, or perhaps bluish, and sometimes dies in an instant; or the child may possibly be found dead in bed without any symptoms having been observed. Sometimes the death is not entirely without warning, and attacks which appear to be laryngospasm, or symptoms suggesting asphyxia from other causes, may be present for several hours. In still other instances there may have been a series of short attacks of the nature described occurring during some weeks or longer, and the patient at last dies in one of these. I have known such short attacks to be of very great frequency. Thus in one case the mother, a foreigner with little command of English, said of her child that it "died every day." Probably the majority of cases of lymphatism of this severest form are not recognized until a sudden fatal attack occurs.

Prognosis.—For the milder cases the prognosis is on the whole good, if dangerous symptoms can be avodied. Under proper management the evidences of the disease may be kept in abeyance, and even the pathological tendency made to disappear as the patient grows older, and particularly as puberty is approached. On the other hand, the prognosis is always doubtful in individual cases, because of the possibility of the occurrence of sudden death.

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[1] Jahrb. f. Kinderheilk., 1892, XXXIV, 118.

Diagnosis.—This can be made if, with the symptoms described, the distinct evidences of lymphatic hyperplasia are discoverable. An *x*-ray examination may show hypertrophy of the thymus gland. Discovery by the same means of enlarged tracheo-bronchial lymph-nodes does not determine whether these are dependent upon tuberculosis or upon the lymphatic diathesis. Similarly the hypertrophy of the tonsillar tissue of the fauces and nasopharynx is not by itself sufficient to warrant a diagnosis of lymphatism, although it is a characteristic of this condition. Often, in fact, lymphatism is entirely unsuspected until sudden death occurs and autopsy shows general hypertrophy of the lymphatic tissue of the body.

Treatment.—The only treatment possible is the careful regulation of the diet and hygiene, inasmuch as it is very probable that these, and particularly the diet, have a decided etiological influence. There is no rule, however, which indicates what the alteration in the diet should be, and possible faults should be sought for in each individual case. Gymnastic exercises and massage may improve the general health. Suitable tonics may be employed to increase the patient's strength and remove anemia. Whenever the lymphatic diathesis is suspected, great care must be taken to avoid the causes which bring about a sudden fatal termination. The use of an anæsthetic must be refrained from if it can be avoided, and, indeed, no operation performed which can in any way be omitted. Cool bathing may be dangerous, as may any sudden mental or other physical shock. Even the giving of antitoxin is to be regarded with anxiety, and the objections to it must be carefully weighed before a decision is reached.

In recent years there has been an increasing number of claims for benefit from the employment in young infants of *x*-ray treatment for symptoms supposedly connected with the thymus gland. When these symptoms have clearly pointed to pressure by the gland, such treatment is certainly indicated. In all other cases, however, there seems to be no logical reason for its use, since the enlargement is probably the result and not the cause of lymphatism. It would seem doubtful in such cases whether apparent improvement can be properly associated with the shrinking of the thymus gland.

CHAPTER VII

ALLERGY

In this connection may well be discussed the occurrence of peculiar symptoms, the result of reactions against protein substances. In speaking of these conditions it is better to use the broad terms *hypersensitiveness* or *allergy*, indicating an altered power of the organism to react (Anderson),[1] rather than *anaphylaxis*, which in its true significance is an induced experimental hypersensitiveness due to specific antibodies (Coca).[2] It may be, however, that these two conditions are identical. The term *idiosyncrasy* might be reserved for reactions against non-protein substances, such as drugs, smoke, light, dust and bacteria, although the last two may perhaps act as protein. It has been shown by Schloss[1] and Talbot[2] that it is the protein which is the active cause of the disturbances in allergy..

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[1] New York Med. Jour., 1923, CXVII, 358

[2] Jour. Immunol., 1920, V, 363.

What follows is only an outline of this interesting subject, particularly as it bears upon early life. Some further consideration will be given in the sections dealing with Asthma (Vol. II, p. 287), Diphtheria (Vol. I, p. 522), Eczema (Vol. II, p. 883) and Urticaria (Vol. II, p. 866); and some reference has already been made to it earlier (see p. 133). Excellent articles and reviews have been published by Schloss,[3] Blackfan,[4] Coca,[5] Wells,[6] Cunningham,[7] Duke,[8] and others, from which we have quoted freely.

Etiology.—Allergy may occur in the human subject from the ingestion of a great variety of proteins contained in animal and vegetable foodstuffs, especially eggs, milk and beef. Although it is food which is of most importance in producing allergic symptoms in infancy and childhood, hypersensitiveness may be excited also by protein substances which have entered the economy in some other way, such as by inhalation of plant-pollens or other products of vegetation, or of emanations from animals; by contact of the intact or broken skin or mucous membrane with plants or animal hairs; and by injection under the skin or into the circulation of blood or serum from other animals. It is possible that some of the symptoms produced by certain animal parasites are of allergic nature.

The knowledge of the mechanism by which the manifestations of allergy are brought about is still in the theoretical stage. The true explanation probably lies in the field of colloidal chemistry (Stitt).[9] Allergy in man may be hereditary or acquired. In the former there is a transmitted tendency to allergic manifestations of some sort, but not necessarily to any special proteins to which the parents may react. (Longcope;[10] Cooke and Vander Veer;[11] Cunningham[12]). In the acquired form there are no symptoms on the first ingestion of or contact with the protein, but the sensitiveness develops only on later exposure (Schloss).[13] In the effort to explain the reaction in infants who have had only human milk, Shannon[14] submits experimental evidence of the transmission of food-proteins from the mother through the breast-milk. Such transmission has been denied by Stuart.[15] The work of Kuttner and Ratner[16] has shown that the human placenta is permeable to diphtheria-antitoxin, and it is possible that other protein-substances may be transmitted in this way and produce sensitization. In order that cow's milk

[1] Amer. Jour. Dis. Child., 1912, III, 341.

[2] Bost. Med. and Surg. Jour., 1918, CLXXIX, 285

[3] Amer. Jour. Dis. Child., 1920, XIX, 433.

[4] Amer. Jour. Med. Sci., 1920, CLX, 341.

[5] *Loc. cit.*

[6] Physiol. Rev., 1921, I, 44.

[7] Amer. Jour. Dis. Child., 1922, XXIV, 247.

[8] Amer. Jour. Med. Sci., 1923, CLXVI, 645.

[9] Practical Bacteriology, etc., 1923, 282.

[10] Amer. Jour. Med. Sc., 1916, CLII, 625.

[11] Jour. Immunol., 1916, I, 201.

[12] *Loc cit.*

[13] *Loc. cit.*

[14] Amer. Jour. Dis. Child., 1921, XXII, 223.

[15] Amer. Jour. Dis. Child., 1923, XXV, 135.

[16] Amer. Jour. Dis. Child., 1923, XXV, 413.

or other food-protein may cause a specific reaction in the organism, it must be absorbed unaltered or only partly changed. Unaltered protein may gain entrance to the circulation through the intestinal mucosa in athrepsia and in diarrheal disturbances (Anderson and Schloss),[1] and it has even been claimed that the normal intestinal tract of young infants is permeable to foreign protein (Grulee and Bonar).[2]

The normal frequency in children of positive response to cutaneous tests with protein, yet without clinical evidence of allergy, is from 10 to 15 per cent. (Baker;[3] Peshkin and Rost[4]). In subjects with eczema and asthma positive cutaneous response to one or more proteins is found in from 35 to 70 per cent., about ½ of these children showing clinical improvement when the offending proteins are eliminated from the diet.

Symptoms.—There are several manifestations of allergy. Particularly in this place should be noted *hypersensitiveness to cow's milk.* This condition is uncommon, but when it exists the ingestion of even a small quantity of the milk may result in immediate projectile vomiting, swelling of the mucous membranes with sneezing and watering of the eyes, erythema or urticaria, and in some instances dyspnea and shock. Not all sensitive infants will react so acutely. A cutaneous test with cow's milk will usually, but not always, be positive in these cases. Similar symptoms appear with hypersensitiveness to other food-proteins, and it is even possible that certain more or less chronic gastro-intestinal symptoms, especially if associated with urticarial rashes, may be allergic manifestations. *Serum-sickness* may occur in two forms. The first of these, which fortunately is rare, closely resembles the true anaphylaxis of the type which may be produced in an experimental animal. Within from a few minutes up to a few hours after the injection of a foreign protein, such as diphtheria-antitoxin, great respiratory difficulty develops with asthmatic breathing and laryngeal stridor; there is cardiac embarrassment with dilatation of the right heart; cyanosis is usually present; vomiting often occurs; and convulsions may be a terminal symptom. Death may ensue in a few moments or after an hour or two. Serious reactions, if they occur, usually do so at the first and not at later injections, and it is very rare for a fatal termination to take place except after the first injection. Dean,[5] however, reports it following the fourth subcutaneous injection of tetanus antitoxin. The more usual type of serum-reaction gives symptoms only after an interval of from 5 to 12 or more days following the injection, although this interval may be shortened in some cases to 24 hours after the second or subsequent injections. The common manifestations are an erythematous or urticarial eruption, edema, malaise, fever, articular pain and swelling, sneezing, coryza, and enlargement of the superficial lymph-nodes. Not all these necessarily occur, urticaria and erythema being the most frequent.

Allergy to pollens and other vegetable-matter, animal hairs and emanations, and bacteria usually results in hay-fever and asthma, and will be discussed later (Vol. II, pp. 252, 287). In some individuals the only manifestation of allergy is urticaria or angioneurotic edema. It has been claimed that a number of other conditions may at times be dependent upon allergy. Among these should be mentioned erythema muliforme, cyclic vomiting (Schloss,[1] Le Boutillier[2]), abdominal pain (Duke),[3] headache (Brown),[4] and nervous manifestations (Shannon).[5] Whether or not the modified and rapidly developing local reaction, occurring after revaccination in a person previously successfully vaccinated, is to be considered an evidence of allergy is an unsettled question, and the same is true of the local and general symptoms following the injection of tuberculin in a tuberculous subject.

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[1] Amer. Jour. Dis. Child., 1923, XXVI, 451.
[2] Amer. Jour. Dis. Child., 1921, XXI, 89.
[3] Amer. Jour. Dis. Child., 1920, XIX, 114.
[4] Amer. Jour. Dis. Child., 1922, XXIII, 51
[5] Jour. Path. and Bact., 1922, XXV, 305.

Diagnosis.—The diagnosis of allergy may at times be made by the history, and often may be confirmed by testing with various protein-materials. An inherited tendency to hypersensitiveness may be suspected if the family-history reveals the occurrence of eczema, asthma, hay-fever or urticaria in the relatives. Contact with, or the inhalation, ingestion, or hypodermic injection of the offending proteins, may be followed by definite allergic manifestations, and the avoidance of these proteins may result in cessation of symptoms. The test with purified protein-substances may be made on the scarified skin or intracutaneously. The former method is favored by Schloss[1] and many others, and, while not so sensitive as the intracutaneous, it is easier to perform and is to be preferred in the average case. A large variety of proteins ready for testing are procurable. When in paste form a small amount is gently rubbed upon the skin which has been previously slightly scarified. If the powdered form is employed it is necessary first to apply a drop of a decinormal sodium hydrate solution to the point of scarification, after which a small amount of the protein material is mixed with this solution and rubbed in. The amount of paste or powder which can be carried on the end of a small flat wooden toothpick is sufficient, a fresh toothpick being used for each protein to avoid contamination. In scarifying it is better not to draw blood. A small cut, ⅛ inch (0.32 cm.) long, may be made with a scalpel, or a Pirquet borer may be employed. At the suggestion of Dr. Bret Ratner of New York we use a Hagedorn needle, the pressure of which on the skin makes sufficient opening. The scarification may be done on the forearm or back, the latter region being preferable in a struggling child. A positive reaction appears in 5 or 10 minutes and persists for about 30 minutes. It consists of an urticarial wheal 5 m.m. (0.20 in.) or more in diameter, with irregular raised edges surrounded by a red areola. Smaller wheals or the occurrence of erythema alone constitutes a doubtful reaction. Occasionally the reaction is delayed. Because the skin of some patients will react with urticarial wheals to simple trauma, a control test, to which protein is not added, should always be made, and the test be performed on the sensitive skin near the bend of the elbow.

The intracutaneous test is made by injecting into the skin 0.01 to 0.02 c.c. (.162 to .324 min.) of standardized solutions of protein. The same type of reaction occurs as in the cutaneous method. The intracutaneous test, although more sensitive, is more likely to cause general allergic reactions. Such occurrences are reported by Schloss,[1] Gerstenberger and Davis,[6] Park,[7] and Cooke,[8] a fatal issue occurring in the last instance. Ophthalmic and nasal tests are sometimes used in the diagnosis of hay-fever. In the former a drop or two of a 1:1000 solution of the pollen is instilled into the conjunctival sac. A positive reaction is indicated by redness and increased secretion lasting more than 5 minutes. The nasal test is performed by spraying the nasal mucous membrane with a 1:1000 solution of the pollen. If a positive reaction occurs there will be sneezing, swelling of the mucous membrane and the discharge of a clear watery secretion.

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[1] *Loc. cit.*
[2] Arch. of Pediat., 1922, XXXIX, 308.
[3] Arch. Int. Med., 1921, XXVIII, 151.
[4] Wisconsin Med. Jour., 1920, XIX, 337.
[5] Amer. Jour. Dis. Child., 1922, XXIV, 89.
[6] Jour. Amer. Med. Assoc., 1921, LXXVI, 721.
[7] Amer. Jour. Dis. Child., 1920, XIX, 46.
[8] Jour. Immunol., 1922, VII, 119.

A negative cutaneous reaction does not necessarily mean that the patient is not sensitive to the particular protein with which the test was made. It may be that a state of temporary desensitization exists, or that the skin does not react (Schloss),[1] or that the protein-material used may have been improperly prepared or may be stale. It is not always possible or desirable to do a great many tests in an individual case, and judgment must be exercised in selecting the proteins to be employed. The history may suggest the causative substance. Certain manifestations point toward definite groups of proteins. Thus hay-fever is frequently dependent upon pollens; asthma upon pollens, animal emanations or hairs; gastro-intestinal and cutaneous symptoms upon ingested animal or vegetable proteins. To spare unnecessary scarifications in a child the proteins may be mixed, and a group of several tested together; a negative reaction ruling out the group, a positive one necessitating individual tests of the proteins of the group.

Treatment.—The patient should be protected from proteins which offend, whether by ingestion, contact, or in other ways. If this is done it is not unusual to find that hypersensitiveness has ceased after several months. The substitution of some other protein in the diet may be successful at times. Thus in allergy to cow's milk, human milk or goat's milk may often be taken without symptoms (v. Goat's milk, p. 108). In mild cases even so simple a procedure as the alteration of the protein in some way, without change of the variety, may be all that is necessary. Some infants, for instance, who are sensitive to raw cow's milk may tolerate it perfectly if it has been boiled for an hour or has been dried. A baked potato may be harmless although a boiled potato may cause allergic symptoms. Wheat may be tolerated when given as toast or as in some of the prepared cereals in which the heat has destroyed the allergic properties of the proteins (Walker).[2] When it is not possible or advisable to eliminate certain offending proteins from the diet, the effort should be made to render the patient tolerant to them. Thus, for instance, in the case of allergy of an infant to cow's milk, a minute quantity of the milk, insufficient to cause symptoms, is ingested daily. It may be necessary to give as little as 0.1 c.c. (1.62 min.) or even 0.001 c.c. (.016 min.). After a few days this amount is slowly augmented, and the process is then steadily continued over a long period of time, always keeping the amount below the point where symptoms are produced. Usually after 5 or 10 c.c. (0.169 to 0.338 fl. oz.) can be given safely the advance can be made quite rapidly. It is not necessary to measure the milk in cubic centimeters, but a drop may be added to several ounces of water, and only one drop of the mixture given. If this is well-tolerated, a steady augmentation of the amount of milk ingested can be made, either by increasing the quantity of the mixture given or its strength.

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[1] *Loc. cit.*

[2] Oxford Medicine, 1920, II, 217.

The same procedure may be used with other proteins such as egg-albumen or beef-juice.

Another method of desensitization is by the hypodermic injection of minute quantities of the special protein causing disturbance. This may be done with almost any protein, but is especially practiced with pollens or animal emanations. The patient is injected every 3 days with increasing amounts of the protein. Dilutions are made varying from 1:1000000 up to 1:10 of it in a centi-normal sodium hydroxide solution. A cutaneous test is first done with these and 0.1 c.c. (1.62 min.) of that dilution which does not cause a reaction is then used for hypodermic injection. After several injections of this dilution in increasing amounts (0.2 c.c., 0.3 c.c., 0.4 c.c. etc.) (3.24 min., 4.86 min., 6.48 min.), a stronger one is tested on the skin, and if no reaction occurs the dilution of this strength is injected in increasing amounts, the process being continued until tolerance is established to strong dilutions such as 1:100 or even 1:10.

Which of the methods of treatment is to be adopted depends largely upon the nature of the case. In the milder instances of allergy the simple avoidance of contact with the protein, or of altering its character, as by heat, may be sufficient, without any direct attempt at desensitization. If these methods are not successful, the alimentary desensitization will be most practical in allergy due to foods; in that excited by inhalation of protein the hypodermic method may be necessary; and in that dependent upon bacterial proteins vaccines may be used.

In patients in whom allergy is known or suspected to be present, great care must be taken in the injection of therapeutic sera. A cutaneous test with a drop of the serum to be employed should first be made, and if this proves positive and the treatment is urgently needed very small doses of the serum should be given subcutaneously every half hour, commencing with 0.02 c.c. (.324 min.) and gradually increasing to 1.0 c.c. (16.23 min.), after which the full remaining dose may be administered (Stitt).[1] In the more acute manifestations of allergy such as serum-sickness or asthmatic attacks, the hypodermic injection of 0.2 to 0.5 c.c. (3.24 to 8.1 min.) of a 1:1000 solution of adrenaline, repeated every 10 minutes if necessary, may give prompt relief. Pituitrin in the same amounts may be used alone or in combination with the adrenaline solution. Hypodermic injections of atropine are also effective in some cases.

CHAPTER VIII

ACIDOSIS AND ALKALOSIS

(ACIDOSIS)

Nature.—It is necessary to define acidosis accurately, as many conditions have been falsely included in this category and considerable confusion has arisen in terminology. It should be understood that a truly acid state of the blood, and therefore of the body, never occurs, because a certain degree of alkalinity is necessary for the carrying out of the normal processes of the body. The term "acidosis" designates a condition in which the acid-base equilibrium of the body is disturbed because acid substances are present in a quantity *relatively* so increased that the normal excess of alkali (base) is lessened; *i.e.*, there is a lowering of the alkaline reserve. Under normal conditions the reaction of the blood remains remarkably constant (pH of 7.3 to 7.5) and, according to the usually accepted views, is maintained so in the following manner: The acids which are constantly being formed are taken up by the blood, unite with the sodium bicarbonate, and the carbon dioxide set free is removed through the lungs. The base of the blood-phosphate also neutralizes acids and the acid-phosphate is excreted through the kidneys. Because of this action of the bicarbonates and the phosphates in maintaining normal hydrogen-ion concentration they are called "buffers." The blood-protein also acts as a buffer. A certain amount of the acid is excreted as ammonia-salts in the urine. The exact manner in which these processes operate is too technical to be discussed in this chapter. This mechanism may be disturbed in several ways. Thus an abnormal amount of acid may be introduced into the body, as is done experimentally in animals and as occasionally occurs in man. An excessive production of acids may take place in such conditions as starvation, acute febrile diseases, severe diarrhea, diabetes, intestinal autointoxication or fermentation and recurrent vomiting. Defective excretion of acids by the kidneys may be caused by nephritis and by anhydremia. Finally, deficient elimination of carbon dioxide may occur in respiratory affections or in the slowing of respiration by narcotics. It is also possible that a sufficient loss of bases may be brought about by diarrhea and hemorrhage to change the acid-base balance. It is not necessary in this place to discuss the factors which might cause a shift toward the alkaline side (alkalosis). (See p. 759.)

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[1] *Loc. cit.* 282

Inasmuch as the bicarbonate content of the blood represents the excess of base which is left over after all the fixed acids have been neutralized, and which therefore constitutes buffer-substance or base which is available for the neutralization of any excess of such acids as may appear, acidosis has been defined by Van Slyke[1] as a condition in which the concentration of bicarbonate in the blood is reduced below the normal level. If this diminution in bicarbonate takes place, but there is no alteration in the reaction of the blood as shown by the presence of a normal hydrogen-ion concentration, the acidosis is *compensated*, but if the hydrogen-ion concentration shows a shift toward the acid side (lowered pH) *uncompensated* acidosis is present. A considerable amount of the bicarbonate may be consumed, up to ¼ of the normal amount or less, before change in the hydrogen-ion concentration occurs. The definition given above covers most varieties of acidosis but would not include a form in which the acid-base equilibrium is upset, not because of lowering of the bicarbonate but because acid-substances are increased. This condition occurs in asphyxial states in which an excess of carbonic acid is present in the blood (Macleod).[2]

A sharp distinction must be drawn between acetonuria and acidosis. This is necessary on account of the wide-spread confusion which has arisen, according to which acidosis is supposed always to be indicated by acetonuria. *Ketosis*, the special type of acidosis dependent upon acetone, or ketone, bodies, viz: B-oxybutyric acid, aceto-acetic acid and acetone, may occur when these substances are retained in the blood and tissues; but *acetonuria* consists merely in the presence of acetone-bodies in the urine. It is a matter of excretion, and may or may not be accompanied by acidosis. These bodies are normally present in small amount in the blood and urine of healthy children, as shown, among others, by Veeder and Johnson[1] and by Moore.[2] An increase of the amount in the urine indicates a greater elimination; which does not necessarily mean that there is at the same time an increased retention in the blood and tissues. This retention is necessary for the production of the *ketosis*-type of acidosis, in which there is actual decrease in the alkali-reserve of the blood; just as there is in other types of acidosis due to carbonic, phosphoric, lactic, sulphuric and similar acids. It is only by the presence of characteristic symptoms and by laboratory tests that the diagnosis of acidosis can be made with certainty.

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[1] Jour. Biol. Chem., 1917, XXX, 289.

[2] Physiol. and Biochem. in Modern Medicine, 3rd Ed., 1920, 39.

Errors in the clinical diagnosis are frequent, on account of the misconception of this contrast. Any starving child, as, for instance, one with repeated vomiting and the consequent emptiness of the stomach, shows an increased amount of the acetone-bodies in the urine; and this has been wrongly assumed to be the proof of the existence of acidosis. So, too, a large proportion of cases of acute febrile disorders in early life exhibits a decided acetonuria, but without suffering in any way from acidosis. On the other hand, it is certainly true that children are especially predisposed to the development of acidosis. This depends either upon the smaller reserve of alkali which they possess, or upon the readiness with which their organism produces the ketone-bodies, or fails to burn them up, without any discoverable explanation.

Acidosis due to ketone-bodies is a common symptom in diabetes at any age. In early life this disease is a less frequent etiological factor merely on account of its less frequent occurrence; since a most dangerous acidosis develops with the greatest suddenness and facility in diabetes in children. A much more common cause is severe diarrhea, especially of the class of "food-intoxication," "cholera infantum," "summer-diarrhea," and the like, in which rapid and profuse loss of liquid and of the bases of the body takes place from the intestine. This is a form of the disease clearly not dependent upon the production of any excess of the acetone-bodies in the blood. It is probable that due to the dehydration there is defective renal elimination and a consequent retention of acid-phosphate (Howland and Marriott).[3]

Acetonuria is a frequent symptom in recurrent vomiting. The relationship of acidosis to this disorder will be discussed later (see Recurrent Vomiting, Vol. II, p. 62). The disease is apparently due to some intoxication, and this may be an acidosis, but the presence of acetonuria is no proof of it. Pneumonia and nephritis may be productive of acidosis, and it is very probable that many of the symptoms denominated uremic may be, in reality, dependent upon an acid intoxication. Finally severe acidosis may occasionally develop without any discoverable reason whatever.

There are certain **symptoms** which are suggestive of the disorder. Among the earliest of these may be mentioned restlessness, sleeplessness, excitement; and later a tendency to prostration, somnolence, and coma. When these appear in a case of diabetes, or in an infant with severe summer-diarrhea and decided oliguria, acidosis can with reason be suspected. The only positive symptom, however, apart from laboratory tests, is hyperpnea. This consists in a remarkable alteration of respiration, with deep and exaggerated inspiration and expiration, usually not increased in rapidity, and constantly present; yet without any functional or organic disorder of the lungs or heart to account for this, and without cyanosis. It may not be recognizable clinically in mild acidosis. The degree of hyperpnea is directly proportional to the reduction of the alkali-reserve of the blood, as demonstrated by laboratory tests. The presence and degree of compensated acidosis may be shown directly by a decreased carbon-dioxide combining power of the blood or blood-plasma, or indirectly by a diminished carbon-dioxide tension in the alveolar air, an increase in the ammonia-excretion in the urine, and an increase in the titratable acidity of the urine. In uncompensated acidosis there will be in addition an increase in the hydrogen-ion concentration of the blood (lowered pH). A valuable and practical test for determination of the alkali-reserve is that of Sellards[1] in which the amount of alkali required to change the reaction of the urine is noted (see Treatment).

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[1] Amer. Jour. Dis. Child., 1916, XI, 291.
[2] Amer. Jour. Dis. Child., 1916, XII, 244.
[3] Amer. Jour. Dis. Child., 1916, XI, 309.

The **prognosis** is in most cases unfavorable. If once the symptoms are well-developed, death is liable to occur. The relief of coma and of hyperpnea may be effected at least temporarily, but with only a deferring of the fatal issue when the acidosis has existed long enough for definite damage of the body-cells to occur. Coma may return and death result.

The **treatment** should be **preventive.** If any diseased condition exists in which it is known that there is danger of acidosis developing, the occurrence of the symptoms must be carefully guarded against. In diabetes the greatest caution must be observed in the removal of the carbohydrates from the diet, without the simultaneous cutting off of the protein, and especially of the fat. Starvation is the procedure indicated for the beginning of treatment. In the case of severe, profuse, diarrheal discharges in infancy, prompt means must be taken to check the loss of liquid from the system. Initial purgation in such cases is dangerous, and astringents and opium should be used at once, unless the presence of fever, tympanites, and the character of the stools indicate that irritating substances are still present in the intestinal canal. In all cases where acidosis is feared there should be a prompt administration of alkali, especially bicarbonate of soda, in sufficient amount to render the urine alkaline. It may be given by the mouth or by the bowel.

Even in cases where **symptoms already present,** indicate well-marked acidosis, efforts should be made to restore the alkali-reserve of the blood, the production of alkalinity of the urine being the indication of the success attained. The bicarbonate should be stopped when the urine becomes alkaline, the best indicator being methyl red, which becomes yellow in an alkaline medium. The test is more sensitive than that with litmus. In normal infants 2 to 3 grams (31 to 46 grains) and in older children 4 to 5 grams (62 to 77 grains) of bicarbonate of soda are sufficient to render the urine alkaline for 12 or more hours; but when acidosis is present from 4 to 10 times this amount may be required (Howland and Marriott).[2] In urgent cases it is best to administer the soda intravenously, using a 4 per cent. solution, and giving at one time 75 c.c. (2.5 fl.oz.), or more in infancy; the determination of the amount and of the number of injections being decided by the production of urinary alkalinity. The solution for this purpose should be prepared by a special method, since the process of sterilization decomposes the sodium bicarbonate and renders the injection very irritating. (See p. 260.) In spite of care, however, sloughing of the tissues may occur if any of the fluid escapes into them. The subcutaneous injection of the fluid is to be deprecated, as damage to the tissues is extremely likely to follow, even with the specially prepared solution. It is true that there is danger of the administration of alkali in excess, even when checked by repeated examinations of the reaction of the urine; but the danger appears to be decidedly less than that caused by the persistence of the acidosis.

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[1] The Principle of Acidosis and Clinical Methods for Its Study, 1917

[2] *Loc. cit.*

Glucose will correct the acidosis due to ketone bodies. It should be given in 5 per cent. solution by mouth or by intravenous injection or hypodermoclysis (100 to 200 c.c.) (3.38 to 6.76 fl.oz.). The fact that the total inorganic base of the blood may not be reduced in certain forms of ketosis would argue against the administration of sodium bicarbonate in these conditions (Gamble, Ross and Tisdall).[1] By some authorities alkalies are not only considered unnecessary but distinctly harmful in ketone acidosis. Certainly it is true that the best clinical results follow the use of glucose and that alkalies have little good effect in recurrent vomiting and diabetes.

In acidosis not due to diabetes and in which the administration of glucose seems to be of positive value the addition of insulin may be employed with advantage. It may be given in cases of recurrent vomiting and in post-operative acidosis (Thalhimer;[2] Fisher and Snell[3]). In addition to other treatment, the administration of water in large amount is important in all cases, and especially in those in which there has been a large loss of liquid from the system through the presence of diarrhoea. If this cannot be given by the mouth, or if not retained by the rectum, it may be administered in the form of normal saline solution, either by hypodermoclysis or, better, by intraperitoneal injection.

ALKALOSIS

Nature.—In alkalosis, in contradistinction to acidosis, there is an increase rather than a decrease of alkali in the blood and tissues. That it occurs more frequently than has been supposed is pointed out by Kast, Meyer and Schmitz.[4] This contribution, as well as that of Koehler,[5] contains valuable bibliography.

Alkalosis may be compensated or uncompensated; in the former the hydrogen ion concentration of the blood being normal (pH of 7.4) but in the latter decreased (pH increased to 7.5–7.8). The form in which it is observed most frequently is that resulting from the excessive administration of bicarbonate of soda, or, less often, of other sodium salts, such as the citrate, acetate and salicylate. It may occur also following the loss of hydrochloric acid from the stomach, which takes place with the frequent vomiting of such disorders as cyclic vomiting and pyloric obstruction; the failure of hydrochloric acid to enter the intestine resulting in lack of stimulation of the flow of the alkaline intestinal secretions, and a consequent retention of alkali in the body. In these conditions an excess of alkali is present in the blood. In another type of alkalosis, of less clinical importance, there is a relative overbalance in blood-alkali because of a decrease of carbon dioxide. This occurs following the removal of carbon dioxide from the blood as a result of voluntary hyperpnea (prolonged deep breathing) and the hyperpnea due to elevation of temperature (fevers and immersion in hot water).

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[1] Transac. Amer. Pediat. Soc., 1922, XXXIV, 289
[2] Jour. Amer. Med. Assoc., 1923, LXXXI, 383.
[3] Jour. Amer. Med. Assoc., 1924, LXXXII, 699
[4] Jour. Amer. Med. Assoc., 1924, LXXII, 1858.
[5] Arch. Int. Med., 1923, XXXI, 590.

In milder forms of alkalosis, the **symptoms** consist of nausea, vomiting, malaise, numbness, tingling and headache; but when the condition becomes more severe tetany and convulsions occur. The typical position of carpopedal spasm is assumed, Chvostek's and Trousseau's signs are present and the electrical excitability of the nerves is increased (see Vol. II, p. 532). It is necessary in this connection to note that the etiological connection between the tetany which occurs in spasmophilia and alkalotic tetany is not clear; in spasmophilia it seems to depend in some way on lowered calcium in the blood, and alkalosis may or may not be present. It is important to remember that, unless tetany supervenes, symptoms of alkalosis may be confused with acidosis. A situation might arise; as, for instance, in diabetes; in which soda administered for the purpose of combating an acidosis would, if continued in excessive dosage, result in an alkalosis (Grant)[1]. Large amounts of soda are especially dangerous when impairment of renal function, such as may obtain in nephritis or pyelitis, results in a failure to excrete alkali (Morse).[2] Alkalosis may develop when soda is given intravenously to combat the acidosis of diarrhea (Howland and Marriott).[3]

The **diagnosis** of alkalosis rests on the development of nausea, vomiting, numbness, tingling, headache, tetany or convulsions, in an individual who is receiving excessive amounts of soda, or who is suffering from persistent vomiting or from any of the other conditions enumerated above, which would predispose to a shift of the acid-base balance toward the alkaline side. The **laboratory diagnosis** of alkalosis depends on the type. If symptoms are present the alkalosis will probably be of the uncompensated variety and the pH of the blood will be increased over the normal of 7.4. In both uncompensated and compensated alkalosis the urine tends to become alkaline and there is diminution of the titratable acidity and a decrease or absence of ammonia. When there is an excess of alkali the carbon dioxide of the alveolar air will be increased, but in the type due to carbon dioxide deficiency it will be decreased. Acetone may be excreted in the urine; a condition serving again to prove that there is no necessary connection between acidosis and acetonuria (see p. 756).

The **treatment** consists essentially in **prevention.** Bicarbonate of soda should not be administered to the point of producing an excess of alkali in the blood and tissues. If used in diabetes or cyclic vomiting it should be discontinued as soon as the urine becomes alkaline, and the laboratory tests mentioned should be performed, when possible, as a check. The presence of acetone-bodies in the urine does not necessarily indicate a need for the administration of soda, because these substances may be found in the absence of acidosis and even in alkalosis. One-half per cent. hydrochloric acid by continuous proctoclysis in doses of 100 to 500 c.c. (3.38 to 16.91 fl. oz.) may be used to combat an **established alkalosis.** If carbon dioxide deficiency has been proved present by a decrease of carbon dioxide tension in the alveolar air, inhalations of 10 per cent. carbon dioxide with 90 per cent. oxygen may be given for periods lasting from 5 to 10 minutes.

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[1] Arch. Int. Med., 1922, XXX, 355.
[2] New York Med. Jour., 1920, CXII, 965.
[3] Quart. Jour. Med., 1918, XI, 289.

CHAPTER IX

DIABETES MELLITUS

Diabetes mellitus is a disease of metabolism in which carbohydrate is not efficiently utilized by the body, thereby causing a derangement of the normal metabolism of proteins and fats as well as of carbohydrates. It is probable that the sugar absorbed from the intestine is no longer properly changed from an inactive into an active form, so that it can be stored in the liver as glycogen or oxidized in the tissues, but that it circulates in increased quantities in the blood (hyperglycemia) and is excreted in the urine (glycosuria). It therefore becomes lost to the body as a source of energy. As a result the store of glycogen is rapidly exhausted and protein is attacked as a source for glucose. As carbohydrate is necessary for the normal metabolism of fat, incomplete combustion of fat occurs in diabetes, resulting in acidosis and coma.[1]

Only the salient features of diabetes, especially as applied to children, can be discussed in this connection. It was known to the ancients, but was first clearly distinguished from diabetes insipidus by Willis.[2] Although much less frequent in early life than later, our own experience accords with that of those who find it occurring at this time much oftener than formerly supposed, or than many statistics would indicate. It is to be distinguished from alimentary glycosuria and transitory glycosuria from other causes. (See Vol. II, p. 432.) Very probably the statistics of diabetes in children include many instances which belong to this class of cases. A characteristic distinction is that pointed out by Allen,[3] that whereas in non-diabetics the ingestion of carbohydrates in excess increases the amount which can be utilized by the subject, in diabetics the tolerance diminishes in proportion as the amount of carbohydrate is increased.

Etiology.—*Age* is an important factor. von Noorden,[4] found in 2800 cases of diabetes 3.8 per cent. occurring in the first 10 years of life. The report of the Registrar General for England and Wales from 1861 to 1870 inclusive gave a very similar percentage (Dickinson).[5] In 6494 deaths from diabetes there were:

Table 93.—Mortality from Diabetes in Early Life, England and Wales

Under 1 year	8
1 year old	19
2 years old	16
3 years old	15
4 years old	16
Total under 5 years	74 (1.1%)
5 to 10 years	114
Total under 10 years	118 (2.9%)
10 to 15 years	200 (3.0%)

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[1] Report of Insulin Committee, Univ. of Toronto, Jour. Amer. Med. Assoc., 1923, LXXX, 1847.

[2] Pharmaceut. ration., 1674, Sect. IV, Cap. III. Ref., Senator in Ziemmsen's Handb. d. spec. Path. u. Therap., 1867, XIII, 2, 117.

[3] Glycosuria and Diabetes, 1913, 1050.

[4] Pfaundler u. Schlossmann, Handb. d. Kinderheilk., 1923, I, 639.

[5] Diabetes, 1875, 66.

Joslin's[1] statistics show a larger number; 5.7 per cent. of his 2611 cases as having commenced in the first 10 years of life. The majority of deaths in early life occur after the age of 5 years, and especially from 10 to 14 years of age. The statistics given show, however, that infants even in the 1st year are attacked. The youngest cases under my care were in girls of 15 and 16½ months respectively. Knox[2] collected 16 cases in the 1st year of life. In Stern's[3] statistics of 117 collected cases in children, 6 were under 1 year of age, and in 1 of these the disease appeared to be congenital. Such a case seems to be that of Cuno,[4] of pancreatic diabetes in a child of 3 weeks. Kochmann[5] observed an instance in an infant of 4½ months. In Wegeli's[6] 108 collected cases in children, not included in Stern's series, there were:

TABLE 94.—INCIDENCE OF DIABETES IN EARLY LIFE

Under 1 year	3 cases
1 to 5 years	26 cases
5 to 10 years	31 cases
10 to 16 years	42 cases

In adult life diabetes is distinctly more common in males than in females, but in childhood it is perhaps somewhat more frequent in girls. *Heredity* plays a very important part, the disease having shown itself in the parents or grandparents, or more than one of the children being affected. This is true in from 10 to 25 per cent. of the published statistics. Consanguinity, gout, nervous disorders, tuberculosis and syphilis of the parents have also been considered causes. An excessive consumption of sugar sometimes results in a permanent glycosuria. Diabetes occasionally follows nervous shock. Not infrequently the first symptoms appear after acute diseases and infections, tonsillitis being especially prominent in this connection. In 56 per cent. of Boyd's[7] 32 cases in children infection preceded the onset of the disease. These facts, taken in conjunction with the decrease in glucose-tolerance which occurs during the course of diabetes following acute infections, may indicate a toxic effect produced by infection on the island-cells of the pancreas. In adults obesity predisposes to diabetes, but we have not observed this to be the case in children. Jews as a race have been supposed to be predisposed to the disease, but this was not true in children studied by Joslin[8] and it only obtained in Jewish adults, who are more obese than Gentiles. Trauma of the head and intracranial tumors, especially of the medulla, have been followed by glycosuria, but these cases probably should not be classed with true diabetes mellitus.

Pathological Anatomy.—There is usually no gross alteration in the pancreas, except a slight diminution in size. On microscopic examination the organ may be normal, or it may appear that the islands of Langerhans are diminished in number and there is hydroptic degeneration (vacuolation) of their cells (Weichselbaum and Stangl).[9] Other portions of the pancreas usually show no inflammatory or degenerative changes.

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[1] Treatment of Diabetes Mellitus, 1923.
[2] Johns Hopk. Hosp. Bull., 1913, XXIV, 274.
[3] Arch. f. Kinderheilk., 1889–90, XI, 82.
[4] Jahrb. f. Kinderheilk., 1910, LXXI, 623
[5] Jahrb. f. Kinderheilk., 1922, XCIV, 20.
[6] Arch. f. Kinderheilk., 1895, XIX, 1.
[7] Arch. of Pediat., 1923, XLI, 291.
[8] Jour. Amer. Med. Assoc., 1924, LXXXIII, 1847.
[9] Wien. klin. Wochenschr., 1901, XIV, 968.

Symptoms.—These do not differ materially from those in adult life, except that the onset is frequently more rapid and the course shorter. The chief symptoms consist in wasting, thirst, great appetite, and excessive secretion of urine containing glucose. In the beginning, and perhaps during several months, the symptoms are but little marked, and only unusual thirst or abundant secretion of urine, or moderate loss of weight and strength are noticed. The child does not seem ill in other repects and is decidedly improved by dietetic treatment. After a variable time the symptoms increase in severity. The thirst is very great and a surprising amount of water is drunk; the appetite is sometimes voracious, sometimes diminished; the tongue is red and dry; the teeth decay readily; the skin is dry and harsh and usually somewhat pale, dependent upon the anemia which is generally present. Perspiration is diminished or absent. Constipation is frequent. Nocturnal incontinence of urine is a common and often an early symptom. There is decided debility and the patient becomes very easily exhausted and suffers from loss of spirits, irritability, headache and diffuse pains. The temperature is subnormal. Wasting is progressive and rapid and finally becomes extreme.

As regards the character of the urine, the total secretion often reaches 3 or 4 times the normal amount, exceptionally even equalling 6 or 8 quarts (5678 or 7570) in 24 hours. It is pale in color, acid, and with the specific gravity ranging from 1025 to even 1050, although occasionally it may not be over 1015. The percentage of sugar varies from a small amount up to 5 per cent. or more, and even as much as 10 or 12 per cent. has been reported. The total quantity of sugar excreted in 24 hours may reach 6 ounces (170) or over. A small amount of albumin is frequent with a few casts. Acidosis (see p. 755) is a characteristic of the disease seen in all advanced cases. As it develops, acetone, diacetic acid and β-oxybutyric acid appear in the urine. With the glycosuria there is always an increase in the blood-sugar. The normal blood-sugar is from 0.08 to 0.12 per cent. It may be as high as 0.15 per cent. without the appearance of sugar in the urine. When the blood-sugar reaches 0.4 per cent. coma is imminent.

Complications.—These are much less frequent than in adult life. Epistaxis, furunculosis, urticaria, erythema, cutaneous abscesses, pruritus of the genitals, and eczema are not uncommon. Gangrene is rarely seen in early life. Cataract has been reported, but is not common at this age. This is true also of optic neuritis and retinitis. Nephritis of moderate degree is not infrequent. Peripheral neuritis and otitis are sometimes seen. Tuberculosis or, oftener, bronchopneumonia is a not unusual complication.

Course and Prognosis.—The course of diabetes in children is usually more rapid than in adults. It is rare that the attack lasts much over a year from the time symptoms are discovered, and usually a shorter time. As the earlier course of the disease is seldom recognized, the total duration of the affection is probably considerably longer than supposed. Some of the acute cases have apparently lasted only a few weeks. I have seen 1 instance in a girl of 4 years in which the interval from apparent health without recognized symptoms of any sort to death in coma equalled only 11 days. Under ordinary circumstances the course is progressive if no intercurrent disease occurs to terminate life.

Weakness and emaciation increase and finally coma develops. Death in coma is especially frequent in children; that from complications much less often seen than in adults. Generally the approach of coma is indicated by loss of appetite, nausea, vomiting, epigastric pain, the presence of acetone in the urine and its odor on the breath, great weakness and nervous irritability with jactitation, or somnolence. Unconsciousness then comes on with the characteristic deep respiration. Under treatment the fatal issue may be delayed, even coma responding favorably to insulin medication and glucose administration. The final prognosis in children is very unfavorable, if care be taken to exclude from the list cases not certainly diabetes, such as instances of transitory or alimentary glycosuria. Yet under newer methods of treatment the prognosis does not appear to be so uniformly bad as was formerly considered to be the case, and it would seem, as pointed out by Riesman[1] and others, that the disease at this time of life may sometimes run a mild course and terminate in recovery. Especially with insulin-treatment life may be prolonged and good nutrition maintained, and it is to be hoped that in early and mild cases the sparing the pancreas which this accomplishes may result in regeneration of the island-cells.

Diagnosis.—The disease would doubtless be oftener recognized were the urine of infants and children examined more systematically. The diagnosis rests principally upon the dryness of the skin, thirst, normal or increased appetite, loss of weight and strength, enuresis, increased secretion of urine and the recognition of glucose in it, and increase in the blood-sugar over the upper normal limit of 0.12 per cent. The blood-fat is usually increased over the normal of 600 mg. per 100 c.c.

Diabetes is especially to be distinguished from other conditions in which some form of sugar is present in the urine. *Transitory glycosuria* following gastro-intestinal disturbance or attacks of infectious diseases may resemble it closely. It lasts, however, but a few weeks at most and is not attended by the characteristic symptoms of diabetes. *Alimentary glycosuria* also may occur in certain individuals when an unusual amount of sugar is contained in the food. In the case of infants lactose may sometimes appear in the urine under similar conditions. The reduction of the amount of sugar given is followed immediately by disappearance of the symptom. *Renal glycosuria* may occur with a normal blood-sugar and no other signs of diabetes than the presence of a reducing substance in the urine. The urinary sugar in this case is usually persistent, less than 1 per cent., and unaffected by carbohydrate intake. It can thus be seen that in order to separate true diabetes from glycosuria of other nature examination of the blood-sugar should be made. It must be remembered, too, that there are other substances in the urine besides sugar which will reduce the copper of Fehling's solution. Other tests, such as fermentation, phenylhydrazin, or the use of the polariscope, must be employed before a positive diagnosis can be given in some cases. I have seen parents given much unnecessary alarm by a too hasty diagnosis.

Treatment. GENERAL DISCUSSION.—In the modern treatment of diabetes the employment of both dietary regulation and the administration of insulin may be necessary, and there is sufficient knowledge about both of these measures to permit, within certain limitations, of almost mathematical precision in the use of them. In an individual case the carbohydrate-tolerance can be ascertained, and the assistance required of insulin can be calculated in terms of units of this substance. It is manifestly impossible to describe all the modifications of treatment which have been recommended and used. In this chapter we shall endeavor to outline a method which is as simple as is consistent with safety, and which takes into consideration the newer knowledge of the disease and its management.

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[1] Amer. Jour. Med, Sci., 1916, CLI, 40.

INSULIN.—Before discussing the details of treatment a brief consideration of insulin is necessary. This substance is an aqueous solution of the active principle, or hormone, of the islands of Langerhans of the pancreas. It was isolated by Banting and Best, working in Macleod's Laboratory at the University of Toronto.[1] The extracts for clinical use were first prepared by Collip,[2] and the effect on human cases of diabetes reported by Banting, Best, Collip, Campbell and Fletcher,[3] since which time an extensive literature has arisen. For details the reader is referred to the report of the Insulin Committee of the University of Toronto[4] and the exhaustive review by Campbell and Macleod.[5] Especially should be mentioned the studies of Joslin[6] and of Allen,[7] and a number of reports by various other authors.[8] As prepared for use, the glucose-equivalent of 1 unit of insulin is about 2; *i.e.*, it is standardized so that 1 unit will enable a diabetic patient to metabolize about 2 gm. of glucose-forming food. It should be remembered that there is an individual variation in this respect, and also that with each added unit given less and less glucose per unit is burned up.

Insulin should be administered subcutaneously, care being taken that it is not injected too near the skin or intramuscularly. In the extreme of coma it may be employed intravenously. When ingested it becomes inert. When dissolved on the tongue or used by inunction it is absorbed slightly but in ineffectual amount. It is best injected ¼ to ½ hour before each meal in severe cases with diacetic acid in the urine; but in milder types twice or perhaps even once a day. After the injection of insulin the blood-sugar begins to fall and the effect lasts from 6 to 12 hours. Insulin by no means replaces dietary measures, but simply assists them by supplying the deficient pancreatic hormone. In order, then, to regulate the amount of insulin to be given it is necessary to know how much glucose is ingested and how much the pateint can metabolize. It must not be administered at all if a diet can be found which will keep the patient's urine sugar-free, and which is so balanced that the fatty acids will be metabolized beyond the ketone-stage, while at the same time the individual is maintained in a normal nutritional state, with allowance for necessary activity. Many adult diabetics can get along without insulin, but practically all diabetic children will be benefited by its use, the severity and progressive character of the disease in them making it difficult to give a diet which will keep them free of urinary sugar and of ketone-bodies, while supplying enough food for growth and development.

DIET.—The diabetic *diet* should be planned with 3 definite factors in mind: (a) The protein-content, (b) the relation between ketogenic and antiketogenic substances, (c) the glucose-tolerance of the individual. It may be assumed that in a child at least 1 gm. of protein will be required for each lb. of body-weight. Protein-containing foodstuffs are selected which will furnish this amount. It has been shown by Woodyatt[1] and by Shaffer[2] that there is a rather definite quantitative relation between the amount of glucose and the amount of fatty acid which can be metabolized without the bodies remaining and the consequent danger of acidosis. That is to say, ketogenic food-substances must be balanced against antiketogenic food-substances. Without entering into a theoretical discussion of the subject, it may be said that, from a practical standpoint, if the diet contains not more than 2.5 grams of fat for each gram of carbohydrate plus each gram of protein, ketosis will not occur. This proportion is based upon the fact that the total fatty acid in the diet should not be more than 1.5 times the total available glucose. The fatty acid (ketogenic) fraction is formed by 90 per cent. of the fat and 46 per cent. of the protein; and the glucose (antiketogenic) fraction by 10 per cent. of the fat, 58 per cent. of the protein and 100 per cent. of the carbohydrate. This may be expressed by the formula $\frac{K}{A} = \frac{0.46P + 0.90\,F}{0.58P + 0.10F + C}$. $\frac{K}{A}$ should not be more than 1.5. To put this in a more convenient form, Fat (gm.) = 2 Carbohydrate (gm.) + 0.5 Protein (gm.), indicating that the number of grams of fat in the diet must not exceed a figure obtained by adding twice the number of grams of carbohydrate and ½ the number of grams of protein present in the food.

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[1] Jour. Lab. and Clin. Med., 1922, VII, 251; 464.
[2] Canad. Med. Assoc. Jour., 1922, XII, 142.
[3] Canad. Med. Assoc. Jour., 1922, XII, 141.
[4] Jour. Amer. Med. Assoc., 1923, LXXX, 1847.
[5] Medicine, 1924, III, 195.
[6] The Treatment of Diabetes, 3rd Ed., 1923.
[7] Amer. Jour. Med. Sci., 1924, CLXVII, 554.
[8] Jour. Metabol. Research, 1922, II, 547–985.

This means, then, that in continuing the planning of the diet, in which protein has already been supplied in the proportion of 1 gm. per lb. of body-weight, the remainder of the food must be added so that there is 1 gm. of carbohydrate and not more than 2.5 gm. of fat for each lb. of body-weight. Children are prone to the development of acidosis, and it is well to keep the fat lower than would be permissible in adults; not much more than 2 gm. of fat being allowed per lb. of body-weight. This is especially important in initial diets until the glucose-burning power of the body is ascertained. The high-fat diets advised by Newburgh and Marsh[3] should be used cautiously in childhood.

Before proceeding with treatment and the prescribing of the diet, the cases must be divided into 3 groups:—(A) Those uncomplicated cases in which diacetic acid is absent from the urine; (B) Those in which diacetic acid is present; (C) Those in which there are symptoms of acidosis and threatening coma. Diacetic acid rather than acetone should be the criterion, because the test for the latter is too delicate. The presence of diacetic acid in the urine indicates a real danger, as it shows a positive disturbance of fat-metabolism.

A. TREATMENT OF UNCOMPLICATED CASES (no diacetic acid).—The details may best be illustrated by giving the procedure to be followed in a hypothetical case. Suppose that a diet is to be prescribed for a diabetic child whose weight is 50 lbs. Table 95 on page 771, is consulted and the foods selected are ordered either by weight or by household-measure as follows:

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[1] Arch. Int. Med., 1921, XXVIII, 125.
[2] Jour. Biol. Chem., 1921, XLVII, 433, 449; 1921, XLIX, 143; 1922, LIV, 399.
[3] Arch. Int. Med., 1920, XXVI, 647; 1921, XXVII, 699.

	P. (gm.)	F. (gm.)	C. (gm.)
Egg (100 gm. or 2 av. size)	13	12	0
Lean meat (100 gm. or size 1 chop)	25	12	0
Butter (30 gm. or 2 butter-balls)	0	24	0
20 % cream (100 c.c. or 3½ fl.oz.)	4	20	4
Olive oil (13 gm. or 1 tbsp.)	0	13	0
Orange (60 gm. or ¼ av. size)	0	0	5
Oatmeal (100 gm. or 2 tbsp.)	5	2	20
Macaroni (50 gm. or 2 tbsp.)	1.5	0.5	8
10 % vegetables (100 gm. or 2 tbsp.)	1	0	6
5 % vegetables (100 gm. or 2 tbsp.)	1	0	3
Soda cracker (6 gm. or 1 cracker)	0.5	0.5	4
	51.0	84.0	50.0

By employing the formula $F = 2C + 0.5\ P$. it is seen that ketone-forming substances are distinctly less than antiketogenic ($84 < 100 + 25.5$). Such a diet may well serve as a basic one for any diabetic child. If the patient's weight is less or more than 50 lbs. the amounts of food can be divided or multiplied by the desired factor: Thus, if a diet is to be ordered for a patient of 25 lbs., ½ the amounts can be prescribed, or if a patient of 75 lbs., 1½ times the amount of food is allowed. Amounts inconvenient for measurement can be eliminated by change in selection of the foods. The food should be divided into 3 parts, so that approximately ⅓ of the carbohydrate is given at each meal; such, for instance, as follows:

	Breakfast C. (gm.)
Orange (¼)	5
Oatmeal (1 tbsp.)	10
Cream (1 oz.)	1.3
Egg	0
Butter (1 ball)	0
	16.3

	Dinner C. (gm.)
Meat (size of a chop)	0
Macaroni (1 tbsp.)	8
10 % Vegetables (2 tbsp.)	6
5 % Vegetables (2 tbsp)	3
Olive oil (1 tbsp.)	0
	17

	Supper C. (gm.)
Oatmeal (1 tbsp.)	10
Egg	0
Cream (2½ oz.)	2.6
Soda-cracker	4
Butter (1 ball)	0
	16.6

The diet just described has fulfilled certain requirements previously outlined. It has also furnished vitamins and residue. Its digestibility is not as great as that of a non-diabetic diet, because of the high fat-percentage and the overbalance of animal protein. Variety can be obtained by substitution of various vegetables, cereals and fruits, and, as increases are made, the selection can be from foods of different character.

There remains to be ascertained whether such a diet is of sufficient caloric value to maintain the child in a good nutritional state and allow for activity and growth. If the number of grams of protein and of carbohydrate are each multiplied by 4 (caloric value of 1 gram of protein and carbohydrate) and the number of grams of fat by 9 (caloric value of 1 gram of fat), the total caloric value of the diet can be obtained. Thus in the diet above planned the caloric value is 1160. If the patient under consideration is 9 years of age, the caloric requirement per lb. per day is about 27 (see Table 96, page 772), and the total calories in the diet should be about 1350. On the diet planned, therefore, it is unlikely that there will be sufficient food allowance for activity and growth, although there may be enough for maintenance. This becomes more apparent if the normal weight is considered (see Table 97, page 772). The diabetic patient who has not been under treatment is usually underweight, but has a caloric requirement which should be judged on the basis of normal weight, and the effort should be to bring the weight gradually up to this standard, although some authorities believe that the nutritional aim should be to maintain the weight at about 10 per cent. below the normal. Care should be taken not to confuse real gain in weight which may occur with the edema which may attend under-nutrition and the administration of sodium bicarbonate, or large amounts of salts in broths and vegetables. If the patient becomes sugar-free in 4 or 5 days and continues so on the diet as planned, diacetic acid is absent from the urine, but the caloric intake is too low, the food may be increased every 2 or 3 days by adding about 5 gm. of carbohydrate and twice as much (10 gm.) fat, the protein remaining about the same. It is not necessary that the carbohydrate and the fat be raised at the same time. In the basic diet it will be noted that the fat is well under the upper limit permitted, and it may be increased before carbohydrate is added, provided it does not supply more than 2.5 gm. per lb. and does not overbalance the keto-genic-forming material,—this latter consideration being determined by the use of the formula $F = 2C + 0.5P$. If glycosuria persists on the initial diet or on the subsequent increases before the caloric intake is sufficient, insulin must be given. It is possible, of course, to diminish the carbohydrate lower than recommended in the initial diet, *i.e.*, below 1 gm. per lb., but a child whose tolerance is below that amount should be treated with insulin rather than starved.

There are several methods of regulating the dose of insulin, of which the following is satisfactory: Determine the amount of sugar in a 24 hour specimen of urine and give as the day's dose ½ as many units of insulin as there are grams of sugar. If only 5 units are necessary this dose may be administered before the morning meal; up to 30 units may be given, equally divided, before the morning and evening meal; over 30 units should be divided in 3 equal parts and given before each meal. Children are especially liable to develop untoward symptoms due to the hypoglycemia caused by too much insulin, and if the urine tests indicate that large doses are necessary (over 20 units) it is better to begin with ½ the indicated dose and gradually increase. When the urine has been found free from sugar for 3 days, the diet may be increased in the manner previously described. If sugar reappears before a diet of proper caloric value has been reached, the insulin dosage must be increased as indicated by the amount of sugar appearing in 24 hour specimens. If the urine is not examined quantitatively for sugar, insulin should be begun in doses as small as 1 or 2 units 3 times a day increased in increments of 1 unit until the qualitative test for urinary sugar is negative. The aim should be to give the smallest efficient dose of insulin. Constant observation of the nutritional state of the patient is necessary, and the urinary sugar should be estimated frequently, in order that an increase or decrease in glucose-tolerance will be met by a corresponding change in insulin-dosage.

B. Treatment of Cases with Diacetic Acid in the Urine.—The appearance of diacetic acid in the urine indicates faulty metabolism of fat and the danger of acidosis and coma. If only a small amount of diacetic acid is found, the diet should be altered in such a way that the fat is decreased and the carbohydrate increased, the dose of insulin being increased approximately 1 unit for each gram of carbohydrate added. For example, if the patient were on a diet such as that prescribed on page 767, most of the fat could be removed by eliminating the butter, cream and olive oil; and the oatmeal, macaroni and vegetables increased or other carbohydrate added as follows:

	P. (gm.)	F. (gm.)	C. (gm.)
Egg (100 gm. or 2 av. size)	13	12	0
Lean meat (100 gm. or size of 1 chop)	25	12	0
Orange (250 gm. or 1 av. size)	1.5	0	20
Oatmeal (200 gm. or 4 tbsp.)	10	4	40
Macaroni (100 gm. or 2 tbsp.)	3	1	16
10 % vegetables (100 gm. or 2 tbsp.)	1	0	6
5 % vegetables (100 gm. or 2 tbsp.)	1	0	3
Bread (30 gm. or 1 slice)	3	0	16
	57.5	29	101

This diet would furnish only 895 calories, but partial starvation is advisable in the face of a possible acidosis. When the diacetic acid has disappeared from the urine, fat and protein are added to the diet in about 5 gm. amounts daily, the carbohydrate and the insulin being diminished until the patient can be treated in the manner outlined for uncomplicated cases.

C. Treatment of Cases with Acidosis and Threatened Coma.—With large amounts of diacetic acid in the urine, and the onset of such symptoms as restlessness, drowsiness and slight hyperpnea, the patient should be put to bed, external heat applied, an enema given and the stomach lavaged. Fluid is forced, and the only food allowed is clear broth free from fat, orange-juice, oatmeal gruel or 5 per cent. glucose solution. If nausea and vomiting are present 5 per cent. glucose should be given by rectum. It is very desirable that a blood-sugar examination be made and that the CO_2 combining power of the blood-plasma be determined. Enough glucose should be given to produce the excretion of a small amount of sugar in the urine. The urine should be examined every few hours for sugar and acetone. From 15 to 30 units of insulin should be administered, the amount depending upon the urgency of symptoms, the age of the patient, the degree of the reaction for diacetic acid in the urine, or the results of blood-analysis; but in any event each unit of insulin should be buffered by the oral administration of 10 c.c. (1/3 ounce) of orange-juice or 1 gm. (1/10 heaping teaspoonful) of glucose or sugar. Subsequent injections of 10 to 20 units should be given every 2 to 4 hours, the amount and frequency being determined by the same considerations as in the case of the original dose. If acidosis has proceeded to the stage of *coma* the same plan should be followed, but the insulin dosage should be greater (30 to 50 units). In advanced cases insulin and glucose should be given intravenously, a 5 per cent. solution of the latter being injected at the rate of 10 c.c. a minute. If the intravenous route is difficult or impracticable, subcutaneous or intraperitoneal injection may be employed. The amount of glucose used will depend on the symptoms, the age and weight of the patient, and the laboratory examinations; but at least 1 gm. of it must be employed for each unit of insulin. When this would mean that an excessive amount of fluid must be injected intravenously, part of the glucose-solution may be administered by some other route than the intravenous, or a stronger solution used.

The value of soda in acidosis has been questioned, and by some investigators it has been considered harmful (see page 759). Certainly insulin has rendered it less necessary, but many students of diabetes are not yet ready to discard its use in acidotic states, especially when there is respiratory distress or hyperpnea. In the precomatose stage a teaspoonful of soda-bicarbonate in 4 or 5 ounces of water may be given every half hour for several doses; if coma is present it may be added in 3 per cent. strength to the glucose solution. An alkaline or neutral reaction of the urine is an indication for the cessation of soda-medication. Methyl red should be used as the indicator, the red color changing to yellow in an alkaline solution.

HYPOGLYCEMIA (Hyperinsulinism, Insulin-shock).—If an overdose of insulin has been administered; *i.e.*, more than can be buffered by the available glucose, certain symptoms will arise within from 1 to 5 hours. These are not usually observed until the blood-sugar is below 0.08 per cent. but they may appear at a level of 0.11 per cent. if a previously high concentration has been too rapidly lowered. Marked shock occurs when the blood-sugar reaches 0.04 per cent. Early symptoms of hypoglycemia are sudden hunger, weakness, restlessness and nervousness, pallor or flushing, dilated pupils and increased pulse-rate. At this stage the ingestion every 10 minutes of such small amounts of carbohydrate as a teaspoonful of corn-syrup or sugar, the juice of an orange or a piece of candy will relieve the symptoms. If a large overdose of insulin has been given there appear the symptoms of sweating, tremor, apprehension, vertigo, diplopia and delirium and, if carbohydrate is not supplied, convulsions, collapse, unconsciousness and death. In advanced states carbohydrate must be administered by stomach-tube, and if improvement is not quickly noted 20 c.c. (.68 fl.oz.) of a 50 per cent. solution of glucose should be injected intravenously. If the latter solution is not available, 10 to 15 minims (0.62 to 0.92 c.c.) of a 1 to 1000 solution of adrenaline or of pituitary extract may be injected subcutaneously. Hypoglycemia should be avoided by the careful balancing of the glucose-intake and insulin-injection. When, for any reason, food is not taken or vomiting and diarrhea occur, the insulin should be discontinued.

MISCELLANEOUS.—There is nothing gained by keeping diabetic children in bed, Joslin's[1] experience in 59 cases of the disease in early life being distinctly in favor of allowing them to be about and to exercise freely. Fatigue and exposure to cold should be avoided. If lack of strength renders rest in bed necessary massage should be employed.

Infections temporarily and often permanently lower glucose-tolerance. During the acute stage of any infection the food-intake in diabetic patients should be lowered ½ or ⅓, and after the acute stage the glucose-tolerance must again be ascertained before the full diet is resumed. Tonsillitis seems to be especially prone to cause a decrease in the ability to metabolize carbohydrate. If the tonsils are badly diseased they may be removed, the operation being a much safer procedure since insulin-treatment has been employed. In some instances tonsillectomy has resulted in improvement, enabling larger amounts of carbohydrate to

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[1] *Loc. cit.*, 462.

Table 95.—Values of Foods As Prepared for Use. (Not Including the Addition of Any Butter, Oil, Lard, Sugar or Flour Which May Have Been Employed)[1]

	Various ordinary portions			100 gm. portions		
	P(gm.)	F(gm.)	C(gm.)	P(gm.)	F(gm.)	C(gm.)
Foods Highest in Protein						
Dried beef (30 gm. or 1 oz.)	12	2	0	40	6	0
Lean meat (100 gm. or size 1 chop)	25	12	0	25	12	0
Chicken, stewed (100 gm. or 1 slice size 1 chop)	22	5	0	22	5	0
Chicken, roasted (100 gm. or 1 slice size 1 chop)	20	16	0	20	16	0
Sweetbread (100 gm. or 4 h. tbsp.)	20	9	0	20	9	0
Cottage cheese (30 gm. or 2 h. tbsp.)	6	0.5	1	20	1.5	3
Fish boiled (100 gm. or 4 h. tbsp.)	18	5	0	18	5	0
Cod, flounder, whitefish, shad, trout, salmon, haddock.						
Egg (50 gm. or 1 av. size)	6.5	6	0	13	12	0
Egg white (33 gm. or 1 av. size)	4	0	0	12	0	0
Milk, whole (30 c.c. or 1 fl. oz.) sweet or lactic acid	1.2	1.2	1.2	4	4	4
Skimmed milk (30 c.c. or 1 fl. oz.) sweet or lactic acid	1.2	0	1.2	4	0	4
Gelatin (30 gm. or 1 h. tbsp.)	1	0	0	3	0	0
Broth (30 c.c. or 1 fl. oz.)	0.75	0	0	2.5	0	0
Foods Highest in Fat						
Olive salad oil or lard (13 gm. or 1 tbsp.)	0	13	0	0	100	0
Butter (15 gm. or 1 ball or 1 r. tsp.)	0	12	0	0	85	0
Bacon (30 gm. or 1 large slice)	3	20	0	10	72	0
Egg yolk (18 gm. or 1 av. size)	2.5	6	0	13	33	0
Cream, 20 % (30 c.c. or 1 oz.)	1.2	6	1.2	4	20	4
Cream, gravity or 16 % (30 c.c. or 1 oz.)	1.2	5	1.2	4	16	4
Baked custard (50 gm. or 1 h. tbsp.)	3.5	3.5	1	7	7	2
Foods Highest in Carbohydrate						
Sugar (10 gm. or 1 h. tsp.)	0	0	10	0	0	100
Cereals:						
Shredded wheat (30 gm. or 1 biscuit)	3	0	23	10	0	75
Cornflakes (20 gm. or 1 h. tbsp.)	1.75	0	15	8.75	0	57
Bread, wheat (30 gm. or 1 small slice 3″ × 3½″ × ½″)	3	0	16	10	0	53
Gluten bread (40 gm. or 1 small slice 3″ × 3½″ × ½″)	12	0	12	30	0	30
Rice (100 gm. or 1 h. tbsp.)	3	0	24	3	0	24
Oatmeal, Ralston's, Pettijohn's (50 gm. or 1 tbsp.)	2.5	1	10	5	2	20
Macaroni or hominy, boiled (50 gm. or 1 h. tbsp.)	1.5	0.5	8	3	1	16
Cream of wheat Farina } (50 gm. or 1 h. tbsp.)	0.75	0	5.5	1.5	0	11
Saltine cracker	0	0	2			
Butter-thin cracker	0	0.5	2.75			
Soda cracker (3″ sq.)	0.5	0.5	4.25			
Graham cracker	0.75	0.75	6			
Green and Root Vegetables						
Potato { Baked (40 gm. or 1 h. tbsp.)	1	0	10	2.5	0	25
Potato { Boiled (50 gm. or 1 h. tbsp)	1	0	10	2.0	0	20
Lima beans (49 gm. or 1 h. tbsp.)	3	0	12	7.5	0	30
Green peas (30 gm. or 1 h. tbsp.)	2	1	4	6.5	3	13
10 % vegetables (50 gm. or 1 h. tbsp.)	0.5	0	3	1	0	6
String beans, carrots, turnips, beets, squash, onions, pumpkin.						
5 % vegetables (50 gm. or 1 h. tbsp.)	0.5	0	1.5	1	0	3
Fruits						
Apricot, dried (8 gm. or 1 av. size)	0	0	5	3	0.5	62
Prune (10 gm. or 1 small)	0	0	6	1.75	0	60
Plum (35 gm. or 1 av. size)	0	0	7	0	0	20
15 % fruits (25 gm. or 1 tbsp.)	0	0	3.75	0.75	0	15
Raspberries, blueberries, cherries, currents.						
Banana (195 gm. or 1 av. size)	1.5	0.75	28	0.75	0	14
Pear (150 gm. or 1 av. size)	0.75	0.5	20	0.5	0	13
Pineapple (50 gm. or ½ slice)	0	0	5	0	0	10
Apple (150 gm. or 1 av. size)	0.5	0.5	16	0	0	10
Grapefruit (200 gm. or ½ av. size)	1.5	0.5	20	0.75	0.	10
10 % fruits (25 gm. or 1 h. tbsp.)	0.5	0	2.5	2	0	10
Blackberries, strawberries, gooseberries.						
Orange (125 gm. or ½ av. size)	0.75	0	10	0.5	0	8
Peach (125 gm. or 1 av. size)	0.5	0	10	0	0	8
Lemon (65 gm. or 1 av. size)	0.5	0	3.5	1	0	6

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[1] Amounts less than 0.5 have been omitted as negligible.
Tbsp. = Tablespoonful—H. Tbsp. = Heaping Tablespoonful—Tsp. = Teaspoonful. R. Tsp. = Rounded Teaspoonful. See also Tables 71 and 72, pp. 191–194.

be given. This is not always the case, however, and is contrary to the experience of some investigators. Allen, Stillman, and Fitz,[1] for example, could see no improvement beyond that noted in other patients.

It is hardly necessary to say that the diabetic patient should be under close observation. If it is possible to do so, a blood-sugar analysis should be made every week or two. The urine must be examined frequently for sugar and diacetic acid. Even children can be instructed to perform these latter tests themselves, and the older ones acquire adeptness in calculating diets. Joslin's Diabetic Manual is very helpful in this connection. The patient or the patient's attendant should be instructed in the dangers of diabetes and in the mishaps of treatment, and it is well to have these written down so that they may be referred to in an emergency. A list of instructions would include: (1) The early symptoms of acidosis and the measures to be employed; (2) the early symptoms of hypoglycemia and the treatment to be given; (3) the indication for the cessation of insulin-injections, such as the refusal of food and the presence of vomiting and diarrhea; (4) the danger of infections and the fact that they lower carbohydrate-tolerance and consequently require a diminution in food-intake. Children must be under close surveillance lest they supplement their restricted carbohydrate in a surreptitious manner.

TABLE 96.—AVERAGE CALORIC REQUIREMENTS

Age (yrs.)	Cal. per lb.	Cal. per kilo.
2–4	34	75
6–7	32	70
7–10	27	59
10–14	22	48

TABLE 97.—APPROXIMATE WEIGHT AT DIFFERENT AGES

Age (yrs.)	Av. wt. lbs.	Av. wt. kilo.
2	27	12.25
3	32	14.5
4	37	17
5	42	19
6	45	20
7	49	22
8	53	24
9	58	26
10	64	29
11	70	32
12	78	35.5
13	87	40
14	96	43.5

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[1] Diabetes, Rockefeller Institute Monograph, 1919, No. XI, 82.

CHAPTER X

DIABETES INSIPIDUS

Increased secretion of urine, temporary or of a more chronic nature, is of common occurrence, and is due to various causes. This *polyuria* is referred to briefly later. (See Vol. II, p. 427.) Such cases are to be distinguished from diabetes insipidus, in which there is a lasting excessive secretion of urine with great thirst. This condition is not common at any time of life. It was first clearly distinguished from diabetes mellitus by Willis.[1]

Etiology.—Age exerts no noteworthy predisposing influence. Of 77 cases of all ages collected by Roberts,[2] 22 (31.4 per cent.) were under 10 years of age, and 7 of these under 5 years; in Strauss's[3] series of 85 cases, 21 (24.7 per cent.) were less than 10 years of age, and 9 less than 5 years; in 124 cases analyzed by Stoermer,[4] 19 (15.3 per cent.) were under 10 years of age and 12 of these less than 5 years, and in Rowntree's[5] 56 cases only 3 (5.2 per cent.) were under 10 years of age. The disease may occasionally appear to be congenital. In children the influence of sex is inconsiderable, whereas in adults rather more males are affected. Inheritance is probably the most powerful predisposing factor, several members of a family perhaps being affected. In Rowntree's series there was no instance of heredity, but, curiously, diabetes mellitus occurred in other members of the family in 4 instances. Various nervous conditions in the parents seem to predispose to the disease in the offspring. Trauma especially of the back of the head or neck has been followed by diabetes insipidus, and this is true also of morbid growths and other diseases of the brain, such as meningitis and encephalitis, acute infectious disorders especially influenza, and syphilis. There has been observed, too, a connection between diabetes insipidus and the pituitary body or the neighboring structures, and tumors or diseases of this gland may be attended by polyuria. (See Vol. II, p. 856.) We[6] have seen 1 instance in which the deposit of xanthoma tuberosum in and about the pituitary body was accompanied by diabetes insipidus. In the majority of instances of diabetes insipidus no cause can be discovered, although the influence of the pituitary body may still be active although unproven. There are no post-mortem lesions except those productive of the disease in secondary cases.

Symptoms.—The most marked early symptoms are great thirst and excessive secretion of urine, the onset of these often being sudden. The amount of fluid imbibed is sometimes remarkable and the daily secretion of urine may much exceed that of diabetes mellitus, reaching from 5 to 15 quarts (4732 to 14,195) or occasionally as much as 35 or 40 quarts (33 or 38 liters). It is pale, with a specific gravity of from about 1001 to 1005.

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[1] Pharmaceut. ration., 1674, sec. IV, cap. III. Ref. Senator, in Ziemssen's Hand-d. spec. Path. u. Therap., 1876, XIII, 2, 254.

[2] Urinary and Renal Diseases, 3d Amer. Ed., 1879, 198.

[3] Dissert. Tübingen, 1870. Ref. D. Gerhardt, Nothnagel's spec. Path. u. Therap., VII, 1.

[4] Dissert. Kiel, 1892. Ref. D. Gerhardt, *ibid.*

[5] Jour. Amer. Med. Assoc., 1924, LXXXIII, 642.

[6] Arch. of Pediat., 1922, XXXIX, 297; 1923, XL, 630.

There is no sugar; albumin is very rarely found, and casts never. The phenolsulphonepthalein elimination is normal or but slightly diminished. The total solids excreted are usually normal, sometimes increased. Inosite has been observed in some cases. Camus and Gournay[1] have found a decrease in the elimination of uric acid and an increase in that of purin bases. Most patients pass more urine during the night than during the day, and nocturnal enuresis is common. The skin is pale and dry with little or no perspiration; the temperature is sometimes subnormal; the blood exhibits nothing characteristic, the blood-sugar and non-protein nitrogen of it being normal; the appetite is variable. The general health is often but little affected. In many cases, however, there is more or less emaciation and malnutrition, and bodily and, less often, mental development may be retarded and a degree of infantilism result. Nervous symptoms may be present, among them neuralgias of various kinds, restlessness, vasomotor disorders, and anorexia or excessive appetite. A polycystic and fibrous form of osteitis may be associated (Heard *et al.*),[2] as may defects in the membranous bones and exopthalmos (Denzer).[3]

Course and Prognosis.—The course of the disease is of an essentially chronic nature. Unless terminated by some intercurrent disorder it may last for years;—sometimes from infancy to adult life. In other cases emaciation and debility progress and death ensues from this cause. Recovery, or, in other instances, tolerance may be finally established, but absolute recovery in well-established cases is rare.

Diagnosis.—This rests upon the persistent polyuria and thirst. After infectious fevers there is often a brief transitory polyuria, which lasts a few days or even weeks, and a similar condition may be associated with hysterical or other nervous symptoms. Chronic interstitial nephritis produces polyuria but not to so great a degree. It exhibits albuminuria and casts, with diminution in the excretion of solids, and is attended by other characteristic symptoms. Evidence of brain-tumor should be sought for.

Treatment.—The principal treatment consists in a careful and gradual restriction of the amount of fluid allowed, not, however, to the extent of producing decided discomfort, since the polyuria and polydipsia are the results and not the cause of the disease. The clothing should be warm, abundance of fresh air given, and the diet of the most sustaining nature. Very many drugs have been tried, none of them proving satisfactory, among those most in vogue being ergot, antipyrine, arsenic, belladonna and opium. It is doubtful whether the meager hope of benefit from any of these counterbalances the harmful secondary effects which they may produce. That the disease may sometimes be associated with syphilis renders antisyphilitic treatment advisable in cases giving a positive Wassermann reaction. Pituitary extract was first used by Francisco[4] and its value has been attested by many. Pituitrin given hypodermically in doses of 0.1 to 1 c.c. (1.62 to 16.23 minims) of the obstetrical or surgical solution, causes a diminution in the polyuria and thirst. It must be administered 2 or 3 times a day, for the effects are not lasting, extending seldom as long as 12 hours. It can, however, be given over a long period without apparent injury, and with temporary improvement of the symptoms and of the comfort of the patient. Administered by the mouth it seems valueless, but some relief

[1] Compt. rend. de la soc. de biol., 1924, XCI, 1137.
[2] Amer. Jour. Med. Sci., 1926, CLXXI, 38.
[3] Amer. Jour. Dis. Child., 1926, XXXI, 480.
[4] Gaz. d. osped. e d. clin., 1913, XXXIV, 1135.

follows the employment of an intranasal spray (Blumgart).[1] Lumbar puncture affords temporary relief, and according to Tucker[2] permanent cure may result in certain cases. It should be done with caution if a tumor of the brain is suspected. Galvanization of the spinal column is claimed to have given good results.

CHAPTER XI

PELLAGRA

The nature of pellagra (*pelle—agra*, rough skin) is still uncertain. Long attributed almost universally to a food-intoxication, the opinion gradually spread that it was an infectious disease, although satisfactory proof of this had not been supplied. More recently the view has developed that it is a "deficiency" disorder due to an absence of certain necessary substances from the diet. It was first recognized as a morbid entity by Gasper Casál[3] in Spain in 1735, although it had previously been known under various popular names; among them that of "Mal de la Rosa." For many years it has been of frequent occurrence in Italy, Roumania, Austria and some other southern countries of Europe, as well as in Egypt and India. Some cases have been reported in England and Scotland. It had been observed from time to time in the United States for a good many years, a case having been reported by Babcock[4] occurring as long ago as 1834, and one by Gray[5] in 1864. Since 1906 it has been recognized with alarmingly increasing frequency. It was estimated by Lavinder[6] that over 30,000 cases had occurred in this country, chiefly in the Southern States; yet nearly every State of the Union has suffered from it. Several cases in Philadelphia have come under my observation, 2 of them in children who had never been beyond the borders of Pennsylvania. The disease, or at any rate the recognition of it, has certainly extended with great rapidity.

Etiology.—Pellagra attacks all ages, although it seems most common in young adults. Studies of the malady as it is found in children in the United States have been made by Snyder,[7] Weston,[8] Rice,[9] Knowles[10] and Murphy.[11] These show that it may occur even as early as 2 months of life, and that it is rather frequent between the ages of 6 and 18 months. In 323 cases studied personally by Grimm,[12] 15 per cent. were in subjects in the first 15 years of life. It is very probable that the incidence in childhood is much greater than this. Females are oftenest attacked, at least in America, in the proportion of about 3:1; yet this relative difference does not apply so well to early life, nor to adults in other countries.

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[1] Arch. Int. Med., 1922, XXIX, 508.
[2] Amer. Jour. Med. Sci., 1922, CLXIII, 668
[3] Hist. Nat. y. Med. de el Princip. de Asturias, 1762, 327.
[4] Trans. Nat. Assoc. for the Study of Pellagra, 1912, 18.
[5] Amer. Journ. Insanity, 1864, XXI, 223.
[6] Trans. Nat. Assoc. for the Study of Pellagra, 1912, 27.
[7] Amer. Jour. Dis. Child., 1912, IV, 172.
[8] Amer. Jour. Dis. Child., 1914, VII, 124.
[9] Trans. Nat. Assoc. for the Study of Pellagra, 1912, 333.
[10] Amer. Jour. Med. Sci., 1915, CXLIX, 859.
[11] Arch. of Pediat., 1917, XXXIV, 254.
[12] Trans. Nat. Assoc. for the Study of Pellagra, 1912, 39.

The disease is very much more frequent in rural districts, and especially under unsanitary conditions and in warm climates. It makes it appearance much oftenest in the spring and summer months. A debilitated state of health seems to predispose to its development. It is not considered communicable, yet a familial occurrence is very frequently observed. Knight,[1] for instance, reported 10 pellagrins in one family, and marked familial predisposition is not uncommon in Italy.

The direct cause is still unknown. The employment of corn-meal as a food had long been considered the chief etiological factor, but later this theory was modified to the belief that the meal must be of an inferior quality. Lombroso[2] especially has championed the theory, advanced by others before him, that pellagra depends upon the action of a mould infecting spoiled corn, which produces a food-intoxication in those eating it. The apparent fact that pellagra occurs also in those who have not eaten corn, and in infants who have been fed only at the mother's breast, as well as the various experimental investigations made, have led to serious doubt of the existence of any etiological relationship between pellagra and corn, or mould growing in it. Alessandrini and Scala[3] maintained that the disease is due to the action of silicon in a colloidal form present in the drinking water. Sambon[4] believed the disorder to be an infectous one, dependent upon a protozoon transmitted by the bite of one orimore species of Simulium fly (black fly; sand-fly; buffalo-gnat). Roberts[5] maintained that it is transmitted by the mosquito. A careful study of the relationship of various insects to the disease made by Jennings and King[6] led to the çonclusion that the Stomoxys calcitrans (stable-fly) was the probable carrier of the infection. That it may be due to a virus or an organism of very minute size might be indicated by the experiments of Harris,[7] who claims to have produced the disorder in a monkey by inoculation with a filtrate from human tissue passed through a porcelain filter. Inoculation experiments by others have been unsuccessful and confirmation is needed. Jobling and Arnold[8] have isolated a photodynamic organism from the stools of pellagrins, which they have never been able to find in the feces of non-pellagrous individuals. This organism, they believe, needs further study as a possible etiologic agent. Siler, Garrison and MacNeal[9] and others favor the theory of infection as the cause. Stewart[10] thinks that it is due to lack of proper function of the thyroid.

More recently the prevailing opinion has veered again to the etiological relationship of the food-supply, and the evidence for this is so strong that the disease may be with reason placed provisionally among the nutritional disorders. Wood's[11] conclusions, based upon his experiments with corn, supported the view expressed by others that the cause is a deficiency in some of the vitamins. Goldberger and his co-workers began the study of pellagra in 1914, since which time they have published the results of their numerous observations.[1] Their studies seemed to point strongly to a fault in the protein, the essential primary etiologic factor being a deficiency in amino-acids. Acting on this assumption they have been able to prevent, cure, and even cause pellagra by manipulation of the diet. The protein-deficiency has not gone without challenge by Jobling and Arnold[2] and Hindhede.[3] More recently Goldberger[4] maintains that the primary dietetic fault appears to be very probably, but not certainly, a lack of a dietary essential heretofore included with the antineuritic under the designation of "vitamin B."

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[1] Jour. Amer. Med. Assoc., 1912, LVIII, 1940.

[2] Trattato profilattico e clinico della pellagra, 1892. German by Kurella, 1898.

[3] Contributo nuovo alla etiologia e patogenesi della pellagra, 1914. (Ref. Jour. Amer. Med. Assoc., 1914, LXIII, 868.)

[4] Jour. Tropical Med. and Hyg., 1910, XIII, 271.

[5] Amer. Jour. Med. Sci., 1913, CXLVI, 233.

[6] Amer. Jour. Med. Sci., 1913, CXLVI, 411.

[7] Jour. Amer. Med. Assoc., 1913, LX, 1948.

[8] Jour. Amer. Med. Assoc., 1923, LXXX, 365.

[9] South. Med. Jour., 1918, XI, 785.

[10] South. Med. Jour., 1919, XII, 238.

[11] Jour. Amer. Med. Assoc., 1916, LXVI, 1447.

Pathological Anatomy.—The lesions are subject to much variation and are uncharacteristic. Those of the skin are at first erythema; but finally are those normal to old age, such as atrophy, pigmentation and sclerotic alterations. Pigmentation may take place in the viscera; the kidney may be cirrhotic; the liver and spleen are often smaller than normal; brown atrophy of the cardiac muscle is common; the fat of the body is much diminished and the muscles atrophied; the fragility of the bones is increased. There may be evidences of chronic inflammatory changes in the brain, meninges and spinal cord. The chief lesions in the nervous system appear to be in the spinal cord, especially the pyramidal tracts, but the posterior columns are also involved. It is questionable, however, whether many of the post-mortem alterations are directly connected with the disease itself, or are not rather those which would be seen in senility or in chronic cachectic states from any cause.

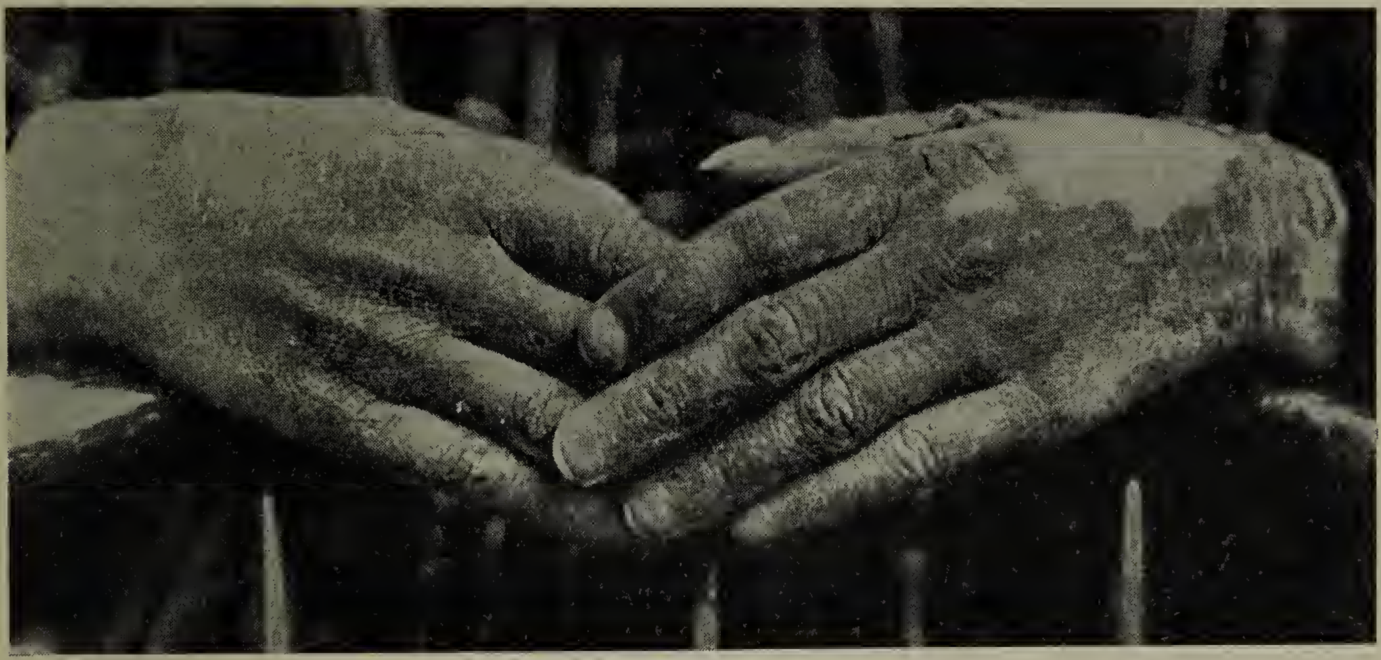

Fig. 233.—Pellagra.

Case in a girl of 9 years, in the Children's Medical Ward of the Hospital of the University of Pennsylvania, under the care of Dr. M. B. Hartzell. The child had never been outside of Pennsylvania. *Courtesy of Dr. Hartzell and of the J. B. Lippincott Co.*

Symptoms and Course.—The method of onset and the symptoms seen are subject to considerable variation. The latter can be divided into *cutaneous; digestive;* and *nervous.* Any one of these groups may appear first or be most prominent. The **cutaneous** are the characteristic manifestations. These may come on very insidiously or suddenly, the first change being a symmetrically developed erythema on the exposed parts of the body, such as the backs of the hands, the face and the neck; or in other cases, if the patient does not wear shoes, also on the feet and legs up to the knees. It resembles sunburn and in mild cases, and especially in young children, may easily escape recognition. In typical instances the erythema becomes darker-red and livid, and is followed in about 2 weeks or more by drying, scaling (*dry-type*) and pigmentation of the epidermis (Fig. 233). At the wrists or on the forearms it is sharply demarcated from the healthy skin above it, giving sometimes the appearance of a glove ("pellagrous glove"); or this may be seen on the foot and leg also ("pellagrous boot"), or around the neck ("Cásal's necklace"); in fact wherever the covered joins the exposed skin. In some cases vesicles and bullæ develop (*wet-type*), or there may be suppuration beneath the scaly, crusted epidermis. After a few months in the milder cases recovery occurs and the skin resumes its normal appearance.

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[1] Publ. Health Rep., 1914, XXIX, 1683; 1915, XXX, 3117; 1922, XXXVII, 462; 1923, XXXVIII, 2361; 1924, XXXIX, 87; Jour. Amer. Med. Assoc., 1916, LXVI, 471; 1922, LXXVIII, 1676; Arch. Int. Med., 1920, XXV, 451; Amer. Jour. Med. Sci., 1922, CLXIII, 928.

[2] *Loc. cit.*

[3] Jour. Amer. Med. Assoc., 1923, LXXX, 1685.

[4] Medicine, 1926, V, 79.

With or preceding the cutaneous symptoms there are **digestive** disturbances, generally most marked in children under 4 years of age. Of these severe diarrhea, often dysenteric, is one of the most prominent and troublesome, and frequently the first to appear. Stomatitis, with redness of the tongue and gums, is often present and is characteristic; salivation may occur, and vomiting is common; or there may be loss of appetite and abdominal pain. **Nervous** and general symptoms, too, may develop at any period. The child may be very little ill, or there may be malaise, loss of flesh, vertigo, depression, insomnia, headache, and cramps in various parts of the body. Occasionally severer nervous symptoms indicating lesions of the cord may be among the earliest manifestations, here especially being paræsthesia and, very often, exaggerated reflexes. The examination of the blood reveals nothing characteristic. There may be secondary anemia, but no leucocytosis or eosinophilia.

This is perhaps the history of the first attack. Entire recovery may seem to have taken place; but in the course of the year, sometimes in the autumn but much oftenest by the next spring, a second attack occurs, and this recurrence may be repeated during several years as spring comes on, the same symptoms developing but of greater severity and of longer continuance. Sometimes several years may pass by without an attack. Other regions of the body may become involved, as the entire forearms, the shoulders and the genital region; and very occasionally the eruption is universal. The skin in these later attacks is often not so vividly red as in the first, but is more deeply infiltrated and undergoes fissuring and finally atrophy, assuming a permanently pigmented, parchment-like character, with exacerbations of the erythema appearing in the springtime. The nervous symptoms are more marked in the later attacks, consisting then of very great lassitude, diffuse pains, paralytic or convulsive conditions, mental depression or insanity. Mental disturbances, however, although frequent in advanced cases in adults, are comparatively uncommon in children; except for a degree of dullness or irritability. In the final stage there is profound prostration, with great emaciation and the development of the typhoid state.

Prognosis.—On the whole this is very unfavorable. The death-rate in America according to Lavinder[1] equalled 37 per cent. in the 15,870 cases collected by him. Recovery in fully developed cases is very unusual. Death took place in the 1st year of the disease in 12 out of 55 cases collected by Tucker.[2] Although apparent recovery often occurs readily, recurrence of the symptoms is very liable to take place; and after perhaps many years the patient dies. Sambon[1] reported a case in a woman of 82 years who had had the disease since she was 17 years of age. Such a duration as this is, however, unusual. Severe cases may be fatal in 2 to 3 years, but generally the disorder lasts a longer time. There is reason to believe that, although pellagra is more rapidly fatal in infancy, in children from the age of 4 to 10 years the course is milder and recovery may take place under suitable treatment. If the deficiency-theory is correct, there is reason to hope that the prognosis in the future may be greatly better under proper dietetic management.

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[1] *Loc. cit.*

[2] Jour. Amer. Med. Assoc., 1911, LVI, 246.

Diagnosis.—The positive diagnosis can be made only upon the development of the characteristic cutaneous eruption, alone or in combination with the digestive and nervous disturbances described. In regions where pellagra is endemic, at least a provisional diagnosis may be made before cutaneous symptoms are seen, based upon the combination of redness of the tongue, diarrhea, lassitude, headache, paræsthesia and other nervous symptoms.

Treatment.—Infants with pellagrous mothers should not nurse from them. This is not because the malady is transmissible, but because the milk is generally inferior in quality, and infants continued at the breast develop intestinal disorders and an atrophic state. In cases of any age the most favorable hygienic surroundings should be chosen, including, if possible, change of environment, especially to a cooler climate. Hydrotherapy has been very valuable. The diet, too, should be made abundant and nourishing, and changed in some way from that which has been employed; especial care being taken to increase the amount of animal or vegetable protein by the inclusion of meat, eggs, milk and legumes. Yeast may be administered. Tonic remedies to improve the general health may be needed, including iron and arsenic either by the mouth or hypodermically; and a search for and removal of associated intestinal parasites is of importance. This treatment is to be followed not only in the developed disease, but as a prophylactic measure, in view of the decided predisposing influence which poverty and ill health exert. For the local treatment exposure to sunshine should be avoided, as this tends to produce or increase the eruption, and soothing applications should be used, such as are indicated in various forms of erythema.

CHAPTER XII

ACRODYNIA

(Erythredema, Pink Disease, Dermato-polyneuritis)

This interesting and distressing condition, the title of which, derived from the Greek, denotes "painful extremities," is of a nature unknown. Although it probably existed earlier, it was not differentiated until the last few years, cases of it doubtless being described as erythema or polyneuritis secondary to other conditions. Several authors attempt to associate it with an epidemic affection occurring in France in 1827 and 1829, but the difference in the age-incidence and the symptomatology of the earlier disease makes the connection uncertain. A condition having symptoms identical with acrodynia, as observed at the present time in the United States, has been recognized since at least 1914 in Australia and described by Swift[1] and others under the name erythredema. Byfield[2] observed instances of the affection in this country as early as 1915, and Bilderback[3] in 1914, although neither of these authors used the term "acrodynia." This title was applied to it later by Weston,[4] employing the name used for the disorder seen in the French epidemic referred to. A rather extensive literature on the subject has developed, and the disease has been recognized in various parts of the United States, Canada and Europe. We have ourselves seen at least 4 instances.

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[1] Trans Nat. Assoc. for the Study of Pellagra, 1912, 87.

Etiology.—Acrodynia, as here described, occurs only in infants and quite young children. The cases are sporadic, but may appear in groups in a way which suggests an epidemic. Sex and season apparently have little influence. It is seen in breast-fed infants whose mothers are on a satisfactory diet, and in the artificially-fed infants and in children no fault in the diet has been discovered which might indicate a causal relationship. There is nothing, therefore, on which to base the theory that it is a deficiency disease, due to lack of vitamins or other essential elements in the food. Byfield[5] is of the opinion that infection of the upper respiratory tract possesses etiological significance, and Rodda[6] is especially impressed with the relationship of the tonsils. The leucocytosis, which is almost always present, and the frequent association with fever support the view that the disease depends upon an infection of some type, and the nasopharyngitis which often is noted is in favor of the upper respiratory tract as the source of this. Vipond[7] discovered a Gram-positive diplococcus in the superficial lymph-nodes in 1 instance, an observation which has not been confirmed. Syphilis can be eliminated as a cause, since there are no symptoms of this and the Wassermann reaction is negative. The nature of the disease would seem to be a peripheral neuritis mainly involving the sensory nerves (Byfield).[5]

Pathological Anatomy.—The gross changes in the skin will be described in the paragraphs on symptoms. Microscopical examination of the skin of the dorsum of the foot, made by Paterson and Greenfield[8] in 1 case, showed nothing abnormal. In a child with acrodynia who died from an advanced pulmonary tuberculosis Byfield discovered gliosis in the spinal cord around the central canal; poor staining quality of some of the cells of the anterior horn of the spinal cord; edema of the myelin-sheath of the sciatic nerve and of the connective tissue about its nerve-fibres, and edema and swelling of the myelin-sheath in the fibres of the dorsal roots of the spinal nerves, in that portion lying between the ganglion and the spinal cord. Paterson and Greenfield[9] in 2 cases found considerable myelin-destruction in some fibres of the peripheral nerves. In the central nervous system in both of their cases there was a diffuse increase of small cells in the grey matter, especially in the lumbo-sacral enlargement of the cord and in the nerve-roots. The nature of these cells was not exactly clear, but in the cord they suggested glial origin rather than lymphocytic invasion, and in the nerve-roots they appeared to be derived from the nucleated sheath of Schwann.

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[1] Lancet, 1918, I, 611.

[2] Amer. Jour. Dis. Child., 1920, XX, 347.

[3] Northwest Med., 1920, XIX, 263; Jour. Amer. Med. Assoc., 1925, LXXXIV, 495.

[4] Arch. of Pediat., 1920, XXXVII, 513.

[5] *Loc. cit.*

[6] Amer. Jour. Dis. Child., 1925, XXX, 224.

[7] Arch. of Pediat., 1922, XXXIX, 699.

[8] Quart. Jour. of Med., 1923, XVII, 6.

[9] *Loc. cit.*

Symptoms.—The onset lasts 2 or 3 weeks and often considerably longer, during which the patient is listless, irritable, loses his appetite and may have moderate, irregular fever. Frequently there is present a rhinitis or naso-pharyngitis. The characteristic cutaneous eruption then appears. This consists of a symmetrically developed erythema of a decidedly pink color, which is most marked at the ends of the fingers and toes but extends over the hands as far as the wrists and the feet as far as the ankles, where it gradually shades off into normal tissue. Fine, red papules the size of a pin-head may develop. The rash is usually constantly present, although it may fade for a week or two, to return later. Desquamation of a branny type also occurs at intervals. The extremities are cold and clammy and occasionally cyanotic and appear swollen, although edema can seldom be demonstrated. The palms and soles may become pigmented. A vesicular or maculo-papular rash may occur on the body in scattered areas, which, on fading, is followed by desquamation; and in a few instances the rash characteristic of the hands and feet has also been seen on the cheeks and the tip of the nose.

A constant accompaniment of the cutaneous manifestations is pain in the extremities which, while constant, is worse at night. Older children will complain of paraesthesia, itching, and tingling and burning sensations, and all patients rub the hands together, and the feet as well, in an effort to obtain relief. Photophobia is another very common symptom, which causes the child to incline his head downward or to bury it in the pillow. There is marked insomnia and the mental state is one of great irritability and restlessness, which may be replaced at times by apathy and stupor. One cannot fail to notice the striking appearance of abject misery and wretchedness which the patients present.

Profuse perspiration is always present, and there may be an increased flow of saliva. Among the gastro-intestinal manifestations the most marked and frequent is anorexia, and there may be in addition loose and foul stools and occasionally vomiting. The teeth may fall out and infection and ulceration of the gums or even of the jaw-bone develop. Alopecia occurs at times and the finger-nails and toe-nails are occasionally shed. All the superficial lymphatic glands may be slightly enlarged. Fever, if present, is of moderate degree and not constant. A mild secondary anemia develops in some cases, and a leucocytosis from 10,000 to 20,000 or more is reported by most authors. The polymorphonuclear cells may be found to be normal or as high as 75 per cent., but there is no characteristic change in the differential count.

Course and Prognosis.—The disease usually lasts for several months, but complete recovery follows in almost all cases unless intercurrent infection occurs. In several reported deaths miliary tuberculosis has been found. Pyelitis is a not infrequent complication. The profuse sweating may so macerate the skin that peeling of that of the hands and feet may take place, as we have seen in 1 instance; and infection may cause a vesicular rash to become pustular. In some cases deep ulceration has occurred and portions of the fingers have been lost.

Diagnosis.—Acrodynia must be distinguished chiefly from pellagra. In the latter disease the rash on the hands is not as pink in color and there is a sharp line of demarcation where the cutaneous lesions cease; dietary error is found preceding the eruption; pain and paræsthesia are not present; and there is usually no leucocytosis. In Raynaud's disease the discoloration is more cyanotic, there is an earlier stage of coldness and pallor, and tingling and numbness are present; but none of the constitutional disturbances of acrodynia.

Treatment.—This is symptomatic and supportive. Soothing lotions, as of boric acid or calamine, may be applied to the extremities and the child restrained from constantly rubbing them. Atropine may help to check the excessive perspiration. Bromides, chloral or even codeine and morphine must be given to control the marked insomnia. Favorable effects sometimes seem to follow the administration of arsenic in some form. The diet ought to be that suitable for the child's age, and gavage resorted to if the anorexia cannot be overcome. General hygienic measures should receive consideration and plenty of fresh air and sunshine obtained. Any possible foci of infection which can be demonstrated should be removed. Good results from this are reported by Byfield[1] and Rodda.[2]

CHAPTER XIII

POISONING

Infants and children are more liable to certain kinds of accidenta poisoning than are adults. They not infrequently ingest such things as exterminators of rats or other vermin, fireworks, and the like. Overdose of patent medicines, the formulas of which are unknown, may be administered to the child or taken by him when carelessly left within his reach; substances known to be poisons may be taken accidentally, or well-known drugs may cause the development of toxic symptoms, either when given in overdose or because of the existence of an idiosyncrasy. Foods, too, may under certain conditions act as intense poisons. In this chapter only the important sources of poisoning in children can be indicated, and their symptoms and treatment briefly discussed.

Poisoning by Patent Medicines.—When the emergency arises, it may be difficult or impossible to determine the ingredients of the substance which has been ingested. Some idea can often be derived from its name, its appearance or odor if some of it is at hand, or the symptoms caused. Immediate analysis is usually not feasible, and the results of it would probably be learned too late to be of service. In view of these facts, it is convenient to have immediately accessible the publications of the American Medical Association, such as the volumes on "Nostrums and Quackery" or the smaller one on "Miscellaneous Nostrums."

Certain general facts may be helpful in this connection. Most patent *liniments* contain such drugs as menthol, capsicum, chloroform, alcohol and acetone. Many *laxatives* have phenolphthalein as their active ingredient, and others contain aloes, calomel and the like. The saline cathartics, as magnesium sulphate, sodium phosphate and sodium and potassium tartrate, are also largely used in such medicines. The

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[1] *Loc. cit.*

[2] *Loc. cit.*

headache, cold and grippe-cures usually consist of caffeine, antipyrine, phenacetin, salicylates, bromides and quinine. The *eczema-cures* often have such active ingredients as salicylic acid and carbolic acid. The *digestants* are harmless as a rule, and consist mostly of chalk, charcoal, pepsin and pancreatin. The medical constituent of most *worm-syrups* is santonin. Most *tonics* contain gentian and laxatives, but many possess such drugs as quinine, strychnine or iron. The numerous *ointments* are quite varied in character, and may consist among other things of carbolic acid, salicylic acid, turpentine and capsicum. There are several so-called *pneumonia-cures* and *salves* in which a lard-base is incorporated with such drugs as camphor, turpentine, carbolic acid, quinine, oil of thyme and oil of eucalyptus. *Asthma-cures* contain the iodides, atropine and some of them cocaine; others have potassium nitrate and stramonium present. The most vicious of all the patent medicines are the so-called *soothing syrups* or *baby's friends*. In these there are often laxatives such as castor oil, calomel or some of the saline cathartics. Carminatives such as oil of anise, oil of peppermint, oil of spearmint or chloroform are frequently present. Most important is the fact that many of them contain opium or its derivatives, patent medicine companies having the privilege of using in their nostrums as much as 2 grains (0.13) of opium, ¼ grain (0.016) of morphine, 1 grain (0.065) of codeine and ⅛ grain (0.008) of heroine to the ounce, and there are no guarded restrictions as to the frequency with which purchase may be made.

INSECTICIDES AND RAT-POISONS.[1]—These are another source of danger to infants and children. Their composition is varied and may include arsenious oxide, copper hydrate, lead oxide, lead arsenate, tobacco (nicotine), phenol, lime and sulphur. In some strychnine is present. Many of the powders for roaches, water-bugs, ants and the like contain sodium fluoride, white hellebore (veratrum viride), or sodium fluosilicate; in others there are lead, sulphur, lime and phosphorus, and in the liquid preparations phenol, nitrobenzene and mercury. *Fly-poisons* are usually some form of arsenic. Some *insect-powders* contain pyrethrum. The *moth-destroyers* are composed of naphthalene, camphor and cedar-gum. In many *insecticides* to be used against mosquitoes and bed-bugs there is methyl alcohol, and in a few tartar emetic and tobacco. It is very difficult to ascertain from the Government the ingredients of these substances.

FIREWORKS.—In some of these phosphorus and mercury are present. Several deaths in children are on record from phosphorus-poisoning caused by the ingestion of a variety of fireworks known as "Spit-Devils," "Son-of-a-Guns," or "Devils-on-the-Walk" (Dwyer and Helwig).[2] Another variety, known as "Pharoh's Serpents" or "Snake-in-the-Grass," contains mercury thiocyanid. We have seen a child of 2 years of age who ingested 3 of these, containing about 18 grains (1.15) of mercury in all; but who finally recovered after exhibiting anuria for 6 days, combined with very palpatable enlargement of the kidneys. (Friedman.)[3]

FOOD-POISONING.—Articles of food may cause disturbances in several ways, some of which, such as the gastrointestinal manifestations resulting from improperly balanced food or too great a quantity of it, are discussed elsewhere. So too is allergy and its symptoms, as also the transmission of parasitic and bacterial diseases by contaminated food. Certain vegetable and animal foods are in themselves poisonous, among which are some varieties of berries, mushrooms and fish. We wish, however, to call attention especially to that form of poisoning induced by the presence in the food of organisms of the colon-typhoid group, such as the Bacillus enteritidis, Bacillus suipestifer and Bacilli paratyphosus A and B. It is to such poisoning that the term "ptomain" is often incorrectly applied; —a ptomain being a toxic substance formed by the action of bacteria when carbon dioxide is split from an amino-acid. It is usually food of animal origin, such as the meat of hogs, cattle and fowls, which is the offender, but the eating of spoiled fish and occasionally of decomposed vegetables and milk-products may produce similar disturbances.

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[1] Information from Court Proceedings and letter from Insecticide and Fungicide Board, U. S. Department Agriculture.

[2] Jour. Amer. Med. Assoc., 1925, LXXXIV, 1254.

[3] Cincin. Med. Jour. 1926, XLIII, 193.

The *symptoms* are those of gastroenteritis and come on within 6 to 12 hours after the ingestion of the food. They are severe, and the diarrhea is apt to be more marked than are the nausea and vomiting. Associated with these there may be headache and vertigo, often fever, and always extreme prostration. The prognosis is good except in very young infants and children; these fortunately not being attacked as often because of their more restricted diet. The *treatment* consists in gastric lavage, after which purgatives should be given. Nothing must be done at first to check the diarrhea, until there is thorough clearing out of the intestinal tract. Supporting and stimulating measures are indicated, as will often be the combating of dehydration by the parenterial injection of fluid.

In this connection there should be noted a special type of food-poisoning known as **Botulism,** due to a partly anäerobic, spore-bearing organism, the Clostridium botulinum (Bacillus botulinus). This produces a toxin, the ingestion of which gives rise to rather definite symptoms. It is important to remember that the toxin is probably not formed in the body, but in the food before it is taken. It is into home-canned products, particularly vegetables but also fruits and meats, that the organism is likely to be introduced and its toxin formed; but commercial canned substances, among which should be mentioned sausage and ripe olives, are also responsible for the condition. The canned products contaminated by the organism are usually obviously decomposed. Heat destroys this toxin. The *symptoms* develop after 1 to 3 days or longer. While there may be mild gastro-intestinal irritation, the chief manifestations are those of peripheral nervous involvement. Thus there are disturbances of vision, such as dilated or irregular pupils, strabismus, diplopia and sometimes nystagmus. Paralysis of the pharyngeal muscles may occur, resulting in difficulty in swallowing. There is marked constipation and abdominal distention dependent upon the nervous involvement and general muscular weakness. No sensory symptoms are present and the mind usually remains clear. The *mortality* is high, over half of the patients succumbing to the poison. *Treatment* is supportive and stimulating in nature. Atropine should not be given since in many ways it produces the same effects as the toxin. A serum has been prepared which has definite prophylactic value, but seems to be of little aid in treatment of the developed disease. Inasmuch as there are at least two groups of the Clostridium botulinum a polyvalent serum should be employed and a single dose of about 20,000 units given at once. Repetition is not indicated since the toxin does not continue to be formed in the body. Experimental work by Bronfenbrenner and Weiss[1] suggests that keeping the patient under the influence of ether or luminal might have a favorable effect. Edmunds[1] claims that the administration of morphine delays the course of the intoxication, and allows a longer time for antitoxin to exert its influence.

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[1] Jour Amer. Med. Assoc., 1921, LXXVI, 1741.

POISONING IN OTHER WAYS.—*Dyes* may be a source of poisoning. Some laundry-inks contain nitrobenzene or anilin. These same substances may also be present in shoe-dyes and in some hair-dyes. Poisoning may occur by the ingestion of these substances or by their absorption through the skin. Cases of poisoning have been reported from the wearing of shoes recently dyed, or in which inks containing these substances have been used for marking clothing. (Muehlberger).[2] In infants there may be sufficient absorption of *phenol* from ointments, when these are applied over an open wound, or even over the diseased skin as in an eczema, to cause general symptoms of poisoning. In the same way *salicylic acid* in ointments, if in large amounts and used over an extensive area, may be absorbed and produce systemic manifestations as well as local irritation. Chronic *lead* poisoning may result from eating paint from cribs or toys; and we have also known instances of it from the employment of a lead nipple-shield by the mother, with lack of care to cleanse the breast before nursing. In these plumbic cases there may be the development of anemia, recurring attacks of violent colic, neuritis, nephritis or encephalitis. The lead dots appear on the gums if teeth are present, and there is stippling of the red blood-cells. *Subacute or chronic arsenical* poisoning may follow the administration of such drugs as Fowler's solution for the treatment of anemia, chorea and the like. Arsenic is also contained in certain dyes used in such materials as wall-paper or stockings, and poisoning may result in this way, apparently by contact or inhalation. The symptoms of acute arsenical poisoning will be detailed below. In the less acute types some of these symptoms may also develop, and the patient suffers from diarrhea, abdominal pain, albuminous and scanty urine, multiple neuritis, and such cutaneous manifestations as herpes, pigmentation, and loss of hair.

GENERAL SYMPTOMS AND TREATMENT OF POISONING

Certain general measures for poisoning will at once suggest themselves and be indicated by the symptoms present. Among these will be gastric lavage and the administration of emetics and purges. In the corrosive poisons demulcent drinks, such as milk, white of egg and bland oils, will be needed. Collapse will be treated by the application of external heat and elevation of the lower part of the body. Stimulants such as strychnine, atropine, camphor, caffeine, coffee by mouth or rectum, adrenaline, and pituitary extract will be employed if needed. Artificial respiration and administration of oxygen may be necessary at times, and in most poisonings the giving of large amounts of water by mouth or parenterally will be helpful. In *poisoning by heavy metals* the administration of sodium thiosulphate has been recommended as an antidote on the ground that it will cause precipitation of metals such as arsenic, lead, mercury, copper, zinc and bismuth. It is supposed that it will not only neutralize those metallic poisons which are free in the gastro-intestinal tract, but will also render soluble those which have already been taken up by the body. The stomach is washed with 500 c.c. (16.9 fl. oz.) of water to which has been added 30 gm. (463 grains) of sodium thiosulphate; after which a similar amount is introduced into the stomach and allowed to remain. If given intravenously Dennie and McBride[1] recommend from 0.3 to 1.8 gm. (4.6 to 27.8 gr.) of the drug in 20 c.c. (0.68 fl. oz.) of distilled water every day for 4 days and then every other day as indicated. Further reports, such as those of Haskell, Henderson and Hamilton[2] seem to indicate that this treatment is of little value.

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[1] Jour. Amer. Med. Assoc., 1924, LXXXIII, 495.

[2] Jour. Amer. Med. Assoc., 1925, LXXXIV, 1987.

In the following paragraphs the chief symptoms and the treatment of poisoning of various kinds are given in outline form:

1. **Acids.**—(Hydrochloric, acetic, sulphuric, nitric, oxalic.) *Symptoms:* Destruction of mucous membrane of mouth; violent gastro-intestinal irritation with pain, nausea and vomiting; bloody diarrhea; prostration; collapse. *Treatment:* Alkalies such as magnesia, chalk, sodium bicarbonate, soap, followed by white of egg and demulcents. Intravenous injections of sodium bicarbonate and of glucose; force fluid. Do not use stomach-tube. In oxalic acid poisoning do not use soda or potash; lime is the best antidote.

2. **Alcohol, methyl.**—(Bed-bug poison; boot-leg liquor.) *Symptoms:* Nausea, vomiting, vertigo, delirium, blindness. *Treatment:* Bleeding and intravenous saline injections; emetics; lavage; force fluids; cathartics; external heat; stimulation or sedatives. No direct antidote.

3. **Alkalies.**—(Ammonia, spirits of hartshorn, lye, caustic potash, washing soda.) *Symptoms:* Those of gastro-intestinal irritation, as with acids. *Treatment:* Dilute acids, vinegar, lemon-juice; force fluids; cathartics.

4. **Antimony.**—(Tartar emetic, some of the insecticides.) *Symptoms:* Severe vomiting and diarrhea, collapse, increased flow of saliva. *Treatment:* Tannic acid, strong tea; cathartics; stimulation; external heat.

5. **Arsenic.**—(Fly-poisons, rat-poisons, some colored crayons and artificial flowers, Paris-Green, wall-paper.) *Symptoms:* Gastro-intestinal irritation and pain, nausea and vomiting, colic, diarrhea; perhaps convulsions. Later albuminous and scanty urine; still later multiple neuritis and dermatitis. *Treatment:* Ferri hydroxidum cum magnesii oxido, 3 oz. (93.), repeated as necessary; lavage; emetics; castor oil; demulcents; adrenaline; fluids.

6. **Atropine.**—(Belladonna.) *Symptoms:* Flushing of skin, dilated pupils, dryness of throat, tachycardia, delirium, convulsions. Death from respiratory failure. *Treatment:* Emetics, lavage; force fluid; cathartics; promote perspiration; chloroform for convulsions. Use morphine cautiously if at all, because of depressing respiratory effect.

7. **Carbolic Acid.**—(Some roach and bug-poisons; various proprietary antiseptic applications.) *Symptoms:* Gastro-intestinal irritation, vomiting and bloody diarrhea, colic; drowsiness; collapse; muscular twitchings, convulsions. Later nephritis with scanty, albuminous and bloody or smoky urine. *Treatment:* First, white of egg; then lavage with 10 per cent. alcohol. Magnesium sulphate. Do not give emetics or oils.

8. **Coal Tar Derivatives.**—(Acetanilid, phenacetin, antipyrine, headache-cures, cold and grippe-cures; anilin.) *Symptoms:* Depression; collapse; cyanosis; clammy sweat; vomiting; low blood-pressure; somnolence; slow pulse made rapid on exertion. Death from respiratory failure. *Treatment:* In addition to emetics, lavage, external heat and stimulation give large doses of sodium bicarbonate. Oxygen has no effect on the cyanosis.

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[1] Jour. Amer. Med. Assoc., 1924, LXXXIII, 2082.

[2] Jour. Amer. Med. Assoc., 1925, LXXXV, 1808.

9. Cocaine.—(Asthma-cures.) *Symptoms:* At first rapid pulse, high blood-pressure, delirium, convulsions, dilated pupils. Later collapse and respiratory failure. *Treatment:* Strong tea; tannic acid; emetics; lavage; cathartics. If collapse, external heat; stimulants. If convulsions, sedatives.

10. Copper.—(Certain rat-poisons, blue vitriol or blue stone.) *Symptoms:* Gastro-intestinal irritation; vomiting and diarrhea, the vomitus and stools being greenish or bluish at first; colic; salivation; collapse. Later, if the patient survives, there may be greenish discoloration of skin, gums and teeth; neuritis and nephritis. *Treatment:* Emetics; lavage; demulcent drinks and purgatives; stimulation; external heat; large doses of sodium bicarbonate. The chemical antidote for copper is yellow prussiate of potassium (potassium ferrocyanide), and its ordinary dose is about 5 grains (0.32).

11. Fluorides.—(Certain bug-poisons.) *Symptoms:* These develop slowly, usually with gastro-intestinal irritation, vomiting and diarrhea. The action of these substances is said to be in precipitating calcium from the tissues. *Treatment:* Emetics, lavage and purgatives; force fluids.

12. Lead.—(Some rat-poisons. Paint. . Chronic poisoning may result from eating paint off cribs or toys.) *Symptoms:* Gastro-intestinal irritation, nausea and vomiting; collapse; numbness; paralysis. Later and in more chronic cases multiple neuritis; nephritis; encephalitis; non-protein-nitrogen retention in blood; scanty urine; blue line in gums; stippling of red blood-cells. *Treatment:* Emetics; lavage; magnesium sulphate; demulcents; stimulation; force fluid.

13. Mercury.—(Certain rat-poisons and insecticides; certain fireworks; medicinal preparations such as calomel, bichloride of mercury; some patent-medicine-laxatives.) *Symptoms:* Gastro-intestinal irritation, vomiting and diarrhea, colic, bloody stools; possibly delirium and coma. Later nephritis and possibly neuritis. *Treatment:* White of egg before lavage; large doses of alkalies by mouth.

14. Muscarine.—(Poisonous mushrooms.) *Symptoms:* Vomiting; colic; loose stools; increased saliva and tears; contracted pupils; vertigo; tremors; convulsions. *Treatment:* Emetics; lavage; tannic acid; atropine; adrenaline; pituitary extract; external heat; glucose infusions.

15. Nicotine.—(Tobacco, certain insecticides.) *Symptoms:* Depression; clammy perspiration; nausea; vomiting; collapse. *Treatment:* Tannic acid; strong tea; lavage; emetics; cathartics; external heat; stimulation.

16. Opium.—(Paregoric, laudanum, morphine, heroine, codeine, soothing syrups.) *Symptoms:* Contracted pupils; slow pulse and respiration; flushing and cyanosis. *Treatment:* Tannic acid and strong tea; emetics; lavage with 1 to 1000 solution of potassium permanganate; stimulation, especially caffeine and strychnine; artificial respiration; keep awake.

17. Phosphorus.—(Certain fireworks; certain rat and bug-poisons. Phosphorus in a poisonous form is now rarely used in the manufacture of matches.) *Symptoms:* Usually develop slowly, *i.e.*, after several hours;—Nausea; vomiting (garlic-odor); prostration; intestinal pain. Later bloody diarrhea; enlargement of liver; jaundice; convulsions; coma; nephritis; sugar in urine; hemorrhage from mucous membranes; increased cholesterol in blood. *Treatment:* Emetics, especially copper sulphate 5 grains (0.32), lavage with 1–1000 potassium permanganate; hydrogen peroxide or ozonized turpentine (½ teaspoonful every hour for 5 doses);

cathartics; stimulation; external heat. Do not give oils. Atkinson[1] and Dwyer and Helwig[2] recommend 50–100 c.c. (1.7–3.4 fl. oz.) liquid petrolatum to act as a solvent of the phosphorus.

18. **Salicylates.**—(Headache and grippe-cures.) *Symptoms:* Flushing of skin; disturbance of vision and hearing; delirium; collapse; clammy sweat; deep sighing respiration; albumin in urine; scanty urine. *Treatment:* Emetics; lavage; cathartics; external heat; stimulation; sedatives if necessary.

19. **Sedatives.**—(Bromides, chloral, headache-cures.) *Symptoms:* Drowsiness; coma; subnormal temperature; nausea and vomiting; slow pulse; feeble respiration; diminished reflexes; collapse. *Treatment:* Emetics; lavage; cathartics; stimulation by strychnine, caffeine, pituitary extract; external heat; artificial respiration.

20. **Strychnine.**—(A few rat-poisons; tincture of nux vomica.) *Symptoms:* Irritability; nervousness; tetanic convulsions; rapid pulse. *Treatment:* Tannic acid; strong tea; emetics, lavage; chloroform-inhalations, sedatives, such as bromides and chloral.

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[1] Jour. Lab. and Clin. Med., 1921, VII, 148.

[2] *Loc. cit.*

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